HAIs are not reported. For the 3 major body sites (blood, lungs, and urinary tract), the great majority of HAIs were in fact device associated. However, tracheobronchitis was almost as common as VAP. Importantly, more than 20% of HAIs fell into the "other" category. Of interest, the rate of CDIs was quite low, despite the frequent use of antibiotics in this patient population. The overall rate of HAIs among our patients was 7.56 per 1,000 patient-days.

Our data demonstrated that the most common pathogens were *P. aeruginosa*, *Acintetobacter* spp., and *S. aureus*. Our frequency of infections due to *Acinetobacter* spp. was elevated in the study time period by an outbreak due to a clonal strain of *Acinetobacter*. Burn centers in Turkey,[7] China,[8] and Bulgaria[9] have reported the same top organisms comprising the top 3 pathogens in burn patients. As with our bacterial strains, a high frequency of MDR strains has been reported for *S. aureus*, *Enterococcus* spp., *P. aeruginosa*, and *Acinetobacter* spp.[10]

In conclusion, infections in our burn ICU were lower than the mean rates reported by NHSN. Most major site infections are device associated. Infections due to *C. difficile* are uncommon. Nonfermentative gram-negative bacilli constitute a large proportion of HAIs. MDR pathogens are common in this patient population. Additional analyses of our HAIs in our burn population are currently under way to further evaluate the interventions that have led to our low rate of HAIs and determine the risk factors for specific HAIs.

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**David J. Weber, MD, MPH;**[1,2] **David van Duin, MD, PhD;**[1] **Lauren M. DiBiase, MS;**[2] **Charles Scott Hultman, MD;**[3] **Samuel W. Jones, MD;**[4] **Anne M. Lachiewicz, MD, MPH;**[1] **Emily E. Sickbert-Bennett, PhD;**[1,2] **Rebecca H. Brooks, RN, BSN;**[2] **Bruce A. Cairns, MD, PhD;**[4] **William A. Rutala, PhD, MPH**[1,2]

Affiliations: 1. Division of Infectious Diseases, University of North Carolina School of Medicine, Chapel Hill, North Carolina; 2. Department of Hospital Epidemiology, University of North Carolina Health Care, Chapel Hill, North Carolina; 3. Division of Plastic Surgery, University of North Carolina at Chapel Hill, North Carolina; 4. Department of Surgery, University of North Carolina at Chapel Hill, North Carolina; and North Carolina Jaycee Burn Center, Chapel Hill, North Carolina.

Address correspondence to David J. Weber, MD, MPH, 2163 Bioinformatics, CB #7030, Chapel Hill, NC 27599-7030 (dweber@unch.unc.edu).

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# Clinical and Molecular Characteristics of an Outbreak Caused by the Pandemic (BI/NAP1/027) *Clostridium difficile* Clone in a Single Center in Israel

*Clostridium difficile* is the most common infectious cause of antibiotic-associated diarrhea and healthcare-related infection in the developed world.[1] Outbreaks of severe *C. difficile* infection (CDI) have been increasingly reported in North America since 2003,[2] later in Europe and Latin America. These outbreaks coincided with the emergence of a hypervirulent new strain of *C. difficile*, designated by various typing methods as BI/NAP1/027.[2,3]

To date, only several cases have been reported from Asia and Australia.[4] In the Middle East, isolated cases have been described in Saudi Arabia[5] and Israel.[6] We recently experienced an increase in the incidence of *C. difficile*–associated

TABLE 1. Comparison of Clinical and Microbiological Variables between gc8 (BI/NAP1/027)–Infected Patients and Those with Infections Caused by Other Strains

| | gc8 ($n$ = 48) | Non-gc8[a] ($n$ = 13) | $P$ |
|---|---|---|---|
| Age, mean (range), years | 81.3 (55–95) | 70.5 (20–96) | NS |
| CCI, mean (median) | 7.3 (7.5) | 5.7 (7) | NS |
| Albumin <2 g/dL[b] | 16 (39) | 2 (20) | NS |
| Creatinine >1.5 mg/dL | 21 (44) | 4 (31) | NS |
| WBC count, mean (median), cells/$\mu$L | 22,408 (19,300) | 11,592 (11,000) | .004 |
| AST of *Clostridium difficile* isolates | | | |
| Moxifloxacin resistant | 48 (100) | 3 (23) | <.0001 |
| Metronidazole MIC, median (IQR), $\mu$g/mL | 1.5 (1–2) | 0.25 (0.19–0.38) | <.001 |
| Vancomycin MIC, median (IQR), $\mu$g/mL | 3 (2–3) | 0.75 (0.75–1) | <.001 |
| Treatment regimen | | | |
| Metronidazole | 36 (77) | 9 (69) | NS |
| Vancomycin[c] | 16 (33) | 2 (18) | NS |
| Fecal transplant | 1 (2) | 0 (0) | NS |
| Metronidazole failure | 19 (40) | 4 (31) | NS |
| Death during hospitalization | 20 (43) | 3 (27) | .5 |

NOTE. Data are no. (%), unless otherwise indicated. AST, antimicrobial susceptibility testing; CCI, Charlson comorbidity index; IQR, interquartile range; MIC, minimum inhibitory concentration; NS, not significant; WBC, white blood cell.

[a] Includes cr-02 ($n$ = 3), y02 ($n$ = 2), 078 ($n$ = 2), hr ($n$ = 2), gr ($n$ = 1), xr ($n$ = 1), fr ($n$ = 1), and sz1 ($n$ = 1).

[b] $n$ = 51.

[c] Vancomycin as primary treatment.

disease, from 0.51 to 1.18 per 1,000 patient-days in 2006–2011 and 2012–2013, respectively, at our medical center in Jerusalem, Israel.

