Concise Communications

Clinical and Epidemiologic Features of Methicillin-Resistant *Staphylococcus aureus* in Elderly Hospitalized Patients

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ABSTRACT

We describe characteristics of elderly patients with MRSA identified in 37 Canadian hospitals between 1995 and 2002. Of these inpatients, 6,613 (66%) were older than 65 years. They were more likely than younger patients to have been colonized without infection and to have had MRSA isolated from urine or the perineum. The epidemiology and clinical features of these patients is distinct from that of younger patients (*Infect Control Hosp Epidemiol* 2005;26:838-841).

Methicillin-resistant *Staphylococcus aureus* (MRSA) remains an important nosocomial pathogen, associated with a substantial burden of disease. Patients with MRSA often are older and have several underlying diseases and recent hospitalization.[1] The epidemiology, transmission, risk factors, and outcome of MRSA among elderly residents of long-term–care facilities have been described.[2-6] Less has been reported about the epidemiology of MRSA in elderly hospitalized patients. In this article, we describe the clinical features and epidemiology of MRSA among elderly hospitalized patients identified through the Canadian Nosocomial Infection Surveillance Program (CNISP), a national hospital-based surveillance network in Canada.[7]

METHODS

Surveillance for MRSA has been conducted by up to 37 hospitals in Canada participating in CNISP since January 1995. Surveillance methods have previously been described.[7] Briefly, when a new case of MRSA was identified from an inpatient, the hospital's infection control practitioner used a standardized data collection form to abstract demographic and clinical information from the patient's medical records, including age, gender, site of MRSA acquisition, anatomic site of MRSA infection or colonization, and the reason for which the culture that yielded MRSA was performed. The presence of infection caused by MRSA was determined according to standard definitions. For MRSA to be considered to have been acquired in a hospital, there had to be no evidence that the organism was present at the time of admission or that it was likely acquired during a previous hospital admission. MRSA was considered to have been acquired in a nursing home if a nursing home resident was found to have MRSA on admission to the hospital and, if in the judgment of the infection control practitioner, there was no evidence of prior hospital acquisition of the organism. Community acquisition of the organism was assumed if, in the judgment of the infection control practitioner, there was no evidence of acquisition in a hospital or long-term–care facility (eg, no hospitalization in the previous 12 months).

MRSA screening specimens included swabs of nasal, perineal, and catheter exit sites. Although MRSA screening criteria may have varied somewhat from hospital to hospital, age was not used as a criterion for screening in any facility. Screening for MRSA was done within 48 hours of admission to the hospital for patients transferred from another healthcare facility, those who had been in another healthcare facility in the preceding 6 to 12 months, and those who had previously been known to be infected or colonized with MRSA. Screening was also done for those who were roommates ("contacts") of a newly identified patient with MRSA, or as part of the investigation of an outbreak occurring in an inpatient unit. Clinical (non-screening) specimens that yielded MRSA on culture were ordered at the discretion of the attending physician.

MRSA isolates were sent to a central laboratory for confirmation of organism identification and antimicrobial susceptibility testing by microbroth dilution in accordance with National Committee for Clinical Laboratory Standards guidelines. All isolates were confirmed to be MRSA by detection of the *mec*A gene by polymerase chain reaction assay.

Demographic, clinical, and epidemiologic data of elderly patients (65 years and older) were compared with those of younger patients (18 to 64 years old). Categorical variables were compared using Fisher's exact test or the chi-square test, as appropriate. Continuous variables were compared using the Student's *t* test. Variables found to be significant on univariate analysis (two-tailed $P < .05$) were included in a backward logistic regression model.

