Endoscopy-associated transmission of carbapenem-resistant Klebsiella pneumoniae producing KPC-2 β-lactamase

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Sir,

Carbapenem resistance in Enterobacteriaceae due to the production of KPC carbapenemase is becoming a significant clinical problem.1,2 Klebsiella pneumoniae producing KPC carbapenemase (KPC-Kp) have been reported from many countries worldwide, including the USA, Colombia, Israel and Greece, and have been associated with increased hospital costs, increased length of stay and higher patient mortality.2-4 Worryingly, the epidemiology in the USA appears to be changing, since outbreaks have now been described in long-term acute care hospitals.5

The positive risk–benefit relationship of endoscopy interventions has been clearly established.6 Although the risk of nosocomial infections from endoscopes appropriately reprocessed is very low, inadequate reprocessing has been reported to be the source of outbreaks.6 We report here a nosocomial outbreak of KPC-Kp in France, with regional inter-hospital dissemination mediated by a contaminated duodenoscope.

An 85-year-old patient with bladder cancer was admitted to the medical intensive care unit (Hôpital de Bicêtre, France) for severe gastrointestinal bleeding. Upon arrival, screening samples (rectal swabs) for multidrug-resistant (MDR) bacteria, as previously described using chromID ESBL (bioMérieux, Marcy-l’Etoile, France), revealed the presence of an extended-spectrum β-lactamase (ESBL)-producing Escherichia coli. The patient underwent endoscopy to stop the bleeding. Five days later, in the course of the weekly MDR screening of the unit, he was screened positive for an MDR K. pneumoniae isolate. Antiibiogram determined by the disc diffusion method and MICs determined by Etest and interpreted according to the CLSI7 revealed that this MDR-Kp strain was resistant to penicillins, cephalosporins, fluoroquinolones, co-trimoxazole, rifampicin and tetracycline, but showed intermediate resistance to imipenem (MIC = 8 mg/L) and susceptibility to gentamicin (MIC = 2 mg/L) and colistin (MIC = 4 mg/L). The presence of the blaKPC-2 gene was identified by PCR and sequencing. The patient underwent surgery for gastrectomy and 2 weeks later had bacteraemia with KPC-Kp that was treated successfully with gentamicin (5 mg/kg/day) and colistin (50 000 IU/kg/day). Screening of patients in the same surgical unit for gut carriage of MDR bacteria identified two contact patients that were KPC-Kp(+), indicating probable nosocomial transmission.

Increased awareness, cohorting of these KPC-Kp(+) patients, dedicated nursing staff and reinforced hygiene precautions prevented further spread in the hospital. The period of time separating the initial screening of the patient and the diagnosis of KPC-Kp positivity may have contributed to the spread of KPC to other patients. Concomitantly, a patient from a neighbouring hospital who underwent endoscopy at the same gastroenterology ward was diagnosed to be KPC-Kp(+). The two patients had their endoscopy on separate days (2 weeks apart), but with the same endoscope. Bacterial cultures from the endoscope revealed KPC-Kp (10^2 cfu/100 mL of wash solution), Pseudomonas aeruginosa and other bacteria common in the digestive tract. Retrospective analysis of the patients that had gastroscopy with the same endoscope identified a Greek patient with KPC-Kp faecal carriage, who was transferred from a hospital in Chania (Crete, Greece) 2 months earlier. Following the endoscopic treatment of this patient, 17 patients, mostly from five regional hospitals (10 patients) and from Bicêtre hospital (7 patients), underwent gastroscopy with the same contaminated endoscope. Of these 17 patients, 10 could be screened; 6 were colonized with KPC-Kp and among these 2 developed KPC-Kp infections (one bacteraemia and one cellulitis).

References

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one bilioma). In addition, in one neighbouring hospital, cross-transmission has also been observed.

All the KPC-Kp isolates were identical to a previously identified K. pneumoniae ST258-type clone that is epidemic in Greece, Israel and the USA, as revealed by PFGE, plasmid analysis, multilocus sequence typing and Tn4401 transposon typing.2,8 PCR experiments, followed by sequencing, identified additional β-lactamase genes coding for the naturally occurring narrow-spectrum SHV-11, the plasmid-encoded narrow-spectrum TEM-1 and extended-spectrum SHV-12.

In France, KPC-Kp remain rare and, to date, have always been linked to a patient transfer from a country where KPC-Kp are endemic.2 This is the first KPC-Kp outbreak in the first worldwide to be linked to a contaminated endoscope.

Although the risk of endoscopy-related infection is low,6 changes to endoscope reprocessing, by replacing a glutaraldehyde decontamination bath with an automated peracetic acid washer (to prevent Creutzfeldt–Jacob transmission), may have been deleterious to the endoscope. However, careful checking of the endoscope by the instrument’s manufacturer did not reveal any obvious signs of degradation. Careful auditing of endoscope reprocessing revealed two possible explanations for the contamination: (i) the pre-wash of the endoscope may have been delayed 24 h, thus resulting in drying of the device; and (ii) after the peracetic wash, the drying procedure was not long enough for the novel automated washer, thus the endoscope was not completely dried. In light of this outbreak, new local guidelines for endoscope reprocessing have been established, taking into account the specificities of the new automated peracetic acid washer.

References

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