

On the basis of our findings, we currently screen patients admitted from the nursing home from which the first 2 CPKP cases were identified. Regarding limitations, we may have underestimated CPKP incidence as a result of the small sample size and by not screening urine in those nursing home residents with chronic bladder catheters in place or screening wounds in those residents with chronic skin breakdown.

In summary, we found that patients with CPKP infection admitted from community and nursing home settings often had low functional status, chronic neurologic disease, immunosuppression, chronic infection, recent antibiotic exposure, recent hospitalization, and previous multidrug-resistant bacterial infection. These characteristics may help in identifying a population for targeted screening if nonendemic hospitals observe large numbers of patients with CPKP infection admitted from nursing homes or the community.

ACKNOWLEDGMENTS

We thank Cindy Charron, RN, for assistance in confirming cases of carbapenemase-producing *Klebsiella pneumoniae* infection at the Rhode Island Department of Health.

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article.

John Mills, MD;¹ Kimberle Chapin, MD;^{1,2,3,4}
Sarah Andrea, BS;³ Gary Furtado, MS;³
Leonard Mermel, DO, ScM^{1,2}

Affiliations: 1. Department of Medicine, Rhode Island Hospital and Warren Alpert Medical School of Brown University, Providence, Rhode Island; 2. Division of Infectious Diseases, Rhode Island Hospital, Providence, Rhode Island; 3. Department of Pathology, Rhode Island Hospital, Providence, Rhode Island; 4. Department of Pathology, Warren Alpert Medical School of Brown University, Providence, Rhode Island.

Address correspondence to Leonard Mermel, DO, ScM, Division of Infectious Diseases, Rhode Island Hospital, 593 Eddy Street, Providence, RI 02903 (lmermel@lifespan.org).

Infect Control Hosp Epidemiol 2011;32(6):629–631

© 2011 by The Society for Healthcare Epidemiology of America. All rights reserved. 0899-823X/2011/3206-0018\$15.00. DOI: 10.1086/660202

REFERENCES

1. Nordmann P, Cuzon G, Nass T. The real threat of *Klebsiella pneumoniae* carbapenemase-producing bacteria. *Lancet Infect Dis* 2009;9:228–236.
2. Ben-David D, Maor Y, Keller N, et al. Potential role of active surveillance in the control of a hospital-wide outbreak of carbapenem-resistant *Klebsiella pneumoniae* infection. *Infect Control Hosp Epidemiol* 2010;31:620–626.
3. Patel G, Huprikar S, Factor S, Jenkins S, Calfee D. Outcomes of carbapenem-resistant *Klebsiella pneumoniae* infection and the impact of antimicrobial and adjunctive therapies. *Infect Control Hosp Epidemiol* 2008;29:1099–1106.
4. Centers for Disease Control and Prevention. Laboratory protocol for detection of carbapenem-resistant or carbapenemase-producing, *Klebsiella* spp. and *E. coli* from rectal swabs. Atlanta:

Centers for Disease Control and Prevention, 2009. http://www.cdc.gov/ncidod/dhqp/pdf/ar/Klebsiella_or_Ecoli.pdf. Accessed April 9, 2010.

5. Centers for Disease Control and Prevention. Guidance for control of infections with carbapenem-resistant or carbapenemase-producing Enterobacteriaceae in acute care facilities. *MMWR Morb Mortal Wkly Rep* 2009;58:256–260.
6. Munoz-Price L, Hayden M, Lolans K, et al. Successful control of an outbreak of *Klebsiella pneumoniae* carbapenemase-producing *K. pneumoniae* at a long-term acute care hospital. *Infect Control Hosp Epidemiol* 2010;31:341–347.
7. Falagas M, Rafailidis P, Kofteridis D, et al. Risk factors of carbapenem-resistant *Klebsiella pneumoniae* infections: a matched case-control study. *J Antimicrob Chemother* 2007;60:1124–1130.
8. Hussein K, Sprecher H, Mashiach T, Oren I, Kassis I, Finkelstein R. Carbapenem resistance among *Klebsiella pneumoniae* isolates: risk factors, molecular isolates, and susceptibility patterns. *Infect Control Hosp Epidemiol* 2009;30:666–671.
9. O’Fallon E, Schreiber R, Kandel R, D’Agata E. Multidrug-resistant gram-negative bacteria at a long-term care facility: assessment of residents, healthcare workers, and inanimate surfaces. *Infect Control Hosp Epidemiol* 2009;30:1172–1179.

