

Nosocomial Transmission of Group B Streptococci Proven by Positive Environmental Culture

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Abstract

Objective: Neonates usually acquire *Group B streptococcal* infection vertically from the maternal birth canal during delivery. In January 2010, a *Group B streptococcal* outbreak investigation was conducted in response to an increased number of clinical specimens from our neonatal intensive care unit.

Methods: Microbiology laboratory records were reviewed to identify *Group B streptococcal* from specimens originating from the neonatal intensive care unit during December 2009 and January 2010. Patients from whom these specimens were collected were identified and their charts reviewed. Environmental samples to screen for *Group B streptococcal* were collected from the unit, clinical and environmental isolates were compared by pulsed field gel electrophoresis. Point prevalence screening was conducted twice before declaring the outbreak over.

Results: Pulsed field gel electrophoresis patterns of three clinical strains from six patients were indistinguishable. One environmental strain was isolated from one of the patients monitor, and had identical pulsed field gel electrophoresis pattern to that of the three clinical strains. Infection control measures were implemented in the neonatal intensive care unit and follow-up point prevalence screening identified no new cases.

Conclusions: Although poor infection control practice has been implicated in previous reports of nosocomial outbreaks of *Group B streptococcal* infection in neonatal intensive care units, our finding provides unique evidence that the environment can act as a reservoir of *Group B streptococcal* and play a key role in nosocomial transmission.

Keywords: Neonates; *Group B streptococcus*; *Streptococcus agalactiae*; Outbreak; NICU; Pulsed field gel electrophoresis (PFGE); Environment.

Introduction

Group *B streptococcus* (GBS), also known as *Streptococcus agalactiae*, causes severe disease in the neonatal period associated with significant morbidity and mortality.¹ Early onset disease occurs during the first 6 days of life, while late onset can manifest any time from 7 days to 3 month of age. The frequency of early onset GBS infection has decreased substantially since the introduction of routine screening for maternal rectovaginal GBS colonization and the use of intrapartum antibiotic prophylaxis^{2,3}. However, the frequency of late onset infection, which is not affected by chemoprophylaxis, continues to cause significant morbidity and mortality in newborn.

Nosocomial outbreaks of late onset GBS infection are uncommon and have been attributed to direct and indirect infant-to-infant transmission via contaminated hands of nursery personnel.⁴⁻⁸ However, an epidemiological link between outbreak strains was not always found in reported clusters to support the theory of transmission.^{4,7,8} In this paper, we describe a genotypically-confirmed cluster of GBS infection in a level 3 neonatal intensive care unit (NICU) associated with environmental contamination by the same strain.

Methods

The NICU at the Hospital for Sick Children in Toronto, Canada is a 38-bed out-borne unit, providing tertiary medical and surgical services for neonates admitted from the greater Toronto area hospitals (figure 1). The typical census ranges from 30 to 34 patients.

On January 26th 2010, the microbiology laboratory informed Infection Prevention and Control of an increase in the number of clinical isolates of GBS from the NICU (5 unique patients isolates over 2 months). Three of the patients were still in the unit; one had been discharged and the other was deceased. Concurrently, a sixth child relocated to the referring hospital developed GBS bacteremia

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on return. Thus, including the latter case, there were six patients with GBS in this outbreak as compared to only one patient with GBS sepsis in 2009.

A case definition was established: any NICU patient from whom GBS was isolated from a clinical specimen since Dec 2009 and an outbreak investigation initiated. The clinical and laboratory records of these six patients were reviewed. Table 1 summarizes the patient characteristics and outcomes. The suspected index case (patient C) was transferred to our unit from a peripheral hospital, where the child’s respiratory secretions had previously grown GBS. The child required prolonged ventilation and frequent suctioning at our institution. Patient C shared a common room in the NICU with patients D and F, who both later developed GBS sepsis and meningitis, respectively (figure 1). Patient A died of early onset GBS sepsis; all the other patients had late onset disease. Patient E’s mother was GBS positive on antenatal screening; this child was in the unit for complex medical and surgical issues and then was transferred back to the community hospital on day 18 of life where he developed GBS sepsis on day 30 of life. In this outbreak, one child died (patient A), and another developed significant neurological sequelae (patient D) and required neurosurgical intervention for hydrocephalus.

