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Outbreak of Infection with a Multiresistant *Klebsiella pneumoniae* Strain Associated with Contaminated Roll Boards in Operating Rooms

Annemarie van 't Veen,[1] Anneke van der Zee,[1] Jolande Nelson,[1] Ben Speelberg,[2] Jan A. J. W. Kluytmans,[3] and Anton G. M. Buiting[1]*

Department of Clinical Microbiology, St. Elisabeth Hospital, Tilburg, The Netherlands[1]; Department of Intensive Care, St. Elisabeth Hospital, Tilburg, The Netherlands[2]; and Department of Clinical Microbiology, Amphia Hospital, Breda, The Netherlands[3]

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An outbreak with a multiresistant *Klebsiella pneumoniae* (MRKP) strain among seven patients admitted to the adult intensive care unit (ICU) of a regional teaching hospital in The Netherlands was investigated. Epidemiologic investigations revealed a short delay between an operation and the acquisition of the MRKP strain. A case-control study comprising 7 cases and 14 controls was conducted to identify the risk factors associated with the acquisition of the MRKP strain. An operation at each of two operation rooms was strongly associated with the acquisition of the MRKP strain: odds ratio of 36 (95% confidence interval, 2.7 to 481.2; $P = 0.003$, Fisher exact two-tailed test). Cultures of environmental specimens of the operation rooms revealed contamination of the roll boards used to transport patients from the bed to the operation table with the MRKP strains. Molecular genotyping of the isolates revealed clonal similarity between the isolates of the seven cases, isolates from environmental specimen cultures, and in addition, an MRKP isolate from a repatriated ICU patient from earlier that year. The outbreak ended after cleaning and replacement of the roll boards in the operation rooms and implementation of additional barrier precautions for colonized or infected patients. It was concluded that two operation rooms played a significant role in the transmission of an MRKP strain between ICU patients during the presented outbreak.

Klebsiella pneumoniae is a gram-negative bacterium that belongs to the family *Enterobacteriaceae* and that has become a well-recognized cause of nosocomial infections (12). The increasing prevalence of extended-spectrum β-lactamase (ESBL)-producing *K. pneumoniae* isolates is of particular concern. These isolates often express resistance to multiple antibiotics and, as a result, complicate antibiotic therapy and interfere with empirical therapy (18).

Hospital outbreaks due to multiresistant *K. pneumoniae* (MRKP) strains have been described throughout the world (7, 11, 13, 14, 15, 20). Outbreaks of *K. pneumoniae* infections have been associated with a wide variety of sources and reservoirs, including sinks (22), ultrasonography gel (6), an intravenous dextrose solution (9), cockroaches (3), food blenders (8), contaminated breast milk (5), bath soap (23), and water baths (1). However, the majority of outbreaks could not be associated with an environmental source. Patient-to-patient transmission via the hands of hospital staff was considered the main route of transmission (14).

The scope of the present study was to investigate a nosocomial outbreak at the intensive care unit (ICU) of St. Elisabeth Hospital, Tilburg, The Netherlands, caused by a *K. pneumoniae* strain that was resistant to gentamicin, tobramycin, and trimethoprim-sulfamethoxazole and that produced an ESBL. The incidence of multiple-resistant *Klebsiella* spp. at our hospital is low and corresponds to the low incidence of ESBL-producing members of the family *Enterobacteriaceae* reported in The Netherlands (21). The initial investigation of defining cases and possible sources was continued by a case-control study to identify the risk factors for the acquisition of an MRKP strain. In addition, molecular genotyping of clinical and environmental isolates was performed.

* Corresponding author. Mailing address: Department of Clinical Microbiology, St. Elisabeth Hospital, P.O. Box 747, 5000 AS Tilburg, The Netherlands. Phone: 31 (0)13 5392676. Fax: 31 (0)13 5391264. E-mail: a.buiting@elisabeth.nl.

MATERIALS AND METHODS

Hospital setting. The studies were performed in a regional 673-bed teaching hospital in The Netherlands. The hospital has an 18-bed ICU divided over three units on one floor. All beds are placed in single rooms; two rooms have an anteroom. The majority of the ICU patients are admitted for surgery, neurosurgery, or internal medicine. The hospital has a special function for neurosurgery and traumatology. The average length of stay in the ICU is 6.8 days for ventilated patients and 2.3 days for nonventilated patients.