RESULTS

A total of 6,613 patients with MRSA were 65 years of age or older, representing 66% of all adults identified with MRSA in CNISP hospitals from 1995 to 2002. Demographic and clinical data for adults with MRSA are presented in Table 1, and the results of the multivariate logistic regression analysis are presented in Table 2. Elderly patients with MRSA were more likely to have been colonized with the organism without infection (odds ratio [OR], 1.8; 95% confidence interval [CI_{95}], 1.7 to 2.0; $P < .001$) and to have had MRSA isolated from urine (OR, 1.4; CI_{95}, 1.2 to 1.7; $P < .001$) or the perineum (OR, 1.4; CI_{95}, 1.3 to 1.6; $P < .001$). They were less likely to have had MRSA isolated from skin or soft tissue (OR, 0.7; CI_{95}, 0.6 to 0.8; $P < $

TABLE 1
DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF HOSPITALIZED ADULT PATIENTS WITH METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS*

Characteristic	Age		P
	≥ 65 y (n = 6,613)	18 to 64 y (n = 3,476)	
Male	3,738 (57%)	2,156 (62%)	< .001
Hospital admission in previous 12 mo	3,561 (54%)	1,880 (54%)	.20
Site of MRSA acquisition			
Hospital	4,643 (70%)	2,472 (71%)	
Long-term–care facility	513 (8%)	66 (2%)	< .001
Community	285 (4%)	238 (7%)	
Unknown	1,172 (18%)	700 (20%)	
Reason for culture			
Clinical indication	2,442 (37%)	1,775 (51%)	< .001
Surveillance or screening	2,685 (41%)	984 (28%)	
Outbreak investigation	1,093 (17%)	518 (15%)	
Unknown	393 (6%)	199 (6%)	
MRSA infection	1,833 (28%)	1,414 (41%)	< .001
Initial site of MRSA infection or colonization			
Nose	2,919 (44%)	1,219 (35%)	< .001
Perineum	1,413 (21%)	536 (15%)	< .001
Skin or soft tissue	1,437 (22%)	995 (29%)	< .001
Surgical site	555 (8%)	523 (15%)	< .001
Respiratory tract	1,116 (17%)	738 (21%)	< .001
Urinary tract	633 (10%)	219 (6%)	< .001
Bloodstream	275 (4%)	217 (6%)	< .001
No. of sites with MRSA			
1	4,601 (70%)	2,306 (66%)	.01
2	1,328 (20%)	725 (21%)	
> 2	608 (9%)	399 (11%)	
Not known	76 (1%)	46 (1%)	
Epidemiologic link with another hospitalized patient	3,142 (48%)	1,644 (47%)	.24

MRSA = methicillin-resistant *S. aureus*.

.001), a surgical wound (OR, 0.6; CI_{95}, 0.5 to 0.7; $P < .001$), or a respiratory specimen (OR, 0.8; CI_{95}, 0.7 to 0.9; $P = .003$).

DISCUSSION

Most studies of MRSA among elderly patients have been performed in nursing homes or other types of long-term–care facilities. In this setting, most individuals remain colonized with the organism for prolonged periods. Although MRSA infection rates are relatively low in nursing homes, those who are colonized with the organism appear to have an increased risk of developing infection due to MRSA as compared with non-colonized residents.[3-5] Risk factors associated with the acquisition of MRSA among elderly residents of long-term–care facilities have included poor functional status, the presence of decubitus ulcers or other skin lesions,

the presence of medical devices such as a urinary catheter or a feeding tube, and recent hospitalization.[3,6,8-12]

Although the elderly comprise the majority of hospital inpatients with MRSA (66% of adult patients in CNISP hospitals), there have been few studies determining the prevalence or describing the epidemiology of MRSA in older adults admitted to hospitals, and most of these studies have described a single center's experience.[13,14] Hori et al. found that being male, having a diagnosis of diabetes mellitus, and having recently been prescribed ciprofloxacin or ampicillin were associated with nasal colonization with MRSA in elderly patients (older than 65 years) admitted to a hospital in Nottingham.[14] Our study was not designed to determine risk factors for the acquisition of MRSA, but it did identify several features that distinguished older hospitalized patients with MRSA from

