American Journal of Infection Control 42 (2014) 802-4

Contents lists available at ScienceDirect

American Journal of Infection Control

journal homepage: www.ajicjournal.org

Brief report

Clostridium difficile in a children's hospital: Assessment of environmental contamination

Simone Warrack BS [a], Megan Duster BS, MT (ASCP) [a], Sarah Van Hoof RN, BSN [b], Michelle Schmitz CIC [b], Nasia Safdar MD, PhD [a,b,c,*]

[a] *Department of Medicine, University of Wisconsin Medical School, Madison, WI*
[b] *University of Wisconsin Hospitals and Clinics, Infection Control, University of Wisconsin, Madison, WI*
[c] *William S. Middleton Memorial Veterans Affairs Hospital, Madison, WI*

Key Words:
Clostridium difficile
Pediatric
Environmental contamination
Pulsed-field gel electrophoresis

Clostridium difficile infection (CDI) is the most frequent infectious cause of health care–associated diarrhea. Three cases of CDI, in children age 2, 3, and 14 years, occurred in the hematology/oncology ward of our children's hospital over 48 hours. We aimed to assess environmental contamination with *C difficile* in the shared areas of this unit, and to determine whether person-to-person transmission occurred. *C difficile* was recovered from 5 of 18 samples (28%). We compared *C difficile* isolated from each patient and the environment using pulsed-field gel electrophoresis, and found that none of the patient strains matched any of the others, and that none matched any strains recovered from the environment, suggesting that person-to-person transmission had not occurred. We found that *C difficile* was prevalent in the environment throughout shared areas of the children's hospital unit. Molecular typing to identify mechanisms of transmission is useful for devising appropriate interventions.

Copyright © 2014 by the Association for Professionals in Infection Control and Epidemiology, Inc. Published by Elsevier Inc. All rights reserved.

In the last decade, the incidence of *Clostridium difficile* infection (CDI) has increased among pediatric inpatients.[1] Furthermore, 25% of hospital-acquired (HA) CDIs among children occur in pediatric cancer patients.[2] The sources of these infections are unclear. At our freestanding university children's hospital, the Infection Control Department performs surveillance for CDI. The combined rate of HA-CDI and health care–associated (HCA) CDI for the children's hematology/oncology unit for 2012 was 20.1 per 10,000 patient-days. In comparison, the 2012 rate of HA-CDI plus HCA-CDI for the corresponding adult hematology/oncology unit was 24.2. In 2012, the pediatric unit had 9 cases of CDI, including 6 HA-CDI and 3 HCA-CDI. There were no new cases of CDI from January 1, 2013, until 3 cases were detected on February 7 (HA-CDI), February 8 (HA-CDI), and February 9 (HCA-CDI). The cluster of new cases in relation to the total number of the previous year was of concern.

Given the frequent use of shared areas, and patient and family movement in pediatric settings, we hypothesized that person-to-person transmission was likely, and that a heavily contaminated environment was a major contributor to CDI transmission. Thus, we undertook an epidemiologic study using molecular typing to characterize patient and environmental strains of *C difficile* in this cluster of cases.

METHODS

The University of Wisconsin Children's Hospital is a 61-bed facility that provides comprehensive medical and surgical care, including solid organ and bone marrow transplantation. We identified high-touch surfaces (Table 1) via communication with the hospital staff, as well as through direct observations of staff, patient, and family activities in rooms and of families and children in shared areas.

Environmental contamination was assessed by aseptically culturing high-touch surfaces using a sterile 2″ x 2″ moistened gauze pad, with processing in the Infectious Disease Research Laboratory using modifications of previously described methods.[3-6] Some surfaces in the same room were pooled together. Gram stain, catalase, and polymerase chain reaction (PCR) analysis for the enolase gene were performed to confirm *C difficile*. For PCR, 1 or 2 colonies preliminarily identified as *C difficile* were picked to tubes containing 50 μL of nuclease-free water (Promega, Madison, WI). The tubes were boiled for 10 minutes and then frozen to extract

* Address correspondence to Nasia Safdar, MD, PhD, Section of Infectious Diseases, University of Wisconsin, Madison, WI 53706.
E-mail address: Ns2@medicine.wisc.edu (N. Safdar).
Conflict of interest: None to report.

0196-6553/$36.00 - Copyright © 2014 by the Association for Professionals in Infection Control and Epidemiology, Inc. Published by Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.ajic.2014.03.008

Table 1
Results of patient and environmental testing for *C difficile*

Patient or environmental site	*C difficile* culture	*C difficile* enolase PCR	PFGE pattern
Patient 1	+	+	A
Patient 2	+	+	B
Patient 3	+	+	C
Family room/visitor lounge couch	+	+	D
Bone marrow transplant couches	+	+	E
Toy room shelves	+	+	E
Toy room strollers and crib	+	+	E
Kitchen vending machine	+	+	E

the DNA. PCR analysis specific to the *C difficile* enolase gene was performed. Primers (forward, GGAGCAATGGGAAGAGCAAT; reverse, GCTGGTTGGTCAAATGCATC) were designed by Josh Smith and obtained from IDT DNA (Coralville, IA). The reaction mixture consisted of 13 μL of nuclease-free water (Promega), 10 μL of ExTaq Premix (Takara Bio, Otsushiga, Japan), 0.5 μL of each primer (each at 20 μM), and 1.0 μL of extract. The running conditions were 5 minutes at 95°C, followed by 30 cycles of 95°C for 30 seconds, 58°C for 30 seconds, and 72°C for 30 seconds, with a final 5-minutes at 72°C. Product DNA (~179 base pairs) was amplified using an Applied Biosystems 2720 Thermal Cycler (Life Technologies, Grand Island, NY) and detected on a 1.2% FlashGel system (Lonza, Rockland, ME). Pulsed-field gel electrophoresis (PFGE) was performed on PCR-positive isolates using SmaI.[7] Each unique PFGE pattern was assigned a letter, from A to E.

