documents. New York: Columbia University, **1994**.
7. Mendelson J, Jones RT. Clinical and pharmacological evaluation of buprenorphine and naloxone combination: why the 4:1 ratio for treatment. Drug Alcohol Depend **2003**; 70(Suppl 1): 29–37.
8. US Food and Drug Administration. Subutex and Suboxone approved to treat opiate dependence. Available at: http://www.fda.gov/bbs/topics/ANSWERS/2002/ANS01165.html. Accessed 10 December 2007.
9. Koch AL, Arfken CL, Schuster CR. Characteristics of US substance abuse treatment facilities adopting buprenorphine in its initial stage of availability. Drug Alcohol Depend **2006**; 83: 274–8.

Reprints or correspondence: Dr. Hannah Cooper, Behavioral Sciences and Health Education, Rollins School of Public Health, Emory University, 1518 Clifton Rd. NE, Rm. 568, Atlanta, GA 30322 (hannah.cooper@sph.emory.edu).

Clinical Infectious Diseases 2008; 46:955–6
 1058-4838/2008/4606-0034$15.00
DOI: 10.1086/528870

Recurrent Infection with Epidemic *Clostridium difficile* in a Peripartum Woman Whose Infant Was Asymptomatically Colonized with the Same Strain

To the Editor—Recent outbreaks of *Clostridium difficile*-associated disease (CDAD) have been attributed to the emergence of an epidemic strain, termed North American PFGE type 1, which produces binary toxin and has a genetic deletion associated with increased toxin production [1]. There have also been recent reports of severe CDAD in low-risk populations, including peripartum women [2]. In 2 cases, peripartum women possibly transmitted *C. difficile* to their children [2]. We report a case of recurrent CDAD due to the epidemic strain in a peripartum woman whose baby was an asymptomatic carrier of the same strain.

A 19-year-old previously healthy woman delivered a baby and was discharged from the hospital 2 days later. She had received azithromycin 6 months earlier but had received no other antibiotics. Diarrhea developed 10 days after delivery, and CDAD was diagnosed on the basis of a positive toxin enzyme immunoassay result. The patient's symptoms resolved with oral metronidazole, but she subsequently developed 3 recurrences that were treated with oral vancomycin for 10 days, vancomycin taper for 6 weeks, and oral nitazoxanide for 10 days, respectively. Her baby remained healthy with no diarrhea.

To investigate whether the baby could be a potential source for re-exposure of the mother, we cultured stool samples obtained from the mother at the time of her third relapse and obtained concurrently from the baby. *C. difficile* isolates were tested for in vitro cytotoxin production and were analyzed for binary toxin gene *cdtB* and partial deletions of the *tcdC* gene, as described elsewhere [3]. Molecular typing was performed using PCR ribotyping [4]. To assess whether the epidemic strain might be circulating on the newborn unit, we performed cultures and typing of stool samples obtained from healthy babies on the neonatal unit and from environmental sites.

Both the mother and the baby carried the epidemic *C. difficile* strain (figure 1). The baby's stool sample contained 6 $\log_{10}$ colony-forming units of *C. difficile* per g of stool. The mother was instructed to perform careful hand washing after changing diapers and to use 10% bleach for surface disinfection. No further recurrences occurred. On the healthy-baby unit, 10 (50%) of 20 stool samples obtained from newborns and 4 (17%) of 24 environmental cultures were positive for *C. difficile*, but none of the isolates were of the epidemic strain.

In summary, we report a case of recurrent CDAD attributable to an epidemic strain in a peripartum woman whose baby carried the same strain asymptomatically. It is not known whether the mother acquired the strain and transmitted it to her baby or vice versa, and the original source of the epidemic strain is unclear. Nevertheless, it is plausible that the baby contributed to the mother's recurrences by providing a source of repeated exposure to *C. difficile* during activities such as diaper changing. McFarland et al. [5] previously reported 5 cases of recurrent CDAD in peripartum women; in 2 cases, the babies carried the same strain as their mothers. CDAD should be considered to be a possible cause of diarrhea in peripartum women, even in the absence of recent antibiotic therapy. Asymptomatically colonized babies have the potential to serve as reservoirs for transmission of North American PFGE type 1 strains.