TABLE 2
RESULTS OF MULTIVARIATE LOGISTIC REGRESSION ANALYSIS OF VARIABLES ASSOCIATED WITH METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS* IN ELDERLY HOSPITALIZED PATIENTS

Characteristic	OR (CI$_{95}$)	P
MRSA colonization without infection	1.8 (1.7–2.0)	< .001
MRSA isolation		
From only one anatomic site	1.2 (1.1–1.3)	< .001
From a urinary specimen	1.4 (1.2–1.7)	< .001
From the perineum	1.4 (1.3–1.6)	< .001
From skin or soft tissue	0.7 (0.6–0.8)	< .001
From a surgical site	0.6 (0.5–0.7)	< .001
From a respiratory specimen	0.8 (0.7–0.9)	< .001

OR = odds ratio; CI$_{95}$ = 95% confidence interval; MRSA = methicillin-resistant *S. aureus*.

younger patients. Older patients were nearly twice as likely to be colonized with MRSA without evidence of an infection, probably because they were more likely to have been identified by a screening culture. Older patients were more likely to have been screened because recent hospitalization (within the past 12 months) was a major criterion for screening in CNISP hospitals. Colonization with MRSA has been associated with a greater risk of developing a staphylococcal infection in the elderly than has colonization with a susceptible strain of *S. aureus*.[4] This study found that 28% of elderly hospitalized patients with MRSA met the criteria for infection, a rate that is substantially higher than that reported among elderly nursing home residents with MRSA.[5,6,15-17]

The sites from which MRSA was initially recovered from elderly inpatients differed from those of younger patients. Older patients were more likely to have a urinary isolate or perineal colonization (OR, 1.4). Ascertainment bias may explain these differences if elderly individuals were more likely than younger individuals to have been screened for MRSA and also more likely to have had perineal or urinary specimens cultured. In approximately 13% of those older than 65 years, the perineum or rectum was the only site that yielded MRSA (data not shown), suggesting that screening for MRSA with a nasal swab alone would be suboptimal in elderly hospitalized patients.

It is recognized that residents in long-term–care facilities may serve as a reservoir for MRSA that may spread into acute care hospitals.[18-21] Indeed, hospital outbreaks and nosocomial transmission of MRSA have been linked to the admission of elderly residents of nursing homes.[18] However, the observation that recent hospitalization is a risk factor for MRSA carriage among residents of long-term–care facilities[6,12,19,22] suggests that many, if not most, residents acquire the organism in the hospital. Similarly, in the current study, only 8% of elderly hospitalized patients with MRSA were thought to have acquired the organism in a long-term-care–facility and in the majority (70%) of the patients, MRSA was judged to have been hospital acquired. It is also possible

that some of the patients thought to have acquired MRSA in a nursing home actually did so during a prior unreported hospitalization. It is evident, therefore, that hospitals, nursing homes, and other long-term–care facilities may serve as reservoirs facilitating the interinstitutional transfer of antibiotic-resistant organisms such as MRSA.

The results of this surveillance indicate that the epidemiology and clinical features of MRSA in elderly hospitalized patients is distinct from that in younger patients. Further studies should be done to confirm and better define these differences and to determine their potential implications for the management and control of MRSA in hospitals and long-term–care facilities.

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Presented in part at the 42nd Annual Meeting of the Infectious Diseases Society of America; September 30-October 3, 2004; Boston, MA.

REFERENCES
1. Bradley SF. *Staphylococcus aureus* infections and antibiotic resistance in older adults. *Clin Infect Dis* 2002;34:211-216.

2. Strausbaugh LJ, Jacobson C, Sewell DL, Potter S, Ward TT. Methicillin-resistant *Staphylococcus aureus* in extended-care facilities: experiences in a Veterans' Affairs nursing home and a review of the literature. *Infect Control Hosp Epidemiol* 1991;12:36-45.

3. Bradley SF, Terpenning MS, Ramsey MA, et al. Methicillin-resistant *Staphylococcus aureus*: colonization and infection in a long-term care facility. *Ann Intern Med* 1991;115:417-422.