Identification of outbreak. In November 2000, three patients in the ICU developed fatal sepsis caused by a *K. pneumoniae* strain expressing resistance to extended-spectrum cephalosporins by means of ESBL production and additional resistance to gentamicin, tobramycin, and trimethoprim-sulfamethoxazole. The infection control department was alerted, and the infection control practitioner conducted a visit to the ICU. A preliminary review of the patients' characteristics and a search of the microbiology reports of the ICU patients for specimens positive by culture for MRKP for the months of October and November were performed. It was noted that all fatal cases were operated on in one of two operation rooms (ORs; OR-X and OR-Y) within 5 days before the development of sepsis. In response, the infection control practitioner visited the OR complex and took environmental samples from OR-X and OR-Y.

Microbiological methods. In addition to the culture of samples for clinical indications, samples for surveillance cultures (respiratory material, urine, drains, and wounds) were obtained from all patients on artificial ventilation and were performed twice a week as standard procedure at the ICU. All identification and susceptibility tests were performed according to the CLSI (formerly the National Committee for Clinical Laboratory Standards). *K. pneumoniae* strains were iden-

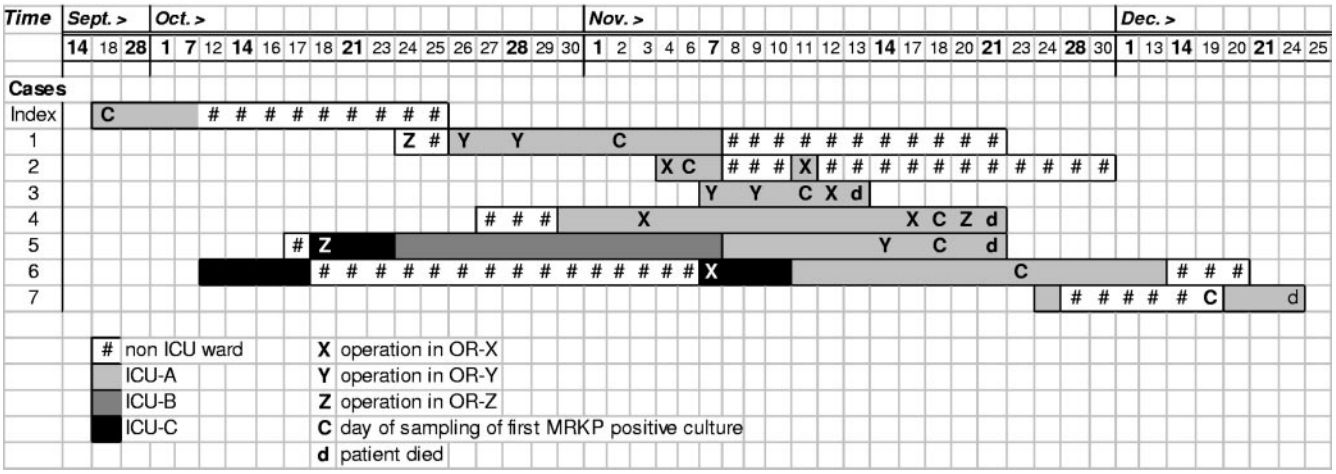

FIG. 1. Course of the outbreak presented in time. The shading of the bars is explained by the legend at the bottom of the figure.

tified by conventional CLSI-recommended biochemical methods, completed with an API 20E test kit (bioMerieux SA, Lyon, France). Antibiotic susceptibility testing was performed by a microdilution method in microtiter panels containing 11 antibiotics: amoxicillin, amoxicillin-clavulanate, and ceftazidime (GlaxoSmith-Kline CH, Zeist, The Netherlands); cefuroxime (Pharmachemie, Haarlem, The Netherlands); ceftriaxone (Roche, Mijdrecht, The Netherlands); ciprofloxacin (Bayer, Mijdrecht, The Netherlands); ofloxacin (Aventis Pharma, Hoevelaken, The Netherlands); trimethoprim-sulfamethoxazole and tobramycin (Sigma-Aldrich Chemie BV, Zwijndrecht, The Netherlands); meropenem (Astra Zeneca, Zoetermeer, The Netherlands); and gentamicin (Bufa BV, Uitgeest, The Netherlands). ESBL production was tested by disk diffusion tests (Oxoid Ltd., Basingstoke, England), as follows: cefpodoxime (10 μg), ceftazidime (30 μg), and cefotaxime (30 μg) were plated with and without clavulanate at 1 μg, 10 μg, and 10 μg, respectively. ESBL production was confirmed by a clavulanate-induced increase in the zone size of ≥5 mm. Environmental specimens for culture were taken with cotton swabs and were plated on blood agar and cultured for 2 days at 35°C. The choice of sites for culture was guided by preliminary investigations of the nature of the outbreak, information presented in the literature, and previous experiences in our hospital.