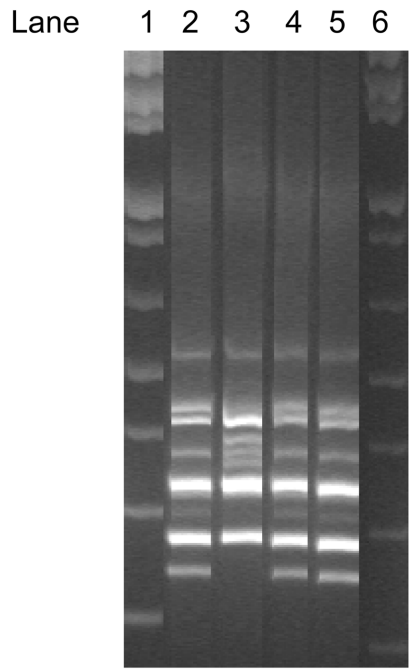

Figure 1. PCR ribotyping results demonstrating carriage of identical epidemic *Clostridium difficile* isolates in stool samples of a peripartum woman with recurrent *C. difficile* infection and her asymptomatic baby. The epidemic control strain and the isolates obtained from the mother and the baby had PCR amplification results positive for binary toxin gene *cdtB* and partial deletions of the *tcdC* gene, whereas the nonepidemic control strain did not. *Lanes 1* and *6,* 1 kb plus ladder; *lane 2,* epidemic control strain (restriction enzyme analysis type BI6, courtesy of Dale Gerding); *lane 3,* nonepidemic control strain (restriction enzyme analysis type J29 or 30); *lane 4,* isolate from the mother; *lane 5,* isolate from the baby.

Acknowledgments

Financial support. Advanced Career Development Award from the Department of Veterans Affairs (to C.J.D).

Potential conflicts of interest. C.J.D. has received research support from Ortho-McNeil, Elan,

Merck, IPSAT Therapies, ViroPharma, Astra-Zeneca, and Optimer and is a member of the speakers' bureau of Ortho-McNeil. All other authors: no conflicts.

Michelle T. Hecker,[1] Michelle M. Riggs,[2] Claudia K. Hoyen,[3] Christina Lancioni,[3] and Curtis J. Donskey[1]

[1]Infectious Diseases Department, MetroHealth Medical Center, and [2]Infectious Disease Section, Louis Stokes Cleveland Veterans Affairs Medical Center, and [3]Department of Infectious Diseases, Rainbow Babies and Children's Hospital, Cleveland, Ohio

References

1. McDonald LC, Killgore GE, Thompson A, et al. An epidemic, toxin gene-variant strain of *Clostridium difficile*. N Engl J Med **2005**; 353: 2433–41.
2. Centers for Disease Control and Prevention (CDC). Severe *Clostridium difficile*–associated disease in populations previously at low risk—four states, 2005. MMWR Morb Mortal Wkly Rep **2005**; 54:1201–5.
3. Riggs MM, Sethi AK, Zabarsky TF, Eckstein EC, Jump RL, Donskey CJ. Asymptomatic carriers are a potential source for transmission of epidemic and non-epidemic *Clostridium difficile* strains among long-term care facility residents. Clin Infect Dis **2007**; 45:992–8.
4. Bidet P, Lalande V, Salauze B, et al. Comparison of PCR-ribotyping, arbitrarily primed PCR, and pulsed-field gel electrophoresis for typing *Clostridium difficile*. J Clin Microbiol **2000**; 38: 2484–7.
5. McFarland LV, Surawicz CM, Greenberg RN, Bowen KE, Melcher SA, Mulligan ME. Possible role of cross-transmission between neonates and mothers with recurrent *Clostridium difficile* infections. J Pediatr Gastroenterol Nutr **2000**; 31:220–31.