RESULTS

The 3 patients who developed CDI were age 2, 3, and 14 years. All cases of CDI occurred within a 48-hour period. Two of the patients were in adjacent rooms during this period, and the third room was in a room in the same corridor, heightening suspicion for person-to-person transmission. Patient 1, age 2 years, was admitted for chemotherapy for acute lymphocytic lymphoma and bone marrow transplantation. CDI occurred at 14 days after admission and thus was classified as HA-CDI (ie, onset after 72 hours of admission). Clinically, the patient was immunocompromised, had a previous history of CDI more than 3 months earlier, and was hospitalized for 43 days. The patient was receiving ceftriaxone and vancomycin at the time of testing, and had received cefepime within the previous 30 days.

Patient 2, age 14 years, was immunocompromised and admitted for continuation of ongoing chemotherapy for neuroblastoma. He was hospitalized for 11 days, with HA-CDI diagnosed on the day of discharge. He had received sulfamethoxazole/trimethoprim and cefepime in the 30 days before this admission, but was not on antibiotic therapy at the time of testing.

Patient 3, age 3 years, was classified with HCA-CDI (ie, onset within 30 days of last discharge) based on the diagnosis made within 24 hours of the current admission and previous discharge from the same unit 5 days earlier. The reason for this admission was administration of blood products, and he was hospitalized for 4 days. He was immunocompromised owing to treatment for neuroblastoma. He had received cefepime within 30 days before this admission and was receiving vancomycin and sulfamethoxazole/trimethoprim at the time of testing.

C difficile was recovered from the stool of all 3 patients, as well as from 5 different environmental locations, including shared lounges and kitchen areas. The patients' isolates did not match any of the environmental isolates by PFGE, and did not match one another (Table 1). The following environmental sites were negative for *C difficile*: toy room: piano, computer, and phone; family room/visitor lounge: TV remote control; family room/visitor lounge: 3 computers (mice and keyboards); laundry room: 2 machines and door handle; kitchen: handles (refrigerator, cabinets, dishwasher, and microwave); common hallway vending machine; shared visitor bathroom/staff shower; clean equipment storage room: high chairs, bouncy chair; nurse station: 2 computers, 2 Meds-canners, iPad.

DISCUSSION

In our investigation of this cluster of pediatric CDI cases, we examined possible mechanisms of transmission by undertaking molecular typing of the isolates from the patients and the hospital environment. Several of our findings have implications for infection preventionists and clinicians. First, we found frequent environmental contamination in shared areas. Although this finding is not surprising, it suggests that cleaning protocols should emphasize shared areas as much as patient rooms, particularly in pediatric units.

Second, although we were concerned about the likelihood of person-to-person transmission given the temporal proximity of the cases, we found that the patient strains were not similar to one another, suggesting that person-to-person transmission is not a likely explanation for the cluster. This finding was important to reassure providers, who were already compliant with the highest standards of hand hygiene and personal protective equipment use, that the cluster was not the result of major breaches in infection control practices.

Third, we found that although environmental contamination was frequent, these strains were not related to the patient strains from the cluster under investigation. This finding does not preclude the possibility that the environmental contamination might have arisen from other patients, asymptomatic or symptomatic, who were not part of the cluster and from whom samples were not collected.

Fourth, given the unique environment of a children's hospital, where interaction and use of shared areas is much more frequent than in health care institutions for adults, careful attention should be given to determining which surfaces to sample. We found that discussions with staff, as well as direct observations of activities in shared areas, allowed us to develop a customized list of environmental surfaces to sample.

Our data add to the literature on CDI outbreaks in children's hospitals. In a previous study of spatial and temporal analysis of CDI in pediatric patients, Rexach et al[8] found that fingerprint analysis identified 4 clusters with indistinguishable banding patterns on 2 of the 4 wards under study. Bustinza et al[9] reported a clinical outbreak of nosocomial diarrhea due to *C difficile* that occurred in the pediatric intensive care unit of a tertiary care hospital with 1700 beds, 150 of them in the pediatric ward. Three patients developed CDI over a period of 15 days. Molecular typing revealed 2 different ribotypes, indicating that this was not a point source outbreak.[9] Our findings were similar, suggesting the need for additional study to characterize the reservoirs of *C difficile* and identify the transmission dynamics of this important nosocomial pathogen.

Our study has some limitations. First, we did not sample patient rooms, because our main focus was on understanding transmission, and thus we limited our sampling to shared areas. Furthermore, we routinely perform fluorescent dye environmental sampling on high-touch surface areas in patient rooms and maintained a high rate of compliance with standard cleaning procedures, and thus we concentrated our investigation on contamination in shared areas. Second, we did not time our sample

دانلود مقاله

http://daneshyari.com/article/2636978


daneshyari
دانشیاری

- ✓ امکان دانلود نسخه تمام متن مقالات انگلیسی
- ✓ امکان دانلود نسخه ترجمه شده مقالات
- ✓ پذیرش سفارش ترجمه تخصصی
- ✓ امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
- ✓ امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
- ✓ دانلود فوری مقاله پس از پرداخت آنلاین
- ✓ پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات