from leukemia, is in concurrence with this observation (Table). However, as Schuller et al. note, malignant blood diseases and lymphoproliferative syndromes are also linked to longer hospitalizations and more frequent use of antibiotics. Although to date there have been few studies on aplasia as a risk factor for *C. difficile* infections, these are not uncommon among patients treated in oncology and hematology departments.[3] Studies concerning immunity of the host in *C. difficile* infections and their recurrences suggest that a prominent part is played by the capacity to produce an effective humoral response against toxin A.[4,5]

In our study, the marked increase in cases of diarrhea was not related to cross-infection between children because each child carried a totally different clone. The high level of genetic diversity in strains infecting patients of oncology units, both adults and children, has been reported by others.[2,6-8] This situation contrasts with that in other hospital settings, which often involve one to two predominant epidemic clones.[8] The extreme example of this is the epidemic strain known as "PCR ribotype 1," which was isolated in 58% of cases of *C. difficile* infection identified in United Kingdom hospitals, according to the United Kingdom Anaerobe Reference Unit.[9] The strict protective isolation of patients with chemotherapy-induced aplasia may play a role in preventing cross-infection with *C. difficile*. Nevertheless, oncology patients have an increased risk of coming into contact with *C. difficile* spores because they are frequently hospitalized. There is a need for longitudinal studies of the course of the infection to determine whether treatment-induced aplasia and the onset of diarrheal symptoms follow a period of asymptomatic colonization. The current study cannot support a hypothesis of endogenous origin of *C. difficile* infection, as there were no data about the children being colonized by *C. difficile* before the onset of infection. On the contrary, Shim et al. identified prior colonization as a factor protective against *C. difficile*–associated disease, although there was no mention of the immunity status of the studied populations.[10]

The absence of cross-contamination in the course of our cluster suggests the effectiveness of infection control measures in the unit. No modification in patient care was made, particularly regarding antibiotics used for enteral decontamination prior to anti-cancer chemotherapy, that could explain the increased incidence of infections during this period. Regardless of the presumed source of a case, rapid diagnosis, isolation, and sporicidal disinfection of equipment and room surfaces are necessary to limit the risk of spread. On an individual level, primary prevention of *C. difficile* infections seems difficult, as little can be done to avoid important risk factors.

The complexity of the epidemiology of nosocomial infection with *C. difficile* is the result of parameters such as the strain in question, the receptiveness of the host, and the infection control measures implemented. As we have illustrated, caution must be exercised before reaching the conclusion that an epidemic exists, particularly in oncology departments. A cluster of epidemiologically unrelated cases cannot be eliminated without the use of particularly discriminating typing techniques such as PFGE.

REFERENCES

1. Wehl G, Allerberger F, Heitger A, Meister B, Maurer K, Fink FM. Trends in infection morbidity in a pediatric oncology ward, 1986-1995. *Med Pediatr Oncol* 1999;32:336-343.
2. Schuller I, Saha V, Lin L, Kingston J, Eden T, Tabaqchali S. Investigation and management of *Clostridium difficile* colonisation in a paediatric oncology unit. *Arch Dis Child* 1995;72:219-222.
3. Gorschlüter M, Glasmacher A, Hahn C, et al. *Clostridium difficile* infection in patients with neutropenia. *Clin Infect Dis* 2001;33:786-791.
4. Kyne L, Warny M, Qamar A, Kelly CP. Asymptomatic carriage of *Clostridium difficile* and serum levels of IgG antibody against toxin A. *N Engl J Med* 2000;342:390-397.
5. Kyne L, Warny M, Qamar A, Kelly CP. Association between antibody response to toxin A and protection against recurrent *Clostridium difficile* diarrhoea. *Lancet* 2001;357:189-193.
6. Gerard M, Defesne N, Daneau D, et al. Incidence and significance of *Clostridium difficile* in hospitalized cancer patients. *Eur J Clin Microbiol Infect Dis* 1988;7:274-278.
7. Lemann F, Chambon C, Barbut F, et al. Arbitrary primed PCR rules out *Clostridium difficile* cross-infection among patients in a haematology unit. *J Hosp Infect* 1997;35:107-115.
8. Rojas S, Cohen H, Tang YJ, Wilson J, Inciardi J, Silva J Jr. Differing epidemiology of *Clostridium difficile*-associated diarrhea between an oncology ward and a general medicine ward. *Infect Control Hosp Epidemiol* 1999;20:14-15.
9. Brazier JS. Typing of *Clostridium difficile*. *Clin Microbiol Infect* 2001;7:428-431.
10. Shim JK, Johnson S, Samore MH, Bliss DZ, Gerding DN. Primary symptomless colonisation by *Clostridium difficile* and decreased risk of subsequent diarrhoea. *Lancet* 1998;351:633-636.