Case-control study. A case-control study was conducted to investigate risk factors and possible transmission routes in the November 2000 outbreak in the ICU. Cases were defined as patients who were admitted to the ICU for >24 h in November 2000 and who were positive for MRKP by culture of specimens taken between 1 November 2000 and 31 December 2000. Samples for culture were taken from specific infection sites or for surveillance, and samples from both colonized and infected patients were included. Case finding was conducted by a retrospective review of all microbiology culture reports from this period. Seven cases were recorded, and subsequently, 14 unmatched controls were randomly selected from a pool of all patients admitted to the ICU for >24 h in November 2000.

Data for the cases and the controls were collected by retrospective chart review and included age; gender; hospital admission dates; ICU admission dates; Apache score at the time of admission to the ICU; attending specialty; wards attended by the patient before and after admission to the ICU; units attended during the ICU admission; mortality at end of the ICU admission; mortality at end of the hospital admission; number of days of artificial ventilation; presence of urinary catheter, artery catheter and central venous catheter; presence of drains; presence of open wounds; surgery during hospital admission; OR number; operating team; antibiotic use; number of specimens presented for culture taken during the study period; date of first MRKP-positive culture; and the MRKP-positive culture sites.

Molecular genotyping. Molecular genotyping was performed by multienzyme amplified fragment length polymorphism (ME-AFLP) analysis, as described previously (24). In short, the genomic DNA of each isolate was extracted with a QiAmp DNA minikit (QIAGEN, Hilden, Germany). After addition of 5 μl of RNase (1 U; Sigma), the concentration of DNA was determined with a spectrophotometer (260 nm; Genequant, Pharmacia, United Kingdom). The restriction-ligation reaction was performed with 500 ng of DNA in a final volume of 30 μl. The reaction mixture consisted of the restriction enzymes EcoRI, PstI, XbaI, and NheI (2 U each; Roche Molecular Biochemicals); 3 μl of T4 ligase buffer; 1 U of T4 ligase (Roche Molecular Biochemicals); and 20 pmol of oligonucleotide adaptors. The restriction-ligation mixture was incubated at 37°C for 3 h. DNA was precipitated by using 2.5 M of ammonium acetate in 100 μl chilled absolute ethanol. After the DNA was washed with 70% ethanol, it was resuspended in 100 μl of TE (Tris-EDTA) buffer and stored at 4°C. PCRs were carried out with Ready-To-Go beads (Amersham Pharmacia, Little Chalfont, United Kingdom) supplemented with 1 μl $MgCl_2$ (25 mM). Each primer (10 pmol) and 100 ng of chromosomal template DNA were used in a final reaction volume of 25 μl. In a DNA thermocycler (GeneAmp PCR system 2400; Perkin-Elmer), the reaction mixtures were preheated for 4 min at 94°C. Thirty-three amplification cycles of 1 min at 94°C, 1 min at 60°C, and 2.5 min at 72°C were performed. Analysis of 10 μl of each PCR product was performed by agarose gel electrophoresis on a 1.5% agarose gel (MP agarose; Roche Molecular Biochemicals). Marker DNA (100-bp ladder; Gibco BRL, Life Technologies, Scotland) was loaded after every four samples. Analysis of the banding patterns on gel images was performed with the software program Gelcompar II, which is included in Bionumerics software (Applied Maths, Kortrijk, Belgium). Cluster analysis of the fingerprints was performed by the unweighted pair group method with arithmetic averages, and similarities between AFLP patterns were calculated by using the Pearson product-moment correlation coefficient (25). The patterns of the strains analyzed were considered identical when there was 90 to 100% homology.

A computer-based search for positive cultures of *K. pneumoniae* resistant to extended-spectrum cephalosporins and aminoglycosides at our hospital was performed for the years 1998, 1999, and 2000. Most of these isolates were stored at −80°C and could be included for molecular typing (one isolate per patient).

Data analysis. Univariate analysis was performed by Fisher's exact test for categorical variables, Student's *t* test, or the Mann-Whitney U test for continuous variables (EpiCalc 2000, version 1.02). Odds ratios were calculated when possible. Statistical significance was defined a priori as a P value of <0.05 (two tailed).

