Contaminated Handwashing Sinks as the Source of a Clonal Outbreak of KPC-2-Producing *Klebsiella oxytoca* on a Hematology Ward

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We investigated sinks as possible sources of a prolonged *Klebsiella pneumonia* carbapenemase (KPC)-producing *Klebsiella oxytoca* outbreak. Seven carbapenem-resistant *K. oxytoca* isolates were identified in sink drains in 4 patient rooms and in the medication room. Investigations for resistance genes and genetic relatedness of patient and environmental isolates revealed that all the isolates harbored the *bla*KPC-2 and *bla*TEM-1 genes and were genetically indistinguishable. We describe here a clonal outbreak caused by KPC-2-producing *K. oxytoca*, and handwashing sinks were a possible reservoir.

The prevalence of carbapenem-resistant *Enterobacteriaceae* has increased over the past 10 years (1). The production of *Klebsiella pneumonia* carbapenemases (KPCs) is one way of conferring resistance to carbapenems belonging to Ambler class A (2). Nosocomial infections due to KPC-producing *Enterobacteriaceae* are of increasing concern, especially in long-term care facilities and intensive care units (ICUs) (2). In 2011, a nosocomial outbreak of KPC-producing *Klebsiella oxytoca* occurred in a medical ICU at the Medical University of Graz in Graz, Austria. Five patients, all of whom survived, were affected (3).

From October 2011 to October 2013, KPC-producing *K. oxytoca* isolates with resistance patterns identical to that of the outbreak strain were identified from 10 more patients in the Division of Hematology ward. As *K. oxytoca* isolates were detected over a 2-year period, a reservoir in the environment was suspected. Nosocomial outbreaks with *K. oxytoca* have been reported previously (4–10). In two publications, handwashing sinks, especially sink drains, were identified as the source of the outbreaks (9, 10). Therefore, we chose to focus our investigation on sinks. Fifty-eight swabs were taken from handwashing sink drains, 23 from handwashing sink overflows (not all sinks had overflows), and 19 from shower drains in the Division of Hematology. Swabs from sink surfaces were taken from contaminated sinks in a second round of testing.

Swabs were seeded onto MacConkey agar and chromID Carba (both from bioMérieux, Marcy l’Étoile, France) and analyzed according to microbiological standards (12). The isolates were screened for the presence of resistance genes with the DNA microarray-based Check-MDR 103 kit (Check-Points, Wageningen, Netherlands) according to the manufacturer’s protocol (http://www.check-points.com/support/manuals/). Sequencing of the detected carbapenemases and other beta-lactamase gene families was performed as previously described (13, 14). Repetitive sequence-based PCR (rep-PCR) was carried out with the DiversiLab system (bioMérieux, Nürtlingen, Germany). In addition, all the isolates were tested using multilocus sequence typing (MLST), as described previously (15).

Eleven *K. oxytoca* isolates were found in the sink drains. Seven screening swabs from dry surfaces are taken and tested routinely 4 times per year by our infection control team and have never yielded *K. oxytoca*. In addition, in two publications dealing with nosocomial outbreaks of *K. oxytoca*, swabs from dry surfaces in the environment surrounding the patients did not yield any *K. oxytoca* isolates. In contrast, *K. oxytoca* was identified in sinks and drains (9, 10). Therefore, we chose to focus our investigation on sinks. Fifty-eight swabs were taken from handwashing sink drains, 23 from handwashing sink overflows (not all sinks had overflows), and 19 from shower drains in the Division of Hematology. Swabs from sink surfaces were taken from contaminated sinks in a second round of testing.

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isolates showed multidrug resistance, including resistance to carbapenems, one was an extended-spectrum beta-lactamase (ESBL)-producing K. oxytoca isolate, and three isolates showed wild-type resistance. The 7 KPC-producing K. oxytoca isolates were found in rooms 23, 25, and 29 (double rooms housing 2 patients each with two sinks), room 37 (a single room), and 32A (a room in which medication is prepared by the nursing staff). Five KPC-producing K. oxytoca isolates were detected in sink drains (in rooms 23, 25, 29, 37, and 32A). One isolate was found in the sink overflow (room 37), and one was found in the shower drain (room 25). Swabs taken from sink surfaces in a second round did not yield any additional KPC-producing K. oxytoca isolates.

All the isolates from the patients and the seven isolates derived from sinks were multidrug resistant, exhibited susceptibility to amikacin, colistin, and fosfomycin only, and were found to be KPC producers. When microarray technology was used, blaKPC-2 and blaTEM were detected in all the isolates and were identified as blaKPC-2 and blaTEM by sequencing. rep-PCR revealed that all strains tested were indistinguishable with a similarity index of >97.5%. MLST yielded only one sequence type, ST4, for all the KPC-2-producing K. oxytoca isolates.

The starting point for this outbreak was a colonized patient from the ICU who later was transferred to the hematology ward. We hypothesize that in the case of this patient, KPC-2-producing K. oxytoca got into the sink most likely during personal hygiene activities or by the disposal of contaminated body fluids where it persisted. In the Division of Hematology, the water from the sink faucets directly hits the mesh that covers the sink drain. We hypothesize that some patients were colonized by contaminated aerosols when using the sinks for personal hygiene; 6 of 10 affected patients stayed in rooms with contaminated sinks, and 2 patients shared a room with a patient who later proved to be infected or colonized. We speculate that cross-contamination took place either by direct contact between the patients or through the hands of health care workers. The potential involvement of the health care workers is indicated by the fact that KPC-2-producing K. oxytoca was found in the sink of the room where medication is prepared by the health care staff.

As a consequence of the prolonged outbreak, the existing infection control measures (isolating colonized patients, enforcing hand hygiene measures, and cleaning the ward, particularly the sinks and equipment) were reinforced. An exchange of all the contaminated sinks was found in rooms 23, 25, and 29 (double rooms housing 2 patients each with two sinks), room 37 (a single room), and 32A (a room in which medication is prepared by the nursing staff). Five KPC-producing K. oxytoca isolates were detected in sink drains (in rooms 23, 25, 29, 37, and 32A). One isolate was found in the sink overflow (room 37), and one was found in the shower drain (room 25). Swabs taken from sink surfaces in a second round did not yield any additional KPC-producing K. oxytoca isolates.

As a consequence of the prolonged outbreak, the existing infection control measures (isolating colonized patients, enforcing hand hygiene measures, and cleaning the ward, particularly the sinks and equipment) were reinforced. An exchange of all the contaminated sinks was under way. Since October 2013, no more KPC-producing K. oxytoca isolates have been identified.

In conclusion, a clonal relationship between environmental isolates and patient strains was determined and pointed to hand-washing sinks as a possible reservoir for the prolonged nosocomial outbreak of KPC-2-producing K. oxytoca.

REFERENCES