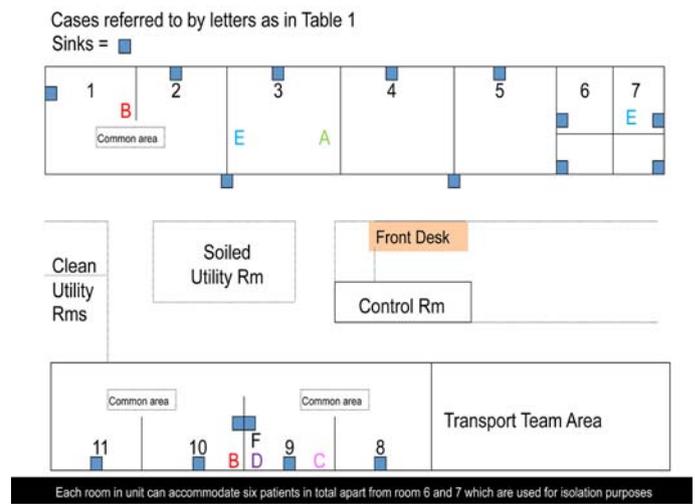


Figure 1: NICU Design and Patients Location.

Patients with GBS, who were still in the NICU, were cohort with designated staff, and contact precautions were initiated. Environmental sampling was performed in the room, where three of the patients were residing. Ten sites in each patient specific zone (monitor, keyboard, mouse, IV pump, ventilator, lamp switch, parent chair- seat and back, thermometer and crib/front rail or

Table 1: Demographic and outcome of neonates with positive GBS culture during outbreak.

Patient	GA weeks	BWT grams	Diagnosis	Admission date	Positive culture date	Age when had positive culture	Positive culture site	Discharge date	Outcome
A	25	900	Severe RDS, GBS Sepsis	December 1	December 4	4 days	Placenta	December 4	Died on day 4 of life
B	36	4366	Hypoglycemia, GBS colonization	December 14	December 24	11 days	Neck lesion and perineum	December 24	Discharged home
C	27	910	Multiple congenital anomalies, GBS pneumonia	October 27	January 10	7 months	ETT/TT	March 11	Transferred to regional hospital
D	26	987	Pneumonia, GBS sepsis and meningitis	December 20	January 19	1 month	Blood + CSF	February 8	Seizures, hydrocephalus, VP shunt was placed prior to transfer to regional hospital
E	29	1465	Isovaleric academia, Necrotizing Enterocolitis, and GBS sepsis	December 26	January 25	1 month	Blood	January 12	Transferred to regional hospital
F	32	1030	IUGR, Congenital heart disease, GBS sepsis and Meningitis	November 7	January 26	2 months	Blood + CSF	April 21	Discharge home

NOTE: Patient C, D, and F shared same room in NICU and had indistinguishable GBS isolates by PFGE

ETT/TT: Endotracheal Tube/Tracheostomy Tube; GA: Gestational Age; RDS: Respiratory Distress Syndrome; CSF: Cerebrospinal fluid; IUGR: intrauterine growth retardation, VP: Ventriculo-Peritoneal shunts

incubator opening) were sampled, as well as ten non-patient specific sites (refrigerator handle, sink rim, diaper scale, phone, infant scale, two keyboards at nursing station, room handle in/out, and rocking chair). A total of 40 environmental samples were collected and sent to the microbiology laboratory for culture; only one site, the patient monitor above (patient D), grew GBS.

Extensive environmental cleaning of the room with the infected cohort was performed. A nursing role as room monitor was implemented to specifically manage cohort room precautions, monitor hand hygiene compliance, and minimize traffic flow. Education sessions reviewing basic infection prevention and control principles including hand hygiene were held for healthcare workers in the unit.

The NICU daily and monthly censuses from October 2009 to February 2010 were reviewed as a proxy for workload. Notably, in November 2009 the NICU reached maximum capacity (35 to 36 patients) for 7 days, had an average daily census of 33 patients, and 52 new admissions. This increased to 66 new patients in December. The average daily census fell to 29-30 patients during the months of January and February 2010. When the outbreak was declared over in February, there were no days where the unit had maximum census. Unfortunately, the nurse-patient ratios for the period were not available for review.

Four of the six clinical GBS isolates and the single environmental isolate were available for genotyping. Patient A's and B's isolates were not saved, as they were cultured from non-sterile sites before the outbreak was recognized. Pulsed-field gel electrophoresis of the GBS isolates was performed as per manufacturer's protocol (BIO-RAD GenePath, Bio-Rad Laboratories (Canada) Ltd., Diagnostic Group, 2403 Guenette, Montreal, Quebec H4R 2E9). Briefly, the GBS strains were grown in PenAssay broth culture overnight and cells harvested were lysed within an agarose 'plug' with Lysozyme/Lyostaphin/Mutanolysin and then deproteinized. After a series of washes, endonuclease restriction was done using Sma I.⁹⁻¹¹ The digested DNA was then separated in a 1% agarose gel by pulse-oriented electrophoresis. DNA fingerprints generated were interpreted by standard Tenover's criteria.¹²

Results

By PFGE analysis, three clinical isolates were indistinguishable (figure 2A). All three patients, from whom those isolates were cultured, were admitted to the same room in the NICU. Patient E's GBS isolate PFGE differed from the outbreak pattern (Figure 2B); this patient had not shared a room with the other infected patients. Only one of the 40 environmental surfaces sampled, from patient D's monitor, grew GBS. This environmental strain was indistinguishable from the strains of the patients, who had been housed in the same room (Figure 2A).