RESULTS

Description of cases and index. The course of the outbreak is depicted in Fig. 1, and patient characteristics are summarized in Table 1. Case 1 was admitted to the surgery ward on 24 October for an elective cholecystectomy. On 26 October she was admitted to ICU-A after a postoperative laparotomy in OR-Y and again on 29 October. An MRKP strain was cultured in the discharge from the abdominal wounds sampled on 2 November. Since it was a surveillance culture and there were no indications for infection, no antibiotic treatment for the MRKP infection was given. Case 1 recovered and was transferred to the surgery ward on 7 November.

Case 2 was admitted to ICU-A on 4 November after an

TABLE 1. Description of cases and index patient

Case	Sex[a]	Age (yr)	Reason for admission	Dates (mo/day) of ICU admission		MRKP isolation			Interval between surgery and MRKP acquisition (days)/OR	Outcome
				Admission	Discharge	First date (mo/day)	First positive specimen(s)	Other specimen(s) positive		
1	F	62	Laparotomy	10/26	11/7	11/2	Abdominal wounds		4/OR-X	Discharge
2	M	35	Subdural empyema	11/4	11/8	11/6	Sputum		2/OR-X	Discharge
3	M	34	Trauma	11/8	11/13	11/11	Blood	Abdominal discharge	2/OR-Y	Fatal sepsis
4	M	70	Pancreatitis	10/30	11/21	11/18	Discharge from ear	Blood and abdominal discharge	1/OR-X	Fatal sepsis
5	M	54	Esophagus resection	10/18	11/21	11/18	Discharge from thorax drain	Blood and sputum	5/OR-Y	Fatal sepsis
6	M	55	Laparotomy	11/7	12/14	11/23	Sputum	Urine	15/OR-X	Discharge
7	M	54	Subarachnoidal bleeding	11/24	11/28	12/19	Liquor	Urine	None	Fatal meningitis
Index	M	31	Trauma, repatriated	9/18	10/9	9/19	Blood and sputum	Urine	None	Discharge

[a] F, female; M, male.

operation for an acute subdural empyema in OR-X. An MRKP strain was recovered from a surveillance culture of sputum sampled on 6 November. No antibiotics were given. Case 2 was transferred to the neurosurgery ward on 8 November and was discharged from the hospital on 30 November. In between he received a second operation in OR-X on 11 November and postoperatively stayed in ICU-A one night.

Case 3 was a patient with multiple traumas admitted to ICU-A on 7 November. He received laporotomies three times: immediately after admission and on 9 and 12 November in OR-Y, OR-Y, and OR-X, respectively. An MRKP strain was isolated from blood sampled on 11 November and an abdominal discharge sampled on 12 November. Cefuroxime and tobramycin treatment was started as empirical therapy on 10 November and was changed to ofloxacin on 12 November. Case 3 died postoperatively of sepsis with multiorgan failure on 13 November.

Case 4, who had pancreatitis, was admitted to the internal medicine ward on 27 October and transferred to ICU-A on 30 October, where he stayed until his death on 21 November. A laporotomy was performed on 3 November in OR-X, on 17 November in OR-X, and on 20 November in OR-Z. Case 4 was treated with cefuroxime for an infected pancreas and was in stable condition. An MRKP isolate was first isolated from discharge from the ear sampled on 18 November and from abdominal rinsing fluid and abdominal drains sampled on 19 November. On November 21 he developed a fatal sepsis, and an MRKP strain was isolated from blood sampled on that day.

Case 5 was admitted to the surgery ward on 17 October; operated upon on 18 October (esophageal resection, OR-Z); and transferred to ICU-C, then to ICU-B (from 25 October), and then to ICU-A (from 7 November). On 14 November an explorative thoracotomy was performed in OR-Y. Case 5 received trimethoprim-sulfamethoxazole and vancomycin from 15 November. An MRKP strain was first isolated from discharge from a thorax drain sampled on 18 November, and 2 days later multiple thoracic drainage sites were positive for MRKP. On 21 November the patient developed a fatal sepsis and an MRKP strain was isolated from the blood sampled on that day.

Case 6 was admitted to ICU-C on 12 October after a thoracic operation in a different hospital. On 18 October he was transferred to the surgery department of St. Elisabeth Hospital but was readmitted to ICU-C on 7 November after an explorative laparotomy in OR-X. On 11 November, case 6 was transferred to ICU-A, where he stayed until 14 December. Since it was a surveillance culture and there were no indications for infection, no antibiotic treatment for the MRKP infection was given. Case 6 was discharged from the hospital on 20 December.