An Evaluation of the Impact of a Single-Dose Intravenous Immunoglobulin Regimen in the Treatment of *Clostridium difficile* Infections

To the Editor—*Clostridium difficile* infection (CDI), which produces a spectrum of clinical symptoms ranging from uncomplicated diarrhea to severe life-threatening pseudomembranous colitis, is a growing concern due to significant morbidity and additional hospital costs.^{1,2} In recent years, CDI has been shown to be associated with increased severity and mortality when linked to a new hypervirulent strain referred to as PCR (polymerase chain reaction) ribotype 027.² Recently, the presence of low serum antibody levels to *C. difficile* toxin A has been reported as a risk factor for developing CDI.^{3,4} Failure to mount an adequate immune response to *C. difficile* toxins has been identified as a critical factor in predisposing patients to severe, prolonged, and recurrent *C. difficile* diarrhea.^{3,5} However, there is no consensus on the immunoglobulin regimen to be followed (ie, dose and duration of treatment) when treating patients with CDI.^{6,7} The objective of our research was to assess the impact of a new hospital treatment policy involving the administration of a single dose of intravenous immunoglobulin (400 mg/kg) on the following patient outcomes: (1) length of stay in the hospital until discharge following the first positive CDI toxin test result, (2) 30-day clinical outcomes (recovered/recovering, ongoing infection), and (3) requirement for surgery.

Our retrospective work was performed as a part of an outbreak investigation that has been comprehensively de-

TABLE 1. Clinical Outcomes for Case Patients Compared with Matched Controls over the Study Period (June 2007–August 2008)

Variable	No. of patients (%)		P value ^a
	Case patients (n = 18)	Control patients (n = 18)	
Median hospital LOS from first positive CDI toxin test result until discharge (days)	36 (19–68)	33 (12–52)	.779
30-day clinical outcomes			
Recovered/recovering	15 (83)	15 (83)	.999
Ongoing infection	3 (17)	3 (17)	.999
Patient required surgery during this episode	0	0	NA

NOTE. CDI, *Clostridium difficile* infection; LOS, length of stay; NA, not applicable.

^a $P < .05$ was considered statistically significant.

scribed elsewhere⁸ and that was registered with the Trust clinical governance department. The study was conducted in the Northern Health and Social Care Trust in Northern Ireland, which serves a population of 450,000 people. Definitions for CDI cases, severity of *C. difficile*, and a description of the procedure for identifying and typing *C. difficile* isolates are detailed elsewhere.⁸ The study was case-control in design, and it involved collecting data retrospectively for *C. difficile*-infected patients over the outbreak period (June 2007–August 2008). Cases involved patients who received a combination of the standard treatment (ie, metronidazole and/or vancomycin) and intravenous immunoglobulin treatment (single doses of 400 mg/kg given in severe cases or in the event of no clinical response to standard treatment), and controls were patients who received only the standard treatment. At the first stage, the number of completed accessible records for patients with CDI (who were followed up from admission until their hospital discharge) was determined; this generated a list of 18 case patients and 122 potential controls. The second stage of the study involved matching case patients with suitable controls, using the following matching criteria: age, gender, comorbidity score (calculated using the Charlson Index),⁹ treatment with probiotics, and clinical severity of CDI at the date of positive toxin test result. In order to minimise bias, controls were chosen on the basis of the alphabetical order of their surnames. Means for differences between case patients and controls in relation to total length of hospital stay were compared using the paired samples *t* test, as within-paired difference data were normally distributed. McNemar's test was used to compare categorical variables. All tests were performed using SPSS, version 17, for Windows.

