deficits regarding CLABSI prevention, nor were we able to obtain postintervention data on compliance with hand hygiene and central venous catheter access and maintenance bundles.

Further work is planned for ongoing collaboration and tracking of infection rates over time. Despite these limitations, the CUORE pilot project demonstrated that real-time interactive conferences for healthcare providers placed in remote pediatric ICUs are feasible and yield a high satisfaction among participants. Future efforts should concentrate on sustaining online training programs for a longer time, eventually involving multiple centers at the same time. We believe this model would also be appropriate for critical care topics other than CLABSI rate reduction. In addition to continuing the CUORE program, we are aiming at developing further online resources (eg, a CUORE Web site containing lecture material).

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Community and Nursing Home Residents with Carbapenemase-Producing *Klebsiella pneumoniae* Infection

To the Editor—Healthcare-associated carbapenemase-producing *Klebsiella pneumoniae* (CPKP) infections have a high mortality.[1-3] In March 2009, the first patient with a CPKP infection identified at the time of presentation to our hospital system was a local nursing home resident. Then another resident with a CPKP infection presented to our hospital system from the same nursing home. A point-prevalence survey was performed using rectal swab samples to assess patients at that nursing home unit. One additional patient from 41 patients screened was identified as a carrier of CPKP. Other cases of CPKP infection were then noted at hospital admission among patients from other nursing homes and from the community. Because there are currently few data on the epidemiology of CPKP infection in patients presenting to US hospitals and clinics, we studied such patients who presented to our hospital system, and we screened nursing home residents at hospital admission to detect asymptomatic CPKP colonization.

We reviewed patients for whom CPKP was grown from clinical cultures in 2009 using the methodology described below at hospital admission to Rhode Island Hospital, Miriam Hospital, or Newport Hospital; during outpatient clinic or emergency department visits at these hospitals; or at a nursing home if the patient's medical care was delivered in our hospital system during 2009. Patients without clinical evidence of infection and admitted patients with samples for cultures that were collected more than 24 hours after hospitalization were excluded. Patient characteristics and risk factors were assessed by medical record review and interviews with primary care physicians and nursing home staff. Functional status was defined as poor if patients were bed-bound or required maximal assistance for ambulation.

During January and February 2010, we screened 100 nursing home patients within 24 hours after admission to the above-noted hospitals using rectal swabs to detect asymptomatic CPKP carriage. Initial CPKP identification was performed using the method recommended by the Centers for Disease Control and Prevention.[4] Swabs were used to inoculate 5 mL of Trypticase soy broth (BD) onto a 10-μg meropenem disk. After overnight incubation, the broth was vortexed, and 100 μL was subcultured onto MacConkey agar, incubated for 48 hours, and examined for lactose-fermenting colonies. The modified Hodge test was performed to confirm carbapenemase resistance.[5] Our Institutional Review Board approved this project.

Seven patients who had community-acquired or nursing home–acquired (long-term chronic care facility) CPKP infection were identified (Table 1). Among the patients with both meropenem and tigecycline minimum inhibitory concentration (MIC) data, MICs (μg/mL) were 1 and 1, respectively, for 2 patients; 1 and 1.5, respectively, for 1 patient; and 4 and 1, respectively, for 1 patient. Samples for culture were obtained from 3 patients at a nursing home; the remaining samples for culture were obtained at hospital admission or during an outpatient clinic or emergency department visit. The 4 hospitalized patients were placed under contact precautions for the remainder of their hospitalization and future admissions. Before the onset of CPKP infection, 2 patients were living in the community, and 5 nursing home patients resided in 2 facilities. All 7 patients had a urinary source of infection; 1 patient developed secondary bacteremia. All 7 patients received antibiotics during the 95 days before their CPKP infection was identified (median, 25 days since the last antibiotic exposure), including 2 patients who received a carbapenem. All 7 had been hospitalized previously (median, 38 days since the last hospitalization). None of the 7 patients were admitted to an intensive care unit or underwent surgery during the 3 months before the CPKP infection. Of note, only 1 case of hospital-acquired CPKP infection or colonization was identified at our 3 hospitals during 2009.

None of the rectal swab samples from 100 screened nursing home residents admitted to our hospitals revealed CPKP colonization, but we incidentally detected a patient colonized with extended-spectrum β-lactamase–producing *Escherichia coli*.

Published guidelines exist for control of CPKP infections.[5] Hospital outbreaks have been controlled by means of strict contact precautions, cohorting of patients, serial screening cultures, enhanced environmental decontamination, and staff education.[2] A bundled intervention in long-term care dramatically decreased CPKP colonization.[6] Risk factors for acquisition of carbapenemase-resistant *Enterobacteraciae* include prolonged hospitalization, intensive care unit stay, use of invasive devices, immunosuppression, and previous antibiotic exposure.[1,7,8] Much of these data focus on nosocomial acquisition; less is known about risk factors in community and nursing home settings.

Compared with patients with hospital-acquired CPKP infection,[7] our patients were more likely to have urinary tract involvement and were more likely to be female. All 7 patients had received antibiotics within the prior 3 months, similar to 73% of nursing home residents colonized with multidrug-resistant gram-negative bacteria.[9] All 7 patients had been hospitalized during the 95 days before presentation. We cannot be certain that CPKP was acquired in the nursing home or community; however, only 1 patient with hospital-acquired CPKP colonization or infection was identified in any of our 3 hospitals during 2009, suggesting that acquisition in our case series likely occurred outside the hospital.

TABLE 1. Patients with Carbapenemase-Producing *Klebsiella pneumoniae* Infection Detected at Hospital Admission or during an Outpatient Visit

	Patient						
Characteristic	1	2	3	4	5	6	7
Age, years	47	69	67	64	76	91	75
Sex	Male	Female	Female	Female	Female	Female	Female
Residence	Nursing home	Nursing home	Home	Nursing home	Nursing home	Nursing home	Home
Site of infection	Urine, blood	Urine	Urine	Urine	Urine	Urine	Urine
Days since last antibiotics	30	70	74	11	2	25	9
Days since last hospital discharge	95	95	76	11	12	38	18
Urinary catheter in place	Yes	Yes	No	No	No	No	Yes
CVC in place	No	No	No	Yes	No	No	No
Immunosuppression	No	Corticosteroids	Corticosteroids, chemotherapy	Corticosteroids	No	No	Corticosteroids
Malignancy	No	No	Yes	No	No	No	No
Diabetes mellitus	No	No	Yes	Yes	No	Yes	No
Congestive heart failure	No	No	No	No	Yes	Yes	No
Chronic kidney disease	No	No	Yes	No	Yes	No	No
Chronic liver disease	Yes	No	No	No	No	No	No
Neurologic disease	No	Yes	No	Yes	No	Yes	Yes
Recurrent or chronic infection	Yes	Yes	Yes	Yes	No	Yes	Yes
Functional status	Good	Poor	Good	Poor	Poor	Poor	Poor
Previous infection with multidrug-resistant pathogen	No	MRSA	VRE	MRSA, VRE, ESBL-producing *E. coli*	No	ESBL-producing *E. coli*	QUI-, AMP-, and TMP-SMX-resistant *Klebsiella*

NOTE. AMP, ampicillin; CVC, central venous catheter; ESBL, extended-spectrum β-lactamase; MRSA, methicillin-resistant *Staphylococcus aureus*; QUI, quinolone; TMP-SMX, trimethoprim-sulfamethoxazole; VRE, vancomycin-resistant *Enterococcus*.

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.

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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-