Case 7, who had subarachnoidal bleeding, was admitted to ICU-A from 24 to 28 November, to the neurosurgery ward from 28 November until 20 December, and to ICU-C from 20 December until death on 24 December. An MRKP strain was first isolated from cerebrospinal fluid sampled on 19 December. Case 7 was not operated upon but received a spinal catheterization for pain relief 6 days prior to the positive culture. This procedure was performed in the recovery ward of the OR complex by an anesthetist who had not been involved in any of the operations on the other cases. Case 7 was treated with meropenem but died as a result of infection with the MRKP strain.

The index patient was a trauma patient repatriated from Morocco and admitted to ICU-A on 18 September. The patient was not operated upon in our hospital. Respiratory specimens, blood, and a thorax drain sampled at admission were culture positive for an MRKP strain. Treatment was started with meropenem. This patient recovered and was transferred to the surgery ward on October 9. Barrier isolation was imposed at both the ICU and the surgery ward during admission of the index patient. Identification of the index case was inferred from molecular genotyping of the MRKP isolates from this patient. The typing patterns of the MRKP isolates from this patient were identical to the typing patterns of the outbreak strains.

Case-control study. Seven cases and 14 unmatched controls were defined, and the data for these individuals are summarized in Table 2. Comparable values of the baseline characteristics were found between the cases and the controls; these included gender, age, and Apache score. The mean length of

TABLE 2. Results of the case-control study

Characteristic	Controls (n = 14)	Cases (n = 7)	P value	Odds ratio (95% CI[a])
Male (no. [%])	10 (70)	6 (86)	1.00	
Age (yr [mean ± SD])	64 ± 19.3	52 ± 13.3	0.120	
APACHE score at ICU admission (mean ± SD)	22.3 ± 11.09	26.4 ± 5.77	0.274	
Admission				
Time in hospital before time in ICU (days [mean ± SD])	9.6 ± 26.30	4.4 ± 9.55	0.524	
No. (%) of patients immediately admitted to ICU	6 (43)	3 (43)	0.659	
Duration of ICU admission (days [mean ± SD])	7.3 ± 5.20	17.0 ± 14.33	0.127	
Duration of hospital admission (days [mean ± SD])	33.7 ± 27.47	31.1 ± 19.18	0.806	
Specialty (no. [%] of patients)				
Surgery	8(57)	4 (57)	1.00	
Internal medicine	3 (21)	1 (14)	1.00	
Neurosurgery	3 (21)	2 (29)	1.00	
Admission to ICU unit[b](no. [%] of patients)				
A	7 (50)	7 (100)	0.047	-
B	3 (21)	1 (14)	1.00	
C	6 (43)	3 (43)	1.00	
Artificial ventilation				
No. [%] of patients	8 (57)	7 (100)	0.061	
Duration (days [mean ± SD])	3.9 ± 4.69	11.3 ± 12.46	0.389	
Catheters (no. [%] of patients)				
Urinary	14 (100)	7 (100)		
Arterial	10 (71)	6 (86)	0.624	
Central venous	7 (54)	6 (86)	0.173	
Drains (no. [%] of patients)	2 (14)	5 (71)	0.017	15.00 (1.63–138.16)
Open wounds (no. [%] of patients)	0 (0)	3 (43)	0.026	
Operations				
No. (%) of patients operated upon	9 (64)	6 (86)	0.612	
No. of operations (mean ± SD)	0.9 ± 1.07	2.3 ± 1.38	0.046	
No. (%) of patients operated on in OR-A or OR-B	2 (14)	6 (86)	0.003	36.00 (2.69–481.23)
Antibiotic use (no. [%] of patients)	9 (64)	7 (100)	0.123	
Mortality during hospital admission (no. [%] of patients)	3 (21)	4 (57)	0.156	

[a] CI, confidence interval.
[b] Admission to more than one ICU is possible.

stay in the ICU, in total (17.0 ± 14.33 days) and prior to the acquisition of an MRKP strain (12.0 ± 11.18 days), was longer for the cases than for the controls (7.3 ± 1.39 days). Also, the cases received ventilatory support, both in total and prior to the acquisition of MRKP, for longer periods than controls. None of these differences was statistically significant (Table 2). An operation performed in OR-X or OR-Y was found to have the strongest association with the acquisition of an MRKP strain, with an odds ratio of 36.0 (95% confidence interval, 2.7 to 481.2; $P = 0.003$). All six cases had undergone an operation before the acquisition of an MRKP strain (Table 1). These six operations were performed by 17 operation team members in total, 12 of which attended only one operation and 5 of which attended two operations. The average number of days between the operation and the subsequent culturing of MRKP was 4.8 (standard deviation, ±5.19; Table 1).