Celine Hernandez, MD
Olivier Meunier, MD, PhD
Strasbourg Institute of Health
Frederique Audeoud, MD
Patrick Lutz, MD, PhD
Paediatric Oncology and Haematology Department
Strasbourg University Hospitals
Michel Bientz, MD, PhD
Strasbourg Institute of Health
Strasbourg, France

Infections Due to Group B Streptococci in Neonates Are Not Associated With Higher Mortality Than Infections Due to Other Organisms

To the Editor:

Group B streptococci (*S. agalactiae*) are known to be common perinatally transmitted infectious disease agents among neonates and may cause sepsis, meningitis, or both[1] associated with substantial mortality (10% to 15%).[2] We investigated all group B streptococci infections among 246 infections in neonates hospitalized in a national referral neonatal center in Bratislava, Slovak Republic, from January 1, 1999, to January 1, 2001.

On comparison of the group of 18 neonates infected with group B streptococci with the 228 neonates infected with other organisms in univariate analysis (Epi-Info, version 2.1; Centers for Disease Control and Prevention, Atlanta, GA), the single important risk factor for group B streptococci infections was an umbilical catheter (Table). Umbilical swabs positive for group B streptococci were the only isolates associated with this type of infection. There were no other significant risk factors

TABLE
COMPARISON OF NEONATAL INFECTIONS CAUSED BY *STREPTOCOCCUS AGALACTIAE* WITH THOSE NOT CAUSED BY *STREPTOCOCCUS AGALACTIAE*

	No.	Neonatal Infections With *Streptococcus agalactiae* (%)	Neonatal Infections Without *Streptococcus agalactiae* (%)	*P*
No. of neonates	246	18	228	
Risk factors				
Gestational age, wk				
< 28	8	0 (0)	8 (3.51)	NS
28 to 32	41	2 (11.11)	39 (17.11)	NS
33 to 38	126	6 (33.33)	120 (52.63)	NS
> 38	71	10 (55.56)	61 (26.75)	.02
Birth weight, g				
< 1,000	8	0 (0)	8 (3.51)	NS
1,000 to 1,500	28	1 (5.56)	27 (11.84)	NS
1,501 to 2,500	104	6 (33.33)	98 (42.98)	NS
> 2,500	106	11 (61.11)	95 (41.67)	NS
Surgery	2	0 (0)	2 (0.88)	NS
Umbilical catheter	112	3 (16.67)	109 (47.81)	.02
Ventilatory support	37	2 (11.11)	35 (15.35)	NS
Corticoid therapy	11	2 (11.11)	9 (3.95)	NS
TPN	30	0 (0)	30 (13.16)	NS
CVC	4	0 (0)	4 (1.75)	NS
Percutaneous arterial catheter	5	0 (0)	5 (2.19)	NS
RDS	11	0 (0)	11 (4.82)	NS
Colonization of another site	7	0 (0)	7 (3.07)	NS
Mother's risk factors				
PROM	21	1 (5.56)	20 (8.77)	NS
Fever	21	2 (11.11)	19 (8.33)	NS
Colonization	29	1 (5.56)	28 (12.28)	NS
Cerclage	16	0 (0)	16 (7.02)	NS
No medical care	6	0 (0)	6 (2.63)	NS
Abortion	39	2 (11.11)	37 (16.23)	NS
Diabetes mellitus	7	0 (0)	7 (3.07)	NS
Drug abuse	8	2 (11.11)	6 (2.63)	NS
Nicotine abuse	11	0 (0)	11 (4.82)	NS
Other	5	0 (0)	5 (2.19)	NS
Type of isolate				
Blood culture/catheter tip	31	2 (11.11)	29 (12.72)	NS
Nasal swab	178	13 (72.22)	165 (72.37)	NS
Throat swab	186	16 (88.89)	170 (74.56)	NS
Ear swab	51	6 (33.33)	45 (19.74)	NS
Eye swab	28	2 (11.11)	26 (11.4)	NS
Skin swab	32	2 (11.11)	30 (13.16)	NS
Umbilical swab	91	12 (66.67)	79 (34.65)	.014
Rectal swab	7	1 (5.56)	6 (2.63)	NS
Gastric contents	57	4 (22.22)	53 (23.25)	NS
Urine culture	65	5 (27.78)	60 (26.32)	NS
Other	14	0 (0)	14 (6.14)	NS
Etiology				
Enterococcus faecalis	99	6 (33.33)	93 (40.79)	NS
Staphylococcus aureus	121	8 (44.44)	113 (49.56)	NS
Staphylococcus epidermidis	148	12 (66.67)	136 (59.65)	NS
Other *Streptococcus* species	106	10 (55.56)	96 (42.11)	NS

TABLE *(cont'd)*
COMPARISON OF NEONATAL INFECTIONS CAUSED BY *STREPTOCOCCUS AGALACTIAE* WITH THOSE NOT CAUSED BY *STREPTOCOCCUS AGALACTIAE*