Two unit wide point prevalence screens (stool and respiratory secretions if intubated) were performed in February. No new patients with GBS were identified (February 4th and 10th) and the outbreak was declared over.

PFGE using Sma1-digested chromosomal DNAs from four GBS Isolates showing indistinguishable pattern.

1 = GBS cultured from ETT secretions of suspected index case ; patient C
2 = GBS cultured from blood of patient D
3 = GBS cultured from blood of patient F
4 = GBS cultured from alarm silence button of patient C.

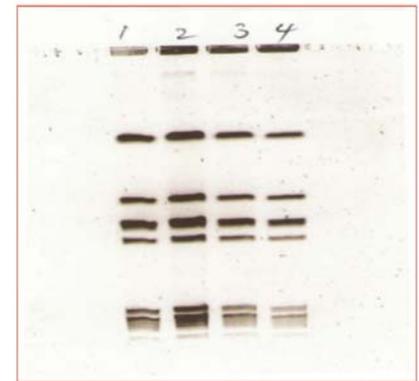


Figure 2A: Pulse-field gel electrophoresis of outbreak isolates.

- PFGE using Sma1-digested chromosomal DNAs Showing different pattern between :
1 = Outbreak GBS Isolates
2 = Patient E GBS Isolate cultured from blood sample.

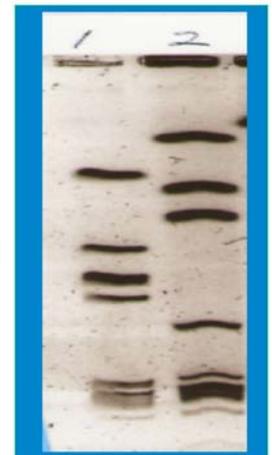


Figure 2B: Patient E GBS Isolate PFGE compared to outbreak PFGE.

Discussion

While previous reports have hypothesized poor infection control practices as the cause of late onset GBS outbreaks in neonates, this is the first to demonstrate an environmental reservoir of GBS during an outbreak. We suspect that during the course of care, a healthcare worker handled the monitor without performing proper hand hygiene and the medical device was not cleaned prior to caring for a different patient.

Environmental contamination is increasingly being appreciated as a reservoir for pathogens involved in outbreaks. Patient monitors and other high touch surfaces have been implicated as the source. Although the importance of environmental cleaning for preventing and controlling outbreaks of infection in the healthcare setting^{13,14} is appreciated, cleaning staff may be uncomfortable cleaning electronic equipment. Failure to clean properly, rather than the actual procedure or product used for cleaning, has been shown, for example, to be responsible for persistence of surface contamination with vancomycin resistant enterococci.¹⁵

This outbreak spanned the Christmas and New Year period, which is usually characterized by relative understaffing and an increase workload per health care worker. In a previous Study by Pittet et al; high workload was reported to be associated with a high risk for cross-transmission to the patients.¹⁶ It was showed that

noncompliance with hand washing was higher in intensive care than in internal medicine units, during procedures that carry a high risk for contamination (before intravenous care, before respiratory care, and care between a dirty and a clean body site), and when the intensity of patient care was high.¹⁷ The consequences of poor infection control practices are magnified in a high risk patient population (eg premature neonates who lack maternal antibody) exposed to a virulent pathogen such as GBS. Despite lack of robust measures of workload, we still posit that seasonal staffing pressures in a full NICU were important risk factors in this outbreak.

Neonatal sepsis remains the leading cause of mortality especially in the preterm babies who needs to be admitted to NICU for long time. The profile of organisms differ between different units but GBS continue to be in the top of the list.^{18,19} GBS infection in neonates carries significant morbidity and mortality. Intrapartum antibiotic prophylaxis has helped make early onset disease preventable, and our study supports the hypothesis that good infection control practices, i.e. hand hygiene and environmental cleaning can render healthcare associated late onset GBS disease preventable as well. These cornerstones of infection prevention and control remain dominant in the ongoing efforts to eliminate all hospital-associated infection.

Conclusion

Although nosocomial transmission of GBS has been implicated in previous outbreaks in newborn units, our finding supports indirect transfer of GBS from patient to patient via contaminated hands of health care workers with involvement of a contaminated environmental intermediary. Hand hygiene and environmental cleaning remain the cornerstones of infection prevention and control; therefore, it must be practiced consistently particularly in busy times.

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