The numbers of patients who had undergone an operation were 6 of 7 cases and 9 of 14 controls. Among the cases, the mean number of operations prior to the acquisition of an MRKP strain (1.7 ± 1.25 days) and during hospitalization (2.3 ± 1.38 days) was higher than that for the controls (0.9 ± 1.07 days). A significant difference in the mean total number of operations during the hospital admission was found ($P = 0.022$, t test), but when that number was corrected for the number of operations prior to MRKP acquisition, statistical significance was not reached (Table 2). Among the cases, three patients died in the ICU, whereas one patient in the control group died in the ICU. This difference did not reach statistical significance ($P = 0.088$). However, when statistical analysis was restricted to patients who had had an operation, i.e., six cases and nine controls, it was found that an operation in OR-X or OR-Y was associated with an increased risk for mortality in the ICU ($P = 0.044$). The presence of drains ($P = 0.017$), the presence of open wounds ($P = 0.026$), and admission to ICU-A ($P = 0.047$) also had a significant association with the acquisition of an MRKP strain (Table 2).

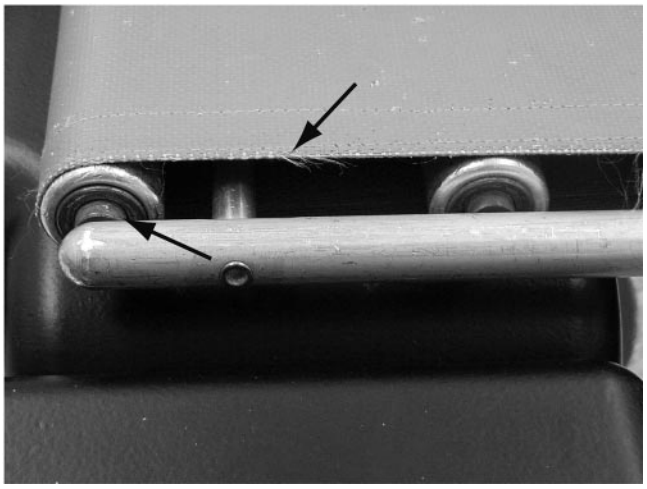

FIG. 2. Contaminated roll boards used to transport patients from their bed to the operation table and vice versa. Note the broken areas and the small ball bearings present (arrows).

Infection control measures. Because the fatal cases had all had an operation in OR-X or OR-Y less than 5 days prior to the development of sepsis caused by an MRKP strain, environmental samples were taken from these ORs. In both OR-X and OR-Y, *K. pneumoniae* was isolated from the roll boards used to transport patients from the bed to the operation table (Fig. 2). The multiresistant *K. pneumoniae* outbreak strain was defined as being resistant to aminoglycosides and trimethoprim-sulfamethoxazole and ESBL positive. Resistance to ciprofloxacin and ofloxacin varied from susceptible to resistant both within patients and between patients. The roll boards were disinfected thoroughly or replaced, if it was worn out. In addition, barrier precautions for colonized or infected patients were reinforced on the ICU. The outbreak ended after these interventions were implemented.

Molecular genotyping. Figure 3 shows the results of molecular genotyping of the *K. pneumoniae* strains. The ME-AFLP patterns of MRKP isolates from cases 1 to 7 and the two MRKP isolates from the OR room roll boards (OR-X and OR-Y) were grouped within one cluster, with similarities ranging from 90 to 100%. A multiresistant *K. pneumoniae* strain isolated from a patient admitted to the ICU in September fell