The objectives of this study were to analyze the clinical and epidemiological characteristics of CDI in our hospital and to explore the clonal structure and antimicrobial susceptibility patterns of the respective *C. difficile* isolates. The study was conducted at Shaare Zedek Medical Center (SZMC), an 800-bed teaching hospital in Jerusalem, and the Israeli National Center of Infection Control laboratory.

All consecutive nonduplicate samples of adult patients from all of the hospital's wards with first-episode CDI were included in the study. We retrospectively retrieved medical records of all patients included and collected demographic data, comorbidities, and clinical characteristics of the CDI. The study was approved by the Institutional Review Board of SZMC.

Laboratory diagnosis of CDI was done by testing nonformed stool samples using an immunochromatographic rapid test that combined glutamate dehydrogenase and toxin A/B (*C. diff* Quik Chek Complete; Alere, Techlab). Positive samples were inoculated on ChromID *C. difficile* agar plates (bioMérieux) and incubated anaerobically for 48 hours. Identification was done on the basis of colony morphology, typical odor, and molecular tests, as described below. Antimicrobial susceptibility testing (AST) was done on supplemented *Brucella* blood agar plates for vancomycin, metronidazole, and moxifloxacin using the gradient method (Etest; bioMérieux). Minimum inhibitory concentration (MIC) criteria for susceptibility were based on Clinical and Laboratory Standards Institute recommendations:[7] moxifloxacin, less than or equal to 2 $\mu$g/mL; metronidazole, less than or equal to 8 $\mu$g/mL; and vancomycin, less than or equal to 4 $\mu$g/mL.

Identification of *C. difficile* was confirmed by polymerase chain reaction (PCR) for the *tpi* gene.[8] Presence of the A and B toxins, the binary toxin, and the in-frame deletions in the *tcdC* gene was tested by PCR.[9] Typing was performed by *slpA* sequencing.[10] Designation of *slpA* types and determination of the inferred ribotype was done on the basis of the nomenclature used by Kato (when present); otherwise, a new name was given. Statistical analysis was done using Epi Info 7 (Centers for Disease Control and Prevention).

From February to September 2013, 66 adult patients with first-episode CDI had frozen stool samples sent for further investigation and culture. *C. difficile* was not cultivated in 3 samples, and nontoxigenic *C. difficile* strains were identified in 2 samples; therefore, 61 patients were included. The mean age was 79 years, 41% were residents of nursing homes, and the mean Charlson comorbidity index (CCI) was high (7 points). Fifty-eight (96%) patients received antibiotics during the 6-week period preceding the CDI; of them, 51 (88%) received cephalosporins and/or quinolones. Most of the cases were hospital acquired (87%), and 39% of the patients had a long length of stay (more than 14 days).

Of the 61 toxigenic *C. difficile* isolates, 48 (79%) belonged to the epidemic *slpA*-type gc8 strain (inferred ribotype 027). In addition to the gc8 strain, the *tcdC* deletion was found in the 078 strain. The results of the ASTs for the gc8 versus non-gc8 strains are presented in Table 1. Resistance to moxi-

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floxacin was present in all gc8 isolates, versus 3 (27%) of the non-gc8 isolates ($P < .001$). Although resistance to vancomycin and metronidazole was not found, the median MIC was significantly higher among the gc8 isolates (Table 1).

CDI patients infected with the epidemic *slpA*-type gc8 strain were more likely to have excessive leukocytosis than patients infected with other strains (mean white blood cell count, 22,408 versus 11,592 cells/$\mu$L; $P = .004$; Table 1). They also had a tendency toward older age, higher CCI, lower albumin, and higher creatinine, but these differences did not reach statistical significance. Mortality rate was high in both groups and was higher in the gc8 strain group (43% vs 27% for other strains), but the difference was not statistically significant.

This is the first outbreak caused by the epidemic ribotype 027 reported from the Middle East and one of the few reported outside Europe and North America. As previously reported,[3] all epidemic strain isolates were resistant to moxifloxacin, compared with only 23% of the other strains. A unique finding of our work is the high MIC of the gc8 strain to metronidazole (median, 1.5 $\mu$L/mL) and vancomycin (median, 3 $\mu$L/mL). Although the significance of a higher MIC to these agents is not clear, we should consider the possibility that it results in CDI that may be only partially treated, leading to treatment failure and contributing to increased morbidity and mortality. A less effective regimen may promote the spread of spores, which in turn enhances the evolution of an epidemic.

The outbreak has increased our prompt attention to infection control practices, including rapid diagnosis, isolation, and cohorting of infected patients; hand washing; environmental disinfection; and antibiotic stewardship. Fortifying each of these components has achieved a decrease in hospital-acquired CDI in our institution, from 1.18 to 0.78 cases per 1,000 patient-days.

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**Yonit Wiener-Well, MD;[1] Eli Ben-Chetrit, MD;[1] Mustafa Abed-Eldaim, MD;[2] Marc V. Assous, MCU-Ph;[3] Tamar Miller-Roll, MSC;[4] Amos Adler, MD[4]**

Affiliations: 1. Infectious Disease Unit, Shaare Zedek Medical Center, affiliated with Hadassah-Hebrew University Medical School, Jerusalem, Israel; 2. Internal Medicine, Shaare Zedek Medical Center, affiliated with Hadassah-Hebrew University Medical School, Jerusalem, Israel; 3. Clinical Microbiology Laboratory, Shaare Zedek Medical Center, affiliated with Hadassah-Hebrew University Medical School, Jerusalem, Israel; 4. National Center of Infection Control, Ministry of Health, affiliated with Tel-Aviv University Sackler Faculty of Medicine, Tel-Aviv, Israel.

Address correspondence to Yonit Wiener-Well, MD (yonitw@szmc.org.il).

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