For the 18 case patients and 18 controls, the median age was 84 and 83 years, respectively, and 22% of both case patients and controls were male. The median comorbidity score for both case patients and controls was 1. A total of 78% of case patients and 83% of controls were treated with probiotics. For 17% of case patients and controls, illness was severe at the date of positive toxin test result. The median length

of stay in the hospital until discharge following the first positive CDI toxin test result was 36 days for case patients compared with 33 days for controls; this difference was not statistically significant ($P = .779$; Table 1). No statistically significant differences were observed in relation to the other studied outcomes (Table 1).

Management of patients with *C. difficile* infection requires treatment with metronidazole as a first-line therapy, with vancomycin being reserved for severe cases. Although the use of these treatments has been shown to reduce morbidity and mortality from CDI, treatment failures with metronidazole appear to be increasing.^{4,6} In addition, 15%–30% of patients with an initial CDI episode that was treated successfully will experience a relapse in symptoms.^{4,6} Thus, treatment of *C. difficile*-infected patients remains challenging, and this highlights the need to develop new therapeutic approaches for the management of CDI. A comparison of the characteristics of patients included in this research project showed that both case patients and controls were comparable in terms of the matching criteria, that is, age, gender, comorbidity score, treatment with probiotics, and clinical severity of CDI at the date of positive toxin test result. The findings of this study showed no statistically significant differences between the studied patients' outcomes, that is, length of hospital stay, 30-day clinical outcomes, and surgery requirement. A possible explanation for the latter findings could be the small sample size utilized; however, the matching of several confounding factors would help to minimize its impact. Another explanation could be related to the immunoglobulin regimen (ie, dose and duration of treatment) employed. A limited number of studies have been conducted to address the use of intravenous immunoglobulin for the treatment of CDI.⁷ Our results confirm the findings of the systematic review⁷ in that there is a lack of evidence to support the use of intravenous immunoglobulin for CDI. The results of this research highlight the need for further research, which should aim at measuring serum antibody levels to *C. difficile* toxin A and then defining an effective intravenous immunoglobulin therapy course for the management of CDI.

ACKNOWLEDGMENTS

Potential conflicts of interest. The authors report no conflicts of interest relevant to this article.

Mamoon A. Aldeyab, PhD;¹ James C. McElnay, PhD;¹
 Michael G. Scott, PhD;²
 Elizabeth Davies, MB, BCH, BAO, FRCPath;²
 Collette Edwards, BSc;² Feras W. Darwish Elhajji, MSc;¹
 Geraldine Conlon, MSc;² Fidelma A. Magee, BSc;²
 Paul J. Barr, PhD;¹
 Mary P. Kearney, MB, BCH, BAO, FRCPath²

Affiliations: 1. Clinical and Practice Research Group, School of Pharmacy, Queen's University Belfast, BT9 7BL Belfast, Northern Ireland, United Kingdom; 2. Northern Health and Social Care Trust, Ballymena BT43 6DA, Northern Ireland, United Kingdom.

Address correspondence to Dr. Mamoon Aldeyab PhD, Clinical and Practice Research Group, School of Pharmacy, Queen's University Belfast, BT9 7BL Belfast, Northern Ireland, United Kingdom (m.aldeyab@qub.ac.uk).
Infect Control Hosp Epidemiol 2011;32(6):631-633

© 2011 by The Society for Healthcare Epidemiology of America. All rights reserved. 0899-823X/2011/3206-0019\$15.00. DOI: 10.1086/660203