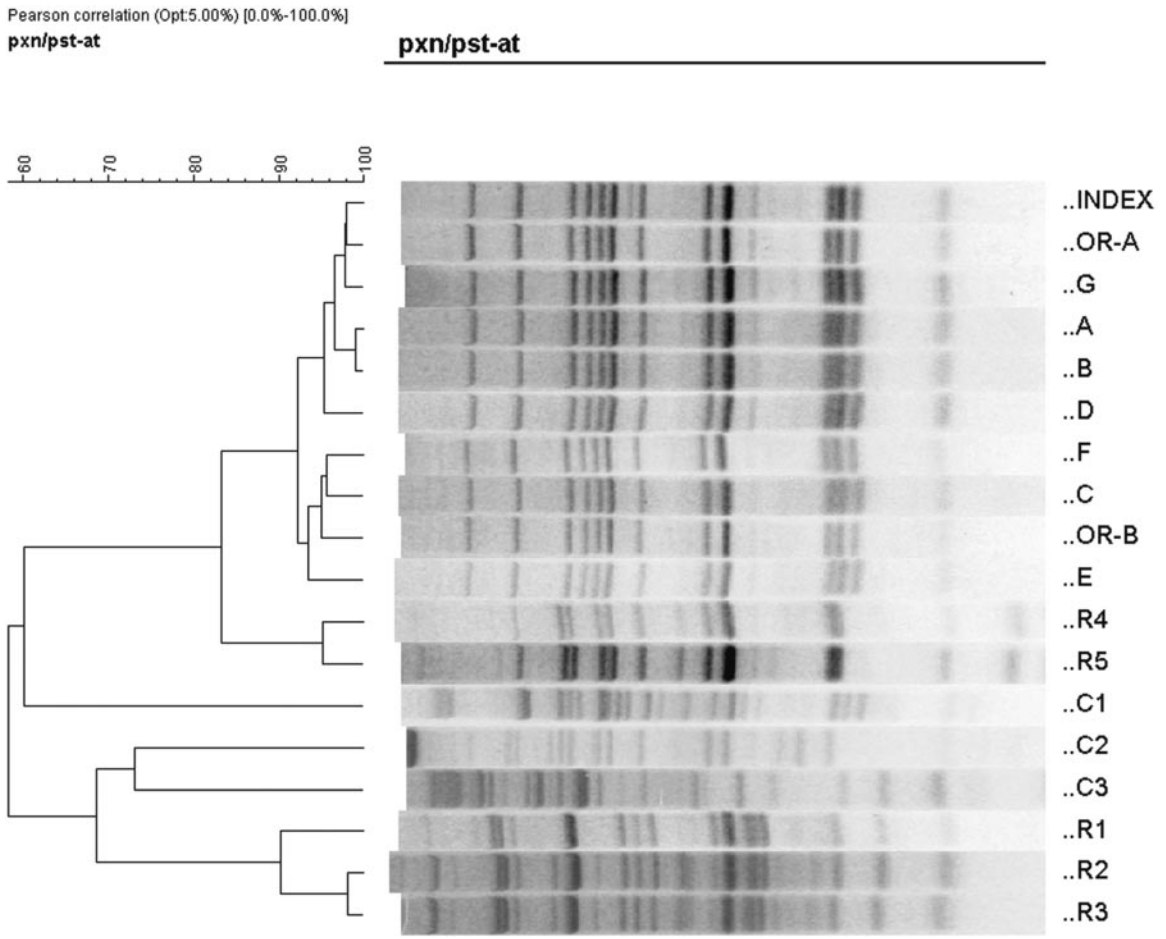

FIG. 3. Dendrogram of genotyping results. A to G represent the ME-AFLP patterns of the MRKP strains cultured from cases 1 to 7 and the index patient presented in Table 1, respectively; OR-A and OR-B represent the patterns of the MRKP strains isolated from the roll boards in OR-X and OR-Y, respectively; R1 to R5, MRKP strains isolated earlier in 2000 (R1 to R4) or isolated beginning in 2001 (R5); C1 to C3, controls that represent *Klebsiella pneumoniae* isolates with a common antibiotic susceptibility pattern.

within the same cluster (index case). The ME-AFLP patterns of five other *K. pneumoniae* strains resistant to extended-spectrum cephalosporins and aminoglycosides all fell in different clusters. Four of these strains (strains R1 to R4) were isolated earlier in 2000, and one strain (strain R5) was isolated in January 2001. ME-AFLP patterns C1 to C3 are the patterns of susceptible *Klebsiella pneumoniae* strains and served as controls (Fig. 3).

DISCUSSION

An outbreak caused by a multiresistant *Klebsiella pneumoniae* strain occurred in the ICU of the St. Elisabeth Hospital in Tilburg, The Netherlands. Seven patients at the ICU became colonized or infected, and in four cases the death of the patient was considered to be caused by infection with the outbreak strain.

A large number of risk factors for the acquisition of ESBL-producing *K. pneumoniae* strains have been reported, including the length of hospital stay (2, 10), the length of ICU stay (4), mechanical ventilation (2, 10, 17), urinary catheterization (2, 10), arterial catheterization (2), the presence of a central venous catheter (10, 19), prior antibiotic treatment (10, 16, 19), and more.