4. Muder RR, Brennen C, Wagener MM, et al. Methicillin-resistant staphylococcal colonization and infection in a long-term care facility. *Ann Intern Med* 1991;114:107-112.

5. Mulhausen PL, Harrell LJ, Weinberger M, Kochersberger GG, Feussner JR. Contrasting methicillin-resistant *Staphylococcus aureus* colonization in Veterans Affairs and community nursing homes. *Am J Med* 1996;100:24-31.

6. Bradley SF. Methicillin-resistant *Staphylococcus aureus* in nursing homes: epidemiology, prevention and management. *Drugs Aging* 1997; 10:185-198.

7. Simor AE, Ofner-Agostini M, Bryce E, et al. The evolution of methicillin-resistant *Staphylococcus aureus* in Canadian hospitals: 5 years of national surveillance. *CMAJ* 2001;165:21-26.

8. Hsu CCS. Serial survey of methicillin-resistant *Staphylococcus aureus* nasal carriage among residents in a nursing home. *Infect Control Hosp Epidemiol* 1991;12:416-421.

9. Murphy S, Denman S, Bennett RG, Greenough WB III, Lindsay J, Zelesnick LB. Methicillin-resistant *Staphylococcus aureus* colonization in a long-term-care facility. *J Am Geriatr Soc* 1992;40:213-217.

10. Cox RA, Bowie PES. Methicillin-resistant *Staphylococcus aureus* colonization in nursing home residents: a prevalence study in Northamptonshire. *J Hosp Infect* 1999;43:115-122.

11. O'Sullivan NP, Keane CT. Risk factors for colonization with methicillin-resistant *Staphylococcus aureus* among nursing home residents. *J Hosp Infect* 2000;45:206-210.

12. von Baum H, Schmidt C, Svoboda D, Bock-Hensley O, Wendt C. Risk factors for methicillin-resistant *Staphylococcus aureus* carriage in residents of German nursing homes. *Infect Control Hosp Epidemiol* 2002;23:511-515.

13. Parnaby RM, O'Dwyer G, Monsey HA, Shafi MS. Carriage of *Staphylococcus aureus* in the elderly. *J Hosp Infect* 1996;33:201-206.

14. Hori S, Sunley R, Tami A, Grundmann H. The Nottingham *Staphylococcus aureus* study: prevalence of MRSA among the elderly in a community hospital. *J Hosp Infect* 2002;50:25-29.

15. Terpenning MS, Bradley SF, Wan JY, Chenoweth CE, Jorgenson KA, Kauffman CA. Colonization and infection with antibiotic-resistant bacteria in a long-term care facility. *J Am Geriatr Soc* 1994;42:1062-1069.

16. Lee Y-L, Cesario T, Gupta G, et al. Surveillance of colonization and infection with *Staphylococcus aureus* susceptible or resistant to methicillin in a community skilled-nursing facility. *Am J Infect Control* 1997;25:312-321.

17. Drinka P, Faulks JT, Gauerke C, Goodman B, Stemper M, Reed K. Adverse events associated with methicillin-resistant *Staphylococcus aureus* in a nursing home. *Arch Intern Med* 2001;161:2371-2377.

18. Hsu CCS, Macaluso CP, Special L, Hubble RH. High rate of methicillin resistance of *Staphylococcus aureus* isolated from hospitalized nursing home patients. *Arch Intern Med* 1988;148:569-570.

19. Thomas JC, Bridge J, Waterman S, Vogt J, Kilman L, Hancock G. Transmission and control of methicillin-resistant *Staphylococcus aureus* in a skilled nursing facility. *Infect Control Hosp Epidemiol* 1989;10:106-110.

20. Goodall B, Tompkins DS. Methicillin-resistant staphylococcal infections: nursing homes act as a reservoir. *Br Med J* 1994;308:58.