REFERENCES

1. Thompson I. *Clostridium difficile*-associated disease: update and focus on non-antibiotic strategies. *Age Ageing* 2008;37:14-18.
2. McFarland LV. Update on the changing epidemiology of *Clostridium difficile*-associated disease. *Nat Clin Pract Gastroenterol Hepatol* 2008;5:40-48.
3. 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.
4. Leffler DA, Lamont JT. Treatment of *Clostridium difficile*-associated disease. *Gastroenterology* 2009;136:1899-1912.
5. Gerding DN, Muto CA, Owens RC Jr. Treatment of *Clostridium difficile* infection. *Clin Infect Dis* 2008;46(suppl)1:S32-S42.
6. Halsey J. Current and future treatment modalities for *Clostridium difficile*-associated disease. *Am J Health Syst Pharm* 2008;65:705-715.
7. O'Horo J, Safdar N. The role of immunoglobulin for the treatment of *Clostridium difficile* infection: a systematic review. *Int J Infect Dis* 2009;13:663-667.
8. Aldeyab MA, Devine MJ, Flanagan P, et al. Multi-hospital outbreak of *Clostridium difficile* ribotype 027 infection: epidemiology and analysis of control measures. *Infect Control Hosp Epidemiol* 2011;32(3):210-219.
9. Tobacman JK. Assessment of comorbidity: a review. *Clin Perform Qual Health Care* 1994;2:23-32.

Prevalence and Type of Microorganisms Isolated from House Staff's Mobile Phones before and after Alcohol Cleaning

To the Editor—Mobile phones may pose a risk for the trans-

mission of multidrug-resistant bacteria from healthcare workers to patients, with evidence of phones as sources of contamination with *Staphylococcus aureus* and several gram-negative bacilli.¹⁻⁵ We report findings of a pilot study to estimate the prevalence and type of microorganisms isolated from the mobile phones of house staff at a Thai hospital before and after alcohol cleansing.

From August 1 to September 30, 2010, swab cultures were obtained from the mobile phones of house staff at Thummasat University Hospital. After consent, the surface of the phone's keypad, mouthpiece, and earpiece was swabbed in a standardized method. The phone was then cleaned with a 70% alcohol pad, and a second culture swab of the keypad, mouthpiece, and earpiece was obtained 1 minute later. *Same-day specimen transport to and processing at the microbiology laboratory of Thummasat University Hospital occurred, with identification of microorganisms according to Clinical and Laboratory Standards Institute criteria.*⁶ Data collection included participants' occupation, hospital unit, number of patients per unit infected with multidrug-resistant microorganisms that each house staff took care of, and the type of microorganism isolated from each house staff's mobile phone. Data on 5 moments of hand hygiene adherence were recorded from the Infection Control Unit as overall adherence in each unit that each house staff worked on at the time of specimen procurement.

There were 80 employed house staff during the study period, and all consented to study participation. The median age was 28 years (range, 24-33 years); 38 participants (47.5%) had exposure to multidrug-resistant bacteria at enrollment, and there was a median of 2 cases (range, 0-5) per house staff with multidrug-resistant bacteria. Participant characteristics and the overall 5-moment hand hygiene adherence stratified by the hospital unit are summarized in Table 1. Three mobile phones (3.8%) had cultures positive for *Acinetobacter* spp. before alcohol cleaning. After alcohol cleansing, no microorganisms were detected. Overall hand hygiene compliance was 39.0% before touching a patient, 29.4% before a clean/aseptic procedure, and 47.5% after touching a patient's surrounding.

Our study is the first to suggest that alcohol pad cleaning can eradicate microorganisms from mobile phones. Although previous reports identified healthcare workers' mobile phones as a reservoir for various multidrug-resistant bacteria, none to date have shown that alcohol cleansing can reduce the detection of bacteria on mobile phones.¹⁻⁵ Notably, overall 5-moment hand hygiene adherence was suboptimal. We acknowledge that we did not distinguish mobile phones by type or structure or evaluate potential behavioral distinctions of the house staff who did and did not have contaminated phones. Nonetheless, these findings suggest a potential environmental and behavioral risk for the transmission of microorganisms to mobile phones via patient-provider encounters. Additionally, our findings support the potential benefit