Reprints or correspondence: Dr. Curtis J. Donskey, Research Service, Louis Stokes Cleveland Veterans Affairs Medical Center, 10701 East Blvd., Cleveland, Ohio 44106 (curtisd123@yahoo.com).

Clinical Infectious Diseases 2008; 46:956–7

1058-4838/2008/4606-0035$15.00
DOI: 10.1086/527568

Initial Hospitalization and Adherence to Highly Active Antiretroviral Therapy

To the Editor—We read with great interest the article by Mariana Lazo et al. [1] regarding factors influencing adherence to HAART. We would like to add to the ongoing adherence debate by describing our own experience, which examines the importance of 1 additional factor, hospitalization [2, 3] at the time of starting HAART.

We reviewed data and compared virological success after 12 months of therapy among all HAART-naive patients with no resistance to antiretroviral agents who started HAART while hospitalized with that among patients who started HAART at our outpatient clinic from January 2004 through January 2006. Adherence was assessed through medical outcomes study questionnaires administered to all patients at 6 and 12 months after initiation of therapy and through pharmacy refill data. Twenty-one patients were hospitalized (group 1), and there were 76 outpatients (group 2). Group 1 was composed of 15 men and 6 women; 15 white persons, 5 African black persons, and 1 Indian person; 10 heterosexual persons, 9 men who have sex with men, and 3 injection drug users; the median age was 41.4 years (range, 28–55 years). All 21 patients had AIDS. The mean CD4 cell count at initiation of HAART was 75 cells/μL (range, 10–298 cells/μL), and the mean HIV RNA level was 283,960 copies/mL (range, 3984 to >500,000 copies/mL). All patients received either zidovudine plus lamivudine or tenofovir plus emtricitabine. Fifteen patients were given a protease inhibitor–based regimen (lopinavir plus ritonavir or fosamprenavir), and 6 were given a nonnucleoside reverse-transcriptase inhibitor–based regimen (2 received nevirapine, and 4 received efavirenz). After discharge from the hospital (mean duration of hospitalization, 24 days), the patients were observed as outpatients.

Group 2 was composed of 40 men and 36 women; 55 white persons and 21 African black persons; and 45 heterosexual persons, 16 men who have sex with men, and 15 previous injection drug users; the median age was 39 years (range, 20–63 years). None of the patients in group 2 had ever experienced serious complications, tumors, or opportunistic infections requiring hospitalization. HAART was started because of low CD4 cell count. The mean CD4 cell count was 220 cells/μL (range, 185–299 cells/μL), and the mean HIV RNA level was 60,615 copies/mL (521–458,109 copies/mL). Thirty-eight patients initiated a nonnucleoside reverse-transcriptase inhibitor–based regimen, 28 initiated a protease inhibitor–based regimen, and 10 received a triple nucleoside reverse-transcriptase inhibitor combination.

Results are shown in table 1 and clearly indicate a far better adherence to HAART among initially hospitalized patients that among patients who initiated HAART as outpatients. All initially hospitalized patients achieved undetectable HIV RNA levels 8–36 weeks after initiation of therapy and maintained undetectable levels at 1 year after initation of therapy. In contrast, only 48 outpatients achieved viral

Table 1. Adherence to HAART among hospitalized patients, compared with outpatients.

Variable	Hospitalized patients	Outpatients
At baseline		
CD4 cell count, cells/μL	75	220
HIV RNA level, copies/mL	283,960	60,615
At 6 months		
CD4 cell count, cells/μL	212	232
HIV RNA level, copies/mL	<50	4697
At 12 months		
CD4 cell count, cells/μL	275	288
HIV RNA level, copies/mL	<50	13,146
No. (%) of patients who were fully adherent to HAART	21 (100)	48 (63)

NOTE. Data are mean values, unless otherwise indicated.