21. Troillet N, Carmeli Y, Samore MH, et al. Carriage of methicillin-resistant *Staphylococcus aureus* at hospital admission. *Infect Control Hosp Epidemiol* 1998;19:181-185.

22. Fraise AP, Mitchell K, O'Brien SJ, Oldfield K, Wise R. Methicillin-resistant *Staphylococcus aureus* (MRSA) in nursing homes in a major UK city: an anonymized point prevalence survey. *Epidemiol Infect* 1997;118:1-5.

Lessons Learned From a Norovirus Outbreak in a Locked Pediatric Inpatient Psychiatric Unit

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ABSTRACT

We report an outbreak of norovirus in a locked pediatric inpatient psychiatric unit with attack rates of 75% among 4 patients and 26% among 38 staff. Factors contributing to the outbreak included environmental contamination, close staff–patient contact including sharing meals, and inability to confine the index patient with the use of contact precautions (*Infect Control Hosp Epidemiol* 2005;26:841-843).

Noroviruses are single-stranded, non-enveloped RNA viruses that are responsible for 68% to 80% of all outbreaks of acute gastroenteritis in industrialized countries.[1] Outbreaks occur in all age groups and in many settings including restaurants, cruise ships, schools, long-term–care facilities, and hospitals. Noroviruses are spread primarily by the fecal–oral route, although airborne and fomite transmission might facilitate spread during outbreaks.[2] Frequently during an outbreak, primary cases result from exposure to a fecally contaminated vehicle (eg, food), whereas secondary and tertiary cases among contacts of primary cases result from person-to-person transmission.

We report an outbreak of norovirus infection that occurred in a locked pediatric psychiatric unit and resulted in high attack rates among patients and staff. Control was complicated by the inability to confine the source-patient to his room on contact precautions.[3] Also, we discuss the unique difficulties of outbreak detection and control in psychiatric units.

METHODS

This outbreak was evaluated by University of North Carolina (UNC) Hospitals' Department of Hospital Epidemiology. Clinical stool samples were processed for enteric pathogens using standard techniques in the UNC Hospitals' microbiology laboratory. Norovirus RNA was extracted from 10% fecal suspensions using the Viral RNA Mini Kit (Qiagen, Valencia, CA) and detected using a broadly reactive reverse transcription polymerase chain reaction (RT-PCR) targeting the polymerase (region A) and the capsid (region D) regions.[4] RT-PCR products underwent sequencing at the Lineberger Sequence Facility at UNC Chapel Hill with the use of the BigDye Terminator Cycle Sequencing Ready Reaction Kit (PE Applied Biosystems, Foster City, CA).[4]

Description of the Outbreak

The locked pediatric psychiatric unit at UNC Hospitals admits patients 6 to 12 years old into a unit that consists of 3 double and 4 single patient rooms and 3 common rooms (a play room, a dining room, and a classroom). The patients have access to all common rooms and share meals with the staff as part of their overall therapy. The index patient, a 9-year-old boy with autism and mood disorders, was admitted on January 11, 2004. The child was unable to manage his own toileting and was therefore using diapers. He also demonstrated a behavior problem of frequent fecal smearing on environmental surfaces. On the day of admission, the child developed nausea, vomiting, and loose stools.

During the evening of January 15, 2004, the hospital epidemiology department was notified that 3 patients and staff members in the locked pediatric psychiatric unit had gastroenteritis, and an outbreak investigation was initiated. The investigation included obtaining stool samples for micro-
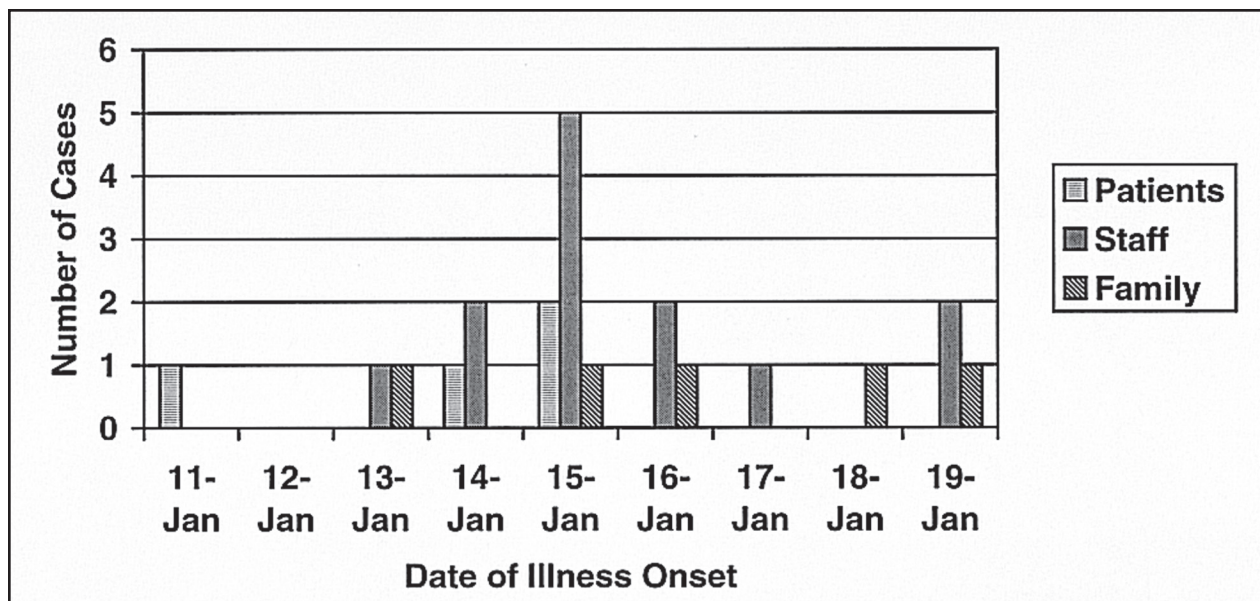
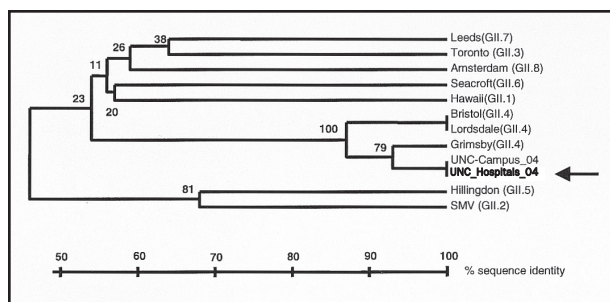**FIGURE 1.** Outbreak curve based on the day of disease onset.

FIGURE 2. Phylogenic analysis of norovirus GII region D nucleotide sequences including sequences of University of North Carolina (UNC) Hospitals and UNC Campus outbreak and norovirus prototype strains previously submitted to GenBank (Leeds, AJ277608; Toronto, U02030; Amsterdam, AF195848; Seacroft, Aj277620; Hawaii, U07611; Bristol, X76716; Lordsdale, X86557; Grimsby, AJ004864; Hillingdon, AJ277607; and Snow Mountain virus [SMV], U75682). Bootstrap values are given for each node. Norovirus strains are classified according to the most recent norovirus genotype nomenclature.[4] The arrow indicates the strain that caused the outbreak.

biologic analysis from the index patient, assessing whether other patients or staff members had symptoms of gastroenteritis, and communicating with the county health department. Stool samples of the index patient were negative for rotavirus, *Salmonella*, *Shigella*, *Escherichia coli* O157:H7, *Campylobacter*, *Yersinia*, *Giardia*, and *Cryptosporidium*.

An outbreak curve was constructed following the initiation of active surveillance (Fig. 1). Ultimately, 3 (75%) of 4 patients, 10 (26%) of 38 permanently assigned staff, 3 staff temporarily floated from other psychiatric units, and 5 family members developed gastroenteritis. Symptoms reported by 13 staff members included loose or watery stools in 92%, nausea in 85%, abdominal pain in 77%, vomiting in 69%, and fever in 31%.

Stool samples from the index patient and 2 staff members with dates of onset of January 15 and 17, 2004, were positive for norovirus by RT-PCR, and the 3 had identical sequences. Phylogenic analysis (Fig. 2) of the polymerase gene revealed that the norovirus strains were similar to a novel genotype GII.4 variant detected in Europe[5] and that the consistent mutation in the polymerase gene associated with this emergent variant was present. During the outbreak period, we also analyzed fecal specimens from an outbreak among UNC students and isolated a genetically similar GII.4 strain.

Interventions

Control efforts were initiated on January 16, 2004, but were hindered by the inability to confine the index patient to his room due to his psychiatric disorder. The following interventions were initiated: the unit was closed to all admissions; all staff with symptoms of gastroenteritis were given sick leave; ill staff were not allowed to work until asymptomatic for at least 2 days; staff were precluded from eating or drinking in the unit; the entire unit was treated as an isolation "room," with all entering staff performing hand hygiene and then donning gloves and a disposable gown; the unit was extensively cleaned and disinfected several times with 1:10 diluted hypochlorite (household bleach); and hand hygiene with soap and water, rather than a waterless alcohol-containing foam, was instituted. The last case of gastroenteritis occurred on January 19, 2004. The unit was reopened on January 24, 2004. No subsequent cases of gastroenteritis were reported in the following 30 days.

DISCUSSION

Characteristics of noroviruses that facilitate outbreaks include a low infectious dose ($< 10^2$ viral particles), prolonged asymptomatic shedding, environmental stability, and lack of lasting immunity resulting in susceptible

populations.[2] Multiple outbreaks have been reported in acute care hospitals.[6-10] Healthcare facilities accounted for 25% of 233 non-bacterial outbreaks reported in the United States between July 1997 and June 2000.[1]

Phylogenic analysis revealed that the norovirus strain associated with this outbreak was similar to an emerging predominant norovirus variant of genotype GII.4 that has a consistent mutation in the polymerase gene.[5] This new norovirus variant may have been responsible for an increase and an unusual seasonal pattern of norovirus gastroenteritis in the United States[11] and Europe in 2002.[5]

Rapid institution of control measures, as in our outbreak, has been reported to control such outbreaks within a few days.[9] The use of contact precautions is recommended for patients with norovirus infection.[3] However, we were unable to confine the index patient in our outbreak to his room due to his underlying psychiatric disorder. Closing the unit to admissions and treating the entire unit as an isolation room was effective in curtailing the outbreak. We recognize that this measure did not protect other patients, but at the time that we introduced it, only a single patient had not developed disease. Had multiple patients been asymptomatic, cohorting patients into non-infected and infected units would have been required.

Eating with patients has been noted to be a risk factor for hospital-associated outbreaks of hepatitis A.[12] The high attack rate among staff in our outbreak was likely facilitated by the participation of staff and patients in common meals. Healthcare facilities should prohibit sharing of meals between staff and patients if either a staff member or a patient has evidence of jaundice or gastroenteritis.

Finally, environmental contamination likely played a role in our outbreak. Noroviruses are stable in the environment and able to survive for days on environmental surfaces.[2] We used comprehensive cleaning with hypochlorite, an agent with demonstrated efficacy against noroviruses.[13] Recent data suggest that hypochlorite is able to decontaminate surfaces contaminated with norovirus as a result of contact with contaminated hands.[14] The efficacy of hypochlorite with a contact time of 1 minute has been confirmed by other investigators using feline calicivirus, a surrogate for human norovirus.[15] However, cleaning followed by a combined hypochlorite–detergent formulation was required to decontaminate fecally contaminated surfaces.[14] Although we chose to use a hypochlorite solution for surface disinfection, a disinfectant containing quaternary ammonium compounds has also been demonstrated to be effective against the feline calicivirus using a contact time of 10 minutes.[15]