In the present study, a number of risk factors for the acquisition of the MRKP strain agreed with those that had been reported previously. We found that (i) the mean length of stay at the ICU was longer, (ii) the number of patients receiving ventilatory support was higher, and (iii) the number of central venous catheters and artery catheters was higher in cases than in the controls.

Our case-control study showed that an operation in OR-X or OR-Y was associated with a 36-fold increased risk of acquiring an MRKP strain and hence presented a significant risk factor for mortality due to infection with MRKP. Also, a short period of time (mean number of days, 4.8) between an operation in OR-X or OR-Y prior to the acquisition of MRKP was found. The contribution of OR-X and OR-Y to the outbreak was supported by the recovery of MRKP from cultures of environmental samples of the roll boards in OR-X and OR-Y. The similarity of these environmental isolates to the outbreak strain was confirmed by molecular typing. Moreover, the outbreak was contained after thorough disinfection and/or replacement of the roll boards in the ORs. Thus, the present study indicated a transmission route which differentiated this outbreak from previously reported outbreaks with multiresistent *Klebsiella pneumoniae* strains.

A minority of the cases could not be related to OR-X or OR-Y. One case had not been operated upon. In a second case, the time between the operation and the first isolation of MRKP took 15 days. The index case patient also did not have an operation, and other than admission to ICU-A, no clear connection could be found between this patient and any of the cases.

It is conceivable that secondary to the transmission at the ORs, cross contamination within the ICU occurred. Six of the seven cases were admitted to unit ICU-A at the time that MRKP was isolated for the first time. In the case-control study, ICU-A was found to be a risk factor for the acquisition of the MRKP strain. Unfortunately, no environmental samples for culture were taken from ICU-A. If, however, an environmental source was present in ICU-A and this source did play a significant role in the outbreak, the outbreak would probably not have been controlled after the interventions taken in the ORs.

To our knowledge, this study is the first to report on the contamination of roll boards in ORs with multiresistant *Enterobacteriaceae*. Roll boards usually do not come into contact with the intact mucosal barriers or sterile tissue, and these utensils are expected to impose only a small risk for the transmission of microorganisms. Prompt cleaning and decontamination are done on a regular basis, e.g., when blood or other potentially infectious material is spilled on the surface. This procedure is regarded to be sufficient and according to Dutch and international guidelines for infection control (2a).

From the results of this study it may be recommended that the construction of roll boards be improved to facilitate effective decontamination; they should have smooth surfaces instead of loosening fibers and open ball bearings. Alternatively, more frequent cleaning and disinfection procedures for roll boards in ORs might be considered to decrease the risk of unwanted infection by a pathogen from environmental sites.

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REFERENCES

1. **Albesa, I., A. J. Eraso, C. I. Frigerio, and A. M. Lubetkin.** 1980. Outbreak of hospital infection, due to members of the Klebsielleae tribe, in an intensive care unit for infants. Rev. Argent. Microbiol. **12:**39–43.
2. **Bisson, G., N. O. Fishman, J. Baldus Patel, P. H. Edelstein, and E. Lautenbach.** 2002. Extended-spectrum β-lactamase-producing Escherichia coli and Klebsiella species: risk factors for colonization and impact of antimicrobial formulary interventions on colonization prevalence. Infect. Control Hosp. Epidemiol. **23:**254–260.

2a. **Centers for Disease Control and Prevention.** 2005. Guidelines for infection control in health care facilities. Centers for Disease Control and Prevention, Atlanta, Ga. [Online.] www.wip.nl, guideline 3b, www.cdc.gov.

3. **Cotton, M. F., E. Wasserman, C. H. Pieper, D. C. Theron, D. van Tubbergh, G. Campbell, F. C. Fang, and J. Barnes.** 2000. Invasive disease due to extended spectrum beta-lactamase-producing Klebsiella pneumoniae in a neonatal unit: the possible role of cockroaches. J. Hosp. Infect. **44:**13–17.
4. **De Champs, C., M. P. Sauvant, C. Chanal, D. Sirot, N. Gazuy, R. Malhuret, J. C. Baquet, and J. Sirot.** 1989. Prospective survey of colonization and infection caused by expanded-spectrum-beta-lactamase-producing members of the family *Enterobacteriaceae* in an intensive care unit. J. Clin. Microbiol. **27:**2887–2890.
5. **Donowitz, L. G., F. J. Marsik, K. A. