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Comparison of early-onset neonatal sepsis caused by *Escherichia coli* and group B *Streptococcus*

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KEY WORDS
Early-onset neonatal sepsis
Group B *Streptococcus*
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Objective: The purpose of this study was to compare maternal characteristics and neonatal morbidity and mortality rates that are associated with early-onset neonatal sepsis that is caused by group B *Streptococcus* and *Escherichia coli*.

Study design: This was a retrospective review of newborn infants with a positive blood culture (and/or cerebrospinal fluid) that was positive for either *E* coli or group B *Streptococcus* during the first week of life. Data were abstracted from maternal and neonatal medical records.

Results: Among 28,659 deliveries during the study period, 102 episodes of early-onset neonatal sepsis were identified, 61 of which were caused by group B *Streptococcus* and 41 of which were caused by *E coli*. *E coli* sepsis cases had a lower birth weight, a higher percentage with 5-minute Apgar score <7, and a longer stay in the hospital neonatal intensive care unit and required mechanical ventilation more frequently. Death after early-onset neonatal sepsis with *E* coli was also more frequent.

Conclusion: Early-onset sepsis with *E coli* is associated with more morbidity and a higher mortality rate compared with early-onset group B *Streptococcus*.

Among very low birth weight infants, the greatest lethal risk is extreme prematurity. Early-onset sepsis (during the first week of life) fortunately is an infrequent complication, which in the past was most frequently caused by group B *Streptococcus* (GBS).

After the development and implementation of consensus guidelines for GBS prophylaxis in 1996,[1] both the frequency of and death from early-onset sepsis that is caused by this bacteria has decreased markedly. The initial guidelines recommended both a screen and a risk-based approach for intrapartum antibiotic prophylaxis. The modified 2002 guidelines recommend universal screening by vaginal and rectal GBS cultures for all pregnant women at 35 to 37 weeks of gestation.[2] In

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Table I Maternal and neonatal variables among cases with early-onset GBS and *E coli* sepsis

Variable	GBS cases (n = 61)	*E coli* cases (n = 41)	*P* value
Vaginal examination (n)*	4.8 ± 31	1.7 ± 1.4	<.01
Duration of rupture of membranes (hr)*	17.5 ± 59.6	1.3 ± 5.2	.044
Chorioamnionitis (% affected)	14.5	0	.034
Fetal scalp electrode (% used)	16.7	9.1	NS
Gestational age at delivery (wk)*	37.0 ± 4.7	32.6 ± 6.2	<.01
Birth weight (g)	3151.7 ± 1032.5	2055.9 ± 1163.3	<.01
5-Minute Apgar score <7 (%)	14.3	36.1	.04
Length of stay (d)*	18.2 ± 24.4	43.0 ± 53.5	.014
Neonatal intensive care unit (d)*	8.8 ± 25.4	32.0 ± 52.9	.021

NS, Not significant.
* Data are given as mean ± SD.

Table II Selected neonatal morbidities among cases with early-onset sepsis caused by *E coli* and GBS

Variable	GBS cases (%)	*E coli* cases (%)	*P* value
Septicemia	56.7	85.3	<.01
Pneumonia	18.3	9.1	NS
Seizure	3.4	21.9	NS
Acidosis	30.5	30.5	NS
Intraventricular hemorrhage	3.4	12.5	NS
Mechanical ventilation	13.6	53.1	<.0001

NS, Not significant.

2003, the overall disease incidence was 0.32, which represented a 34% decline in incidence since 2000 to 2001.[3]

Intrapartum antibiotic prophylaxis has been shown to be most effective to prevent GBS early-onset sepsis, regardless of which of the aforementioned strategies is used to prevent this disease. The widespread use of antibiotics has led to concern over the possible selection for other organisms and/or increased antibiotic resistance. There is a concern that early-onset sepsis could be caused by other more virulent organisms in the future.[4,5] Although Gram-positive organisms (most commonly GBS) were responsible most infections between 1991 and 1993, Gram-negative organisms (most commonly *Escherichia coli*) became the most frequent cause of early-onset sepsis between 1998 and 2000.[5,6] The objectives of this study were to compare maternal characteristics and neonatal morbidity and mortality rates that were associated with early-onset neonatal sepsis caused by *E coli* and GBS.

Study design

This was a retrospective study of early-onset neonatal sepsis cases caused by either *E coli* or GBS at Jackson Memorial Hospital between January 1, 1998, and June 30, 2002. Approval from the University of Miami's Institutional Review Board was obtained. Early-onset sepsis was defined as a positive blood or cerebrospinal fluid culture that was obtained between birth and the end of day 6 of life. Analyzed maternal variables included demographics, the number of vaginal examinations, the duration of rupture of membranes, the use of fetal scalp electrodes and/or intrauterine pressure catheters, the development of chorioamnionitis, and the gestational age at delivery. Neonatal variables included birth weight, Apgar scores at 5 minutes, length of stay, number of days in the neonatal intensive care unit, and neonatal morbidities (specifically septicemia, pneumonia, intraventricular hemorrhage, seizures, acidosis [pH <7.20], and the need for mechanical ventilation).

Statistical analyses were performed with SPSS for Windows (version 10.0; SPSS Inc, Chicago, Ill). Descriptive statistics were obtained for all variables. Continuous variables were analyzed with 2- sample *t*-test, and dichotomous variables were analyzed by chi-squared tests.

Results

There were 28,659 live births during the study period. One hundred two cases of early-onset neonatal sepsis were caused by GBS (61 cases) or *E* coli (41 cases). No significant differences were noted with respect to maternal demographics. There was no perceptible increase in the number of cases of *E coli*–induced early-onset sepsis during the era of risk-based GBS prophylaxis. A comparison of intrapartum factors between cases of early-onset GBS and *E coli* sepsis in the neonate is shown in the Table I.

Of the most common morbidities that are associated with both *E* coli and GBS early-onset neonatal sepsis, only septicemia and mechanical ventilation were significantly higher in the *E coli* group (Table II). Most importantly, the neonatal mortality rate was increased

among neonates who were diagnosed with *E coli* sepsis compared with GBS sepsis (19% vs 7%; $P < .007$).

Comment

Early-onset sepsis is a rare, but potentially lethal problem, that affects primarily neonates of low birth weight.[1] Vertical transmission of GBS during labor or delivery may result in an invasive infection in the newborn infant during the first week of life, which results in approximately 1600 cases and 80 deaths annually, according to the latest report from the Centers for Disease Control.[3] The incidence of invasive GBS infections among pregnant women in the United States decreased by 21% from 1993 to 1998 to an incidence of 0.23 per 1000 live births,[7] with a further decrease in 2003.[3] The emergence of other pathogens and an increase in the incidence of early-onset sepsis caused by *E coli* during or after this interval have been reported.[4] However, we found no increase in the number of cases of early-onset *E coli* sepsis at our institution.

There were a number of differences that were noted between cases of *E coli*– and GBS-induced early-onset sepsis, which were related mostly to intrapartum events in the GBS cases. Infants who received a diagnosis of *E coli* sepsis were born at an earlier gestational age and of lower birth weights and had a higher percentage of Apgar scores of <7 at 5 minutes than those with GBS sepsis. It follows then that these infants had a longer length of stay and a greater number of days spent in the neonatal intensive care unit. The 2 morbidities that were significantly higher in the *E coli* group were septicemia and a need for mechanical ventilation. More importantly, the neonatal mortality rate was significantly higher with *E coli* sepsis, compared with GBS sepsis. The increased mortality rate in the *E coli* sepsis group may be confounded by the higher prematurity rate among the affected neonates in our study group.

In conclusion, *E coli* sepsis occurred in a more premature patient population in comparison to GBS and was associated with higher morbidity and mortality rates. On-going surveillance of infecting organisms and antimicrobial prophylaxis that is directed at GBS must continue.

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