MAJOR ARTICLE

Population-Based Surveillance for Postpartum Invasive Group A Streptococcus Infections, 1995–2000

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Estimates of disease burden and data on the sources of invasive postpartum group A streptococcus (GAS) infections will help guide public health action. Active, population-based surveillance was conducted in 9 regions from 1995 through 2000. A case of GAS infection was defined as isolation of GAS from a sterile site in a resident of a surveillance area who was pregnant or in the postpartum period. Census and live birth data were used to calculate rates. Eighty-seven cases of postpartum GAS infection (2.2% of 3957 invasive GAS infections) occurred at 3%–8% of hospitals annually. We estimate that 220 cases occurred annually in the United States. Two or more cases were noted during 6 months at 8 hospitals, during 1 year at 13 hospitals, and during 2 years at 16 hospitals. Cases due to identical *emm* types clustered more frequently than expected by chance. Although postpartum GAS infections are rare, the clustering of infections due to identical strains suggests that some invasive cases may have a common source and, therefore, may be preventable.

Streptococcus pyogenes, also called group A β-hemolytic streptococcus (GAS), is an uncommon but serious cause of postpartum infections. Obstetric patients are particularly vulnerable to invasive GAS infections acquired via disrupted cutaneous or mucosal barriers during delivery. Semmelweis's original report [1] described a case of epidemic puerperal sepsis, now recognized to have been caused by GAS, in which infection was associated with nosocomial transmission by birth attendants who failed to use aseptic techniques. Postpartum GAS infections in the modern era are a mixture of illnesses of endogenous origin and acquired infections. Women exposed to GAS prior to delivery may develop invasive disease in the peripartum period. Without prior exposure to GAS, women may develop invasive postpartum GAS infection from exposure to an infected health care worker. Although the sources of sporadic GAS infections are typically not known, outbreaks of postpartum GAS infections have been associated with health care workers who are carriers of GAS. Health care workers who were asymptomatic carriers were identified in 6 of the 10 outbreaks of postpartum infection reported since 1965 [2–7]. Although the proportion of cases of invasive postpartum GAS infection attributable to infected health care workers is unknown, investigation of clusters suggests that prompt institution of investigation and control measures may prevent further postpartum morbidity [2]. Guidelines for appropriate epidemiologic investigation after the identification of an index case of potentially hospital-acquired invasive postpartum GAS infection may assist these preventive efforts. To facilitate development of guidelines for epidemiologic responses to cases or possible clus-

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ters, we quantified the burden of invasive postpartum GAS disease in a multistate population.

METHODS

Surveillance for invasive postpartum GAS infection. From 1995 through 2002, active population- and laboratory-based surveillance for invasive GAS infections was conducted through the Centers for Disease Control and Prevention's (CDC) Active Bacterial Core Surveillance (ABCs) program, which is part of the Emerging Infections Program Network. Data on cases of postpartum invasive GAS infection were subsequently abstracted. ABCs conducted surveillance in counties in 9 areas of the United States that had a minimum aggregate population of 23.1 million and a mean of 248,480 births in ~200 hospitals annually. Project personnel routinely contacted all participating clinical microbiology laboratories in the following areas: California, 3 counties (since 1995); Connecticut, all counties (since 1995); Colorado, 5 counties (since July 2000); Georgia, 8 counties (since 1995), then 12 additional counties (since 1997), and then the whole state (since 2000); Maryland, 5 counties and Baltimore (since 1997); Minnesota, 7 counties (since 1995), then the whole state (since 1999); New York, 7 counties (since 1997), then 8 more counties (since 1999); Oregon, 3 counties (since 1995); and Tennessee, 5 counties (since 1 January 1999), then 6 additional counties (since 1 August 1999). To ensure complete case ascertainment, periodic laboratory audits were conducted in each area.

We defined a case of invasive postpartum GAS infection as isolation of GAS from a normally sterile site (e.g., blood or CSF) or from a wound tissue culture when accompanied by necrotizing fasciitis or streptococcal toxic shock syndrome in a resident of a surveillance area who was pregnant or in the postpartum period (i.e., $\leq$30 days after delivery) or who had clinician-defined puerperal fever, chorioamnionitis, or septic abortion. Cases in which GAS were isolated from amniotic fluid or the placenta alone were excluded. Demographic and clinical data for patients with invasive GAS infection were gathered from standardized case report forms and summarized. Invasive GAS isolates were sent to the CDC, where they were subtyped by sequencing of the type-specific region of the M virulence protein gene *(emm)* by use of methods described elsewhere [8, 9].

Estimates of disease burden and multiple cases of postpartum GAS infection in hospitals. We determined the number of ABCs hospitals that identified cases of invasive postpartum GAS infection and summarized the number of hospitals that identified $\geq$1 case per year, as well as the proportion of hospitals that identified $\geq$2 cases per year. Estimates of disease incidence were calculated using the most recent available population data from the US Bureau of the Census (1999) and live birth data from the National Center for Health Statistics (1997).

