TABLE 1. *Clostridium difficile* Culture Results: Effectiveness of Manual Cleaning Versus UV-C

	Post Bleach CFUs		Post UV-C CFUs	
Site	≥10 CFUs	<10 CFUs	≥10 CFUs	<10 CFUs
Over-bed table	13	41	0	54
Toilet seat	9	65	0	74
Computer keyboard	3	19	1	43
Bathroom doorknob	2	20	0	22
Faucet handles	2	39	0	41
Bed side rails	1	3	0	4
Bedside commode	1	11	0	12
Recliner chair table	1	6	0	7
Call light	0	2	0	2

NOTE. CFU, colony-forming units; UV-C, ultraviolet light at 254 nanometers.

detection of CFU following UV-C treatment (odds ratio [OR], 0.027; 95% confidence interval [CI], 0.0006–0.1664; $P < .0001$). The hospital-onset *C. difficile* cases decreased to 11.4 per 10,000 patient days from 12.9 for the same period the prior year (during which UV-C had not yet been utilized).

In rooms of patients with confirmed CDI, UV-C treatment significantly reduced the amount of *C. difficile* spores present on the surfaces tested. Manual bleach cleaning alone resulted in residual spores in 13% of high-touch-surface cultures. These cultures were obtained as part of daily routine cleaning without the knowledge of Environmental Services workers. The performance of our cleaning staff is regularly evaluated by VeriClean blacklight audits, another objective evaluation tool demonstrating areas potentially missed by the cleaning staff. The average pass rate was 90.8% during the study period. Data recently published by Wong et al[7] are consistent with our findings; they reported that 5 of 22 rooms (22%) were positive for *C. difficile* after terminal cleaning.[7] The hospital-onset *C. difficile* rate decreased as well, even without 100% compliance of UV-C treatment of discharge enteric contact isolation rooms.

In patient rooms of those with confirmed CDI, adding UV-C treatment to daily bathroom and terminal discharge cleaning reduces the amount of *C. difficile* spores present on frequently contaminated surfaces. UV-C disinfection represents an additional measure for room cleaning to avoid inadvertent transfer of *C. difficile* spores to hands or other surfaces.

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Resolution of Carbapenemase-Producing *Klebsiella pneumoniae* Outbreak in a Tertiary Cancer Center; the Role of Active Surveillance

Carbapenem-resistant *Enterobacteriaceae* (CRE) are a source of healthcare-associated infections with high attributable mortality.[1] Carbapenemase-producing CRE (CP-CRE) (eg, KPC, OXA-48, NDM, IMP or VIM) are more commonly acquired

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exogenously than non–CP-CRE.[1] The most common CP-CRE in the United States harbors the *Klebsiella pneumoniae* carbapenemase (KPC) gene (bla_{KPC}).[1] Here, we report our investigation of a cluster of KPC-producing *K. pneumoniae* (KPC-KP) in a large academic medical facility cancer center.

The Johns Hopkins Hospital is a 1,059-bed academic medical facility in Baltimore, Maryland with an NCI-designated Comprehensive Cancer Center comprising 6 geographically separate units. Overall, 3 units were involved in the outbreak: units A and B (16- and 15-bed hematology-oncology units) and unit C (a 15-bed bone marrow transplant [BMT] unit). Patients on these units undergo admission and weekly routine active surveillance for CRE. In July 2016, the hospital epidemiology and infection control staff was alerted by the microbiology laboratory that 2 patients had phenotypically identical CRE *K. pneumoniae* with similar antimicrobial susceptibility profiles in perirectal surveillance cultures. Pulsed-field gel electrophoreses (PFGE) confirmed the same strain. Epidemiological investigation revealed that the patients had consecutively occupied the same patient room in unit B 2 months prior: Patient 2 was admitted to the room 3 days after patient 1 was discharged. Patients 1 and 2 had negative admission and weekly perirectal surveillance cultures preceding the surveillance cultures that grew KPC-KP (13 and 36 days after admission, respectively), indicating probable acquisition during their hospital stays. Outbreak investigation and risk mitigation strategies were initiated.

Case-finding strategies included provider notification to identify suspected cases, microbiology laboratory request to alert for all KPC-KP isolates, and point-prevalence survey. On units B and C, where case patients were located, perirectal swabs were obtained from 24 patients, urine was obtained from 3 patients, and wound cultures were obtained from 2 patients. Contact-based precautions were used for all patients until point-prevalence results were obtained to interrupt potential transmissions from undetected carriers. There were no positive results. However, 2 other cases of KPC-KP were identified through clinician and laboratory notification; patient 3, who was readmitted to unit C with a positive blood culture and patient 4, who had not been recently admitted but who had had routine perirectal surveillance with KPC-KP during a prior admission to unit A. PFGE again confirmed the same strain. Whole-genome sequencing of the 4 case-patient isolates revealed a clonal *K. pneumoniae* strain producing KPC-2, CTX-M-15, TEM-1B, SHV-28, and OXA-1 sequence type 15. Environmental samples were taken for culture (13 high-touch surfaces and 2 bathroom sink drains) from the rooms and bathrooms of patients 1 and 2. A molecularly identical KPC-KP grew in the culture from an IV pole sample in the room of patient 2. Multiple other gram-negative organisms were cultured from the drains in both patient bathrooms.

Mitigation strategies included reinforcement of strict compliance with infection control precautions, enhanced environmental cleaning and disinfection, universal contact

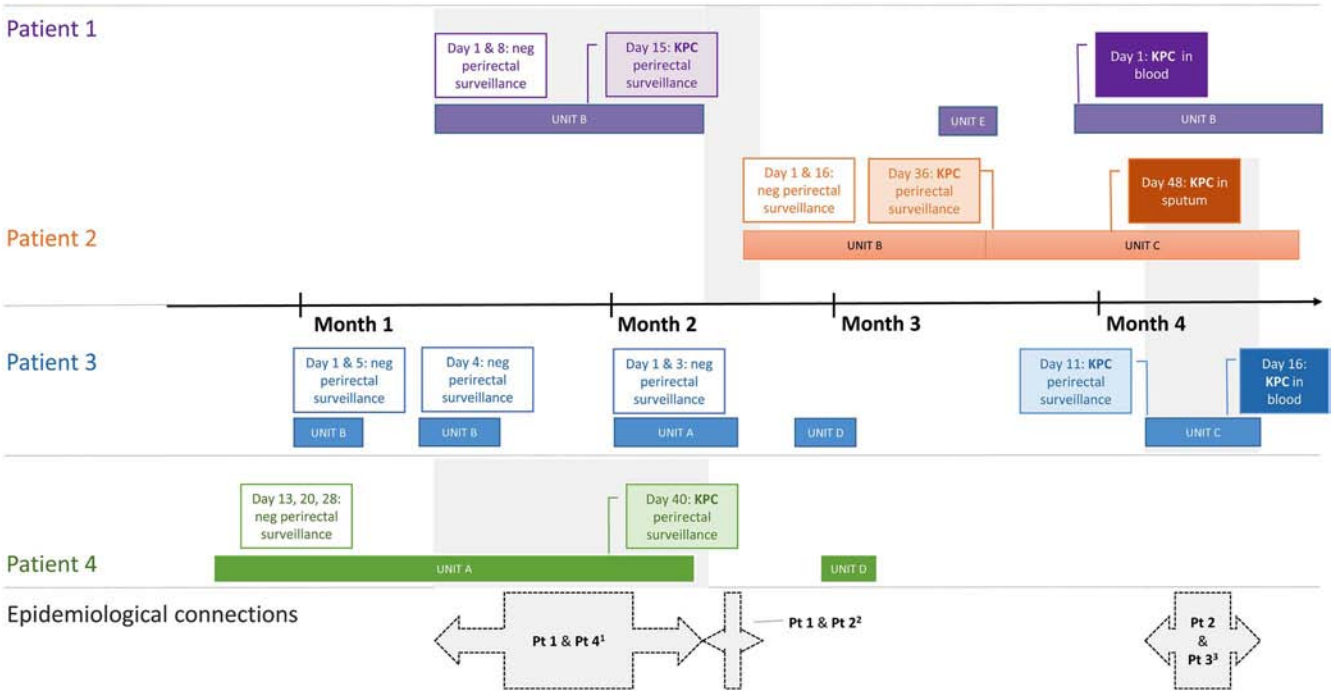

FIGURE 1. Timeline of KPC-KP patient inpatient stays and surveillance and clinical cultures. Patients are numbered in the order KPC-KP was identified. Solid lines denote inpatient stays. Inpatient days are numbered from the first inpatient day (ie, day 1) of each admission. Shaded areas indicate epidemiological connections: [1]Patients 1 and 4 were admitted to different units on the same floor. [2]Patient 2 occupied the same room after patient 1 was discharged. [3]Patients 2 and 3 were admitted to the same unit.

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precautions for units with case patients pending negative point-prevalence surveys and discharge of all case patients, dedicated equipment for case patients, and cohorting patients with dedicated nursing staff. Enhanced environmental disinfection using hydrogen-peroxide vapor technology was used post discharge for rooms inhabited by a known case patient and, when feasible, for all other rooms and shared equipment on affected units. Patient 3, who had significant comorbidities, died from progressive multiorgan failure with neutropenic sepsis and KPC-KP bacteremia. Two patients died from unrelated causes and 1 patient proceeded to successful BMT. Figure 1 shows the timing of cultures and epidemiological connections. Patients 1 and 2 had consecutive stays in the same room; patients 2 and 3 were on the same unit at the same time. The link between patient 4 and the other patients is not immediately evident. Patient 4 was an inpatient at the cancer center at the same time as patient 1 but on a different unit. The surveillance cultures of these 2 patients first grew KPC-KP on the same day. However, equipment and staff are not confined to 1 unit in the cancer center.

There are reports of KPC-KP outbreaks persisting for up to 2 years.[2–4] In 2011, the NIH Clinical Center had a largely ICU-based outbreak involving 19 patients who became colonized or infected with bla_{KPC}-positive *K. pneumoniae*, with 6 attributable deaths.[2] Patients with unrecognized CRE colonization may have served as undetected sources.[5,6] The interval between detection of colonization and infection in our outbreak ranged from 6 to 67 days. Active surveillance cultures at the cancer center allowed earlier identification and initiation of contact and other precautions rather than relying solely on clinical cultures, potentially contributing to the timely resolution of this outbreak. In addition, real-time molecular genotyping allowed for timely identification of full-genotype of β-lactamase genes, which was confirmed in additional patients.

This report highlights the importance of the hospital environment in the transmission of CRE as evidenced by 2 case patients having consecutive stays in the same hospital room and finding identical KPC-KP on the i.v. pole of a different room with a known infected patient. CRE have been found in patient rooms, including on equipment and sink drains.[2,4,7] Also, CRE can persist despite equipment and environmental cleaning and disinfection.[2,4] Our use of hydrogen peroxide vapor for shared equipment and once for each discharge room on affected units during the outbreak, rather than only rooms that had housed a known KPC-KP patient, may have shortened the outbreak period.

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