Along with enhanced environmental disinfection, we recommended hand hygiene with soap and water, rather than a waterless alcohol-containing foam, because a non-enveloped virus inoculated on the hands was not inactivated by alcohol applied for 10 seconds in a recent study.[16] Recent data demonstrated that hand hygiene with ethanol for 30 seconds was effective in eliminating more than 3 logs of feline calicivirus.[17] Whether ethanol-based hand hygiene agents would be effective with a contact time of 10 seconds, as is typical for hand hygiene, is unknown.[18]

Psychiatric units present enhanced opportunities for the spread of diseases transmitted by the fecal–oral route because their patients may be unable to contain excretions and may not cooperate with isolation precautions and because unusually close contact may occur between patients and healthcare providers. Rapid institution of unit-wide contact precautions, prohibition of staff eating with patients, furlough of ill staff, enhanced environmental disinfection, and, when necessary, cohorting of patients should allow for control of diseases such as norovirus that are transmitted by the fecal–oral route.

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REFERENCES

1. Fankhauser RL, Monroe SS, Noel JS, et al. Epidemiology and molecular trends of Norwalk-like viruses associated with outbreaks of gastroenteritis in the United States. *J Infect Dis* 2002;186:1-7.
2. Centers for Disease Control and Prevention. Norwalk-like viruses: public health consequences and outbreak management. *MMWR* 2001; 50(RR-9):1-17.
3. Garner JS. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53-80.
4. Vinjé J, Hamidjaja RA, Sobsey MD. Detection of novel genotypes using VP1 typing for genetic classification of genogroup I and II noroviruses. *J Virol Methods* 2004;116:109-117.
5. Lopman B, Vennema H, Kohli E, et al. Increase in viral gastroenteritis outbreaks in Europe and epidemic spread of new norovirus variant. *Lancet* 2004;363:682-688.
6. Green J, Wright PA, Gallimore CI, et al. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. *J Hosp Infect* 1998;39:39-45.
7. Caceres VM, Kim DK, Bresee JS, et al. A viral gastroenteritis outbreak associated with person-to-person spread among hospital staff. *Infect Control Hosp Epidemiol* 1998;19:162-167.
8. Cunney RJ, Costigan P, McNamara EB, et al. Investigation of an outbreak of gastroenteritis caused by Norwalk-like virus, using solid phase immune electron microscopy. *J Hosp Infect* 2000;44:113-118.
9. McCall J, Smithson R. Rapid response and strict control measures can contain a hospital outbreak of Norwalk-like virus. *Commun Dis Public Health* 2002;5:243-246.
10. Billgren M, Christenson B, Hedlund KO, Vinjé J. Epidemiology of Norwalk-like human caliciviruses in hospital outbreaks of acute gastroenteritis in the Stockholm area in 1996. *J Infect* 2002;44:26-32.
11. Centers for Disease Control and Prevention. Norovirus activity: United States, 2002. *MMWR* 2003;52:41-45.
12. Weber DJ, Rutala WA. Choosing vaccines for healthcare workers. *Infect Control Hosp Epidemiol* 1997;10:682-687.
13. Keswick BH, Satterwhite TK, Johnson PC, et al. Inactivation of Norwalk virus in drinking water by chlorine. *Appl Environ Microbiol* 1985;50:261-264.
14. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J Hosp Infect* 2004;58:42-49.
15. Sattar SA. Microbicides and the environmental control of nosocomial viral infections. *J Hosp Infect* 2004;56:S64-S69.
16. Sickbert-Bennett E, Rutala WA, Weber DJ, Gergen-Teague MF, Sobsey MD. Comparative efficacy of hand hygiene agents in the reduction of bacteria and viruses. *Am J Infect Control* 2005;33:67-77.
17. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J Hosp Infect* 2004;56:49-55.
18. Boyce JM, Pittet D. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *Infect Control Hosp Epidemiol* 2002;23(suppl):S3-S40.