To identify possible common-source clusters, we noted the occurrence of multiple cases of GAS infection at a single hospital during 6-, 12-, and 24-month periods. We performed *emm* sequencing to subtype GAS isolates [8, 9]. Within the United States, there is a high correlation between the *emm* sequence type and the specific GAS clonal type [10]. If *emm* typing data were available, we also summarized the occurrence, during the same time period and at the same hospital, of infections with GAS isolates with the same *emm* type. We also compared the *emm* types of isolates responsible for clusters of postpartum infection with the *emm* types of all isolates from cases of invasive GAS infection at each ABCs site. The binomial distribution was used to assess the likelihood that isolates from 2 cases of invasive postpartum GAS infection at a single hospital would be due to the identical *emm* type. We calculated the probability that the observed frequencies of invasive postpartum GAS infection due to isolates belonging to the various *emm* types at a single hospital were due to chance alone by using the observed *emm* type distributions of all cases of GAS infection that occurred during the same time periods as the expected baseline frequencies.

RESULTS

Description of cases of invasive postpartum GAS infection. From 1995 through 2000, ABCs identified 3957 cases of invasive GAS disease. Of these, 87 (2.2%) met the case definition for invasive postpartum GAS infection; 11–20 cases were detected each year. The proportion of all cases of GAS infection that were postpartum infections was stable over time. The ages of case patients ranged from 14 to 42 years (mean, 29 years). Sixty-four percent of cases occurred in white patients and 28% in African American patients, a racial distribution similar to that for all cases of invasive GAS disease [11]. (A greater percentage of cases occur among white patients because the size of the white population is greater; however, the *rate* of invasive GAS infection is 1.6–2.0-fold greater among African Americans [11, 12].) The principal clinical syndromes manifested by case patients were bacteremia without focus, endometritis, and peritonitis (table 1).

Seventy-nine (91%) of 87 isolates were from blood samples, 4 (5%) from peritoneal fluid samples, and the remainder from surgical (2%), joint (1%), and tissue (1%) specimens. In 3 cases, isolates were recovered from multiple sterile sites. We performed *emm* typing for 73 (84%) of the isolates. The most common *emm* types were type 28 (21%), type 1 (14%), and types 4, 11, 12, and 13 (7% each); each of the remaining *emm* types accounted for $\leq$5% of the total. For comparison, 2912

Table 1. Diseases and conditions seen among patients with postpartum group A streptococcus infection, Active Bacterial Core Surveillance, 1995–2000.

Disease or condition	No. (%) of patients (n = 87)
Bacteremia without focus	40 (46)
Endometritis	24 (28)
Peritonitis	7 (8)
Septic abortion	6 (7)
Cellulitis	3 (3)
Septic arthritis	3 (3)
Necrotizing fasciitis	3 (3)
Streptococcal toxic shock syndrome	3 (3)
Chorioamnionitis	3 (3)
Pneumonia	1 (1)
Abscesses	0 (0)
Other[a]	3 (3)

[a] Meningitis, pelvic thrombophlebitis, and septic emboli.

(75%) of the 3870 isolates recovered from nonpostpartum cases of invasive GAS infection were typed, and the most common *emm* types were type 1 (20%), type 3 (9%), type 28 (8%), and type 12 (7%); each of the remaining *emm* types accounted for ⩽3% of the total.

Eighty-six (99%) of the 87 case patients with invasive postpartum GAS infection were hospitalized, and 3 died. The case-fatality rate for invasive postpartum GAS disease among infected women (3.5%) was lower than that for other invasive GAS infections (9.4%) among women of reproductive age (aged 10–50 years). Of the 3 patients who died, 1 patient had streptococcal toxic shock syndrome, septic abortion, and endometritis; 1 patient had peritonitis and endometritis; and 1 patient had bacteremia only. The majority of the women with invasive postpartum GAS infection reported having no underlying disease; 5 (6%) reported having asthma or collagen vascular disease; and none of the 3 women who died was reported to have an underlying illness. Information on fetal outcome was available for 73 women with invasive postpartum disease: 60 infants (82%) did not develop any apparent illness, 4 infants (5%) had a nonspecific clinical disease, 7 pregnancies (10%) resulted in septic abortion or stillbirth, and 2 pregnancies (3%) resulted in an induced abortion. No neonatal deaths were reported.

Estimates of the burden of invasive postpartum GAS infection. An average of 204 hospitals (range, 169–255 hospitals) were included in the ABCs GAS surveillance each year. The 87 cases of invasive postpartum GAS infection detected during the 6-year surveillance period occurred at 52 hospitals. At least 1 case of invasive postpartum GAS disease occurred in 3.5%–7.5% of all ABCs hospitals per year. The proportion of hospitals with ⩾2 cases during each calendar year was significantly lower (0.4%–1.6%).

The average number of cases of postpartum GAS infection in the ABCs surveillance areas in 1997 was 0.06 cases per 1000 live births. Given that 3.9 million live births occurred in 1997 in the United States, we estimate that ~232 cases of invasive postpartum GAS infection occurred nationwide (National Center for Health Statistics 1997 live birth data).

We also used the complete surveillance data on invasive GAS infection from 1999 to estimate the burden of invasive postpartum GAS disease. Taking into account the proportion of invasive postpartum GAS infections among all cases of invasive GAS infection in 1995–2000 (average, 2.2% of cases per year) and the national projection for the year 2000 for cases of invasive GAS infection estimated from ABCs data (9500 cases), we estimate that 209 cases (i.e., 2.2% of 9500 cases) of invasive postpartum GAS infection occur in the United States each year, which closely approximates the estimate derived from live birth data. The average of the results of the 2 methods is 220 cases of invasive postpartum GAS infection per year in the United States.

Multiple cases of postpartum GAS infection in hospitals. Among hospitals surveyed during 1995–2000, two or more cases were noted during a 6-month period at 8 hospitals, during a 12-month period at 13 hospitals, and during a 24-month period at 16 hospitals. Cases that occurred in each shorter time period were also included in the tally for longer time periods. Three or more cases occurred during 24 months at 9 hospitals; in 1 instance, 5 cases occurred during 24 months at a single hospital.

No cluster of ⩾3 cases with identical *emm* types was noted in ABCs hospitals during any 24-month period during 1995–2000. Therefore, we chose to analyze the least likelihood that the cases might be related by analyzing case pairs. Paired *emm* typing results were available for 31 (86%) of the 36 case pairs identified during the longest time period (24 months; table 2). Identical *emm* types were noted in 11%–16% of the case pairs evaluated; the most common *emm* type in case pairs was *emm* type 28. The probability of observing ⩾1 pair infected with the same *emm* type in a collection of 9 pairs was 0.33. The probability of observing ⩾3 pairs infected with the congruent *emm* type in a collection of 20 pairs was 0.095; and the probability of observing ⩾5 pairs infected with the congruent *emm* type in a collection of 31 pairs by chance alone was 0.03 (table 2). Cases of infection with identical *emm* types clustered more frequently than expected by chance, especially when these infections occurred during a 24-month period. For 2 case pairs, the infecting *emm* type corresponded to the second or third most common *emm* types among nonpostpartum cases of invasive GAS infection that occurred at the relevant ABCs site;

Table 2. Frequency and congruence of *emm* types of group A streptococcus (GAS) isolated from cases of invasive postpartum infection that occurred at Active Bacterial Core Surveillance (ABCs) hospitals during various time intervals, 1995–2000.

Period of observation, months[a]	No. of hospitals with ⩾2 cases during 24 months	No. of case pairs at all hospitals during 24 months	No. (%) of case pairs with *emm* typing data available for both cases	No. (%) of case pairs with identical *emm* types	Shared *emm* type (no. of case pairs)
6	8	11	9 (82)	1 (11)	28 (1)
12	13	23	20 (87)	3 (15)	28 (2), 11 (1)
24	16	36	31 (86)	5 (16)	28 (2), 11 (2), 13 (1)

NOTE. An average of 204 hospitals (range, 169–255 hospitals) were included in the ABCs GAS surveillance each year.

[a] The period of observation during which ⩾2 cases were noted. The pairs of cases in the 12-month period include those from the 6-month period, and the pairs of cases in the 24-month period include those from the 6-month and the 12-month periods.

the *emm* types of the remaining 29 case pairs were not among the most common *emm* types among the cases of invasive GAS infection identified in the surrounding community.

DISCUSSION

This study provides the first population-based estimate of the incidence of invasive postpartum GAS infections among women in the United States, and it is also the first systematic evaluation of the frequency of such cases across a large number of heterogeneous hospitals. We found that invasive GAS infections in the postpartum period were relatively rare: we estimated that 220 cases occur nationwide annually and determined that up to 8% of hospitals saw a single case and <2% saw multiple cases each year. According to data collected by the CDC's ABCs system during 6 years, the proportion of invasive postpartum GAS infections among all invasive GAS infections remained constant, at ~2.2%. Although rare, invasive disease due to GAS in the postpartum period resulted in death in 3.5% of cases among these otherwise healthy women.

Some of these cases of invasive postpartum GAS infection may be preventable. Reported outbreaks of invasive postpartum GAS infections have generally been associated with exposure to an infected health care worker [2–7], and there are rare instances of transmission attributed to another patient [13] or contaminated hospital equipment (e.g., a bidet [14] or a showerhead [15]). Isolates from invasive postpartum GAS infections should be saved. With use of typing methods, these isolates can be compared with isolates from later cases or from colonized health care workers to look for evidence of relatedness. In our review of the surveillance data, isolates with identical *emm* types occurred in only 11%–16% of clusters. However, probability analysis indicated that the number of pairs of infections due to the same *emm* type that occurred during a 24-month period at the same hospital was unlikely to have been due to chance alone ($P = .03$). Although it is possible that the strain that the clusters of cases had in common may be the most prevalent circulating strain in the community at that time, our analysis showed that the common strain in the clusters typically differed from the most prevalent strain circulating in the community. It is possible that an infected health care worker was involved in the transmission of some of the cases of invasive postpartum GAS infection. This raises the question of whether prompt investigation could have prevented additional infections. Some experts have proposed that an epidemiologic investigation be conducted after a single GAS case is identified [2, 16], but the cost and benefit of such evaluation have not been determined. We sought to quantify how many single and paired cases of invasive postpartum GAS infection are likely to occur within a single hospital facility. Our assessment of the burden of sporadic invasive postpartum GAS infections can serve as a reference point for hospital infection control personnel and health departments weighing decisions to investigate after identification of a single or multiple cases.

Infections are the most common cause of serious maternal morbidity [17]; postpartum infections occur in an estimated 6% of all pregnant women [18]. Postpartum infections are more frequent following cesarean section than following vaginal delivery (rates, 7.9% vs. 1.8% [17]). In contrast to group B streptococcus (GBS) infection, which causes illness and death in newborns disproportionately more often than it does in mothers, perinatal GAS infection primarily affects mothers. For example, it is estimated that, in 1998, invasive GBS disease occurred in 0.23 women per 1000 live births but in 0.6 newborns per 1000 live births [19]. The ratio of maternal to neonatal GBS infections contrasts with the ratio for GAS infections in this study: 87 invasive infections in women during 1995–2000, compared with only 15 invasive GAS infections in infants during the first month of life during the same time period.

Because our data are population based, they circumvent the traditional biases of case reports and case series, such as increased reporting of more-severe cases and selective reporting of clusters of cases in which a common strain was identified. Our estimate of invasive postpartum disease burden, based on

ABCs data (0.06 cases per 1000 births), approximates that obtained from population-based surveillance data from Ontario, Canada (0.05 cases per 1000 births [20, 21]). Both estimates are significantly lower than an estimate derived from a published hospital series (0.33–3.16 cases per 1000 births) [22], likely because of the diversity of hospitals included in the 2 active surveillance systems, the avoidance of selection bias, and the larger sample size.

All current surveillance methods likely underestimate the total burden of invasive postpartum GAS disease. ABCs detects invasive cases of disease, but most cases of invasive postpartum illness are not evaluated by means of blood or endometrial cultures [6, 15, 16]. There is increasing evidence that endometritis can be treated successfully with empiric antimicrobial agents [23], and little incentive exists for health care practitioners to obtain specimens for microbiologic evaluation from women with fever during the peripartum period.

Consistent with previous reports, we found that the most common *emm* type among isolates from both sporadic invasive postpartum infections and potentially related case pairs was *emm* type 28 [24], which is the third most common type among all cases of invasive GAS infection. A summary of 13 outbreaks of postpartum and postsurgical GAS infection found that *emm* type 28 was the epidemic strain in 7 outbreaks [25]. Of 10 reported outbreaks of postpartum GAS infection, 6 were due to a single strain traced to health care workers, and 3 of these outbreaks were caused by *emm* type 28 [3, 6, 7]. The clinical significance of the predominance of *emm* type 28 in invasive postpartum GAS infections is unknown, but the predominance may be related to tropism of this *emm* type for vaginal tissue [24].

This study supports the idea that some postpartum GAS infections that occur at a single institution, even when widely separated in time, might arise from a common source and, therefore, be preventable. Although postpartum GAS infections at a single institution may have a source in the community, even if they are due to a single clone, we propose that a conservative approach to investigating postpartum GAS infections should be taken. Postpartum GAS infections are relatively rare, and nosocomial clusters due to a single health care worker who is a carrier of GAS have been known to persist for up to 40 months [6]. The likelihood that 2 cases that occur during 24 months will be due to the same *emm* type by chance alone is very low. Therefore, we suggest that isolates from invasive postpartum GAS infections be saved for later typing to determine whether later cases are related. Conversely, the majority of cases of invasive postpartum GAS infections are isolated instances. Possible strategies for reduction of the number of invasive postpartum GAS infections include heightened awareness to identify additional cases after the occurrence of a single case and initiation of epidemiologic investigation to identify a common source after 2 cases due to the same strain have been identified.

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