	No.	Neonatal Infections With *Streptococcus agalactiae* (%)	Neonatal Infections Without *Streptococcus agalactiae* (%)	P
Acinetobacter baumannii	4	1 (5.56)	3 (1.32)	NS
Citrobacter freundii	6	1 (5.56)	5 (2.19)	NS
Escherichia coli	69	5 (27.78)	64 (28.07)	NS
Klebsiella/Enterobacter species	111	8 (44.44)	103 (45.18)	NS
Listeria species	3	0 (0)	3 (1.32)	NS
Moraxella catarrhalis	1	0 (0)	1 (0.44)	NS
Proteus species	5	0 (0)	5 (2.19)	NS
Stenotrophomonas maltophilia	32	2 (11.11)	30 (13.16)	NS
Candida albicans	2	0 (0)	2 (0.88)	NS
Candida non-*albicans*	45	2 (11.11)	43 (18.86)	NS
Haemophilus influenzae	4	0 (0)	4 (1.75)	NS
Other	2	0 (0)	2 (0.88)	NS
Diagnostic indicators				
C-reactive protein	117	11 (61.11)	106 (46.49)	NS
Procalcitonin	17	2 (11.11)	15 (6.58)	NS
Thrombocytes, < 50,000	17	0 (0)	17 (7.46)	NS
Leukocytes, < 10,000	110	7 (38.89)	103 (45.18)	NS
Leukocytes, > 30,000	9	0 (0)	9 (3.95)	NS
Septic score, > 2 points	19	0 (0)	19 (8.33)	NS
Localization of infection site				
Perinatal infection	173	16 (88.89)	157 (68.86)	NS
Bacteremia/sepsis	25	2 (11.11)	23 (10.09)	NS
Meningitis	3	0 (0)	3 (1.32)	NS
Pneumonia	91	6 (33.33)	85 (37.28)	NS
Conjunctivitis	47	3 (16.67)	44 (19.3)	NS
Thrush stomatitis	34	3 (16.67)	31 (13.6)	NS
Omphalitis	23	1 (5.56)	22 (9.65)	NS
Other	30	3 (16.67)	27 (11.84)	NS
Therapy				
Amikacin	1	0 (0)	1 (0.44)	NS
Netilmicin	41	2 (11.11)	39 (17.11)	NS
Gentamicin	80	3 (16.67)	77 (33.77)	NS
Ampicillin	6	0 (0)	6 (2.63)	NS
Penicillin	35	4 (22.22)	31 (13.6)	NS
Spiramycin + erythromycin	7	1 (5.56)	6 (2.63)	NS
Ampicillin/sulbactam	148	9 (50)	139 (60.96)	NS
Ampicillin/cloxacillin	17	3 (16.67)	14 (6.14)	NS
Amoxicillin/clavulanate	27	2 (11.11)	25 (10.96)	NS
Meropenem	11	0 (0)	11 (4.82)	NS
Ceftazidime	2	0 (0)	2 (0.88)	NS
Ceftriaxone	26	1 (5.56)	25 (10.96)	NS
Cefuroxime	13	0 (0)	13 (5.7)	NS
Cefotaxime	9	0 (0)	9 (3.95)	NS
Clindamycin	4	1 (5.56)	3 (1.32)	NS
Fluconazole + ketoconazole + clotrimazole	39	3 (16.67)	36 (15.79)	NS
Ketoconazole	21	2 (11.11)	19 (8.33)	NS
Clotrimazole	5	0 (0)	5 (2.19)	NS
Vancomycin	3	0 (0)	3 (1.32)	NS

TABLE (*cont'd*)
COMPARISON OF NEONATAL INFECTIONS CAUSED BY *STREPTOCOCCUS AGALACTIAE* WITH THOSE NOT CAUSED BY *STREPTOCOCCUS AGALACTIAE*

	No.	Neonatal Infections With *Streptococcus agalactiae* (%)	Neonatal Infections Without *Streptococcus agalactiae* (%)	P
Outcome				
Cure	239	18 (100)	221 (96.93)	NS
Death of infection	4	0 (0)	4 (1.75)	NS
Death of underlying disease	3	0 (0)	3 (1.32)	NS
PVL	2	0 (0)	2 (0.88)	NS
IVH (grades III and IV)	7	1 (5.56)	6 (2.63)	NS

NS = not significant; TPN = total parenteral nutrition; CVC = central venous catheter; RDS = respiratory distress syndrome; PROM = prolonged rupture of membrane; PVL = periventricular leukomalacia; IVH = intraventricular hemorrhage.

for group B streptococci infection related to the neonates or their mothers. Also, the outcome and complications (eg, neurologic sequelae) were similar for neonates with group B streptococci infection compared with infection caused by other organisms.

REFERENCES

1. Krcmery V, Paradisi F. Nosocomial bacterial and fungal meningitis in children: an eight year national survey reporting 101 cases. *Int J Antimicrob Agents* 2000;15:143-147.
2. Huttova M. *Infectious Diseases in Neonates.* Trnava, Slovak Republic: University of Trnava; 2001:255.

B. M. Hafed, MD, PhD
E. Bilikova, PhD
J. Koprnova, PhD
G. Kovacicova, PhD
I. Svetlansky, PhD
V. Krcmery, PhD
School of Health
Trnava University
Bratislava, Slovak Republic

https://doi.org/10.1086/503506 Published online by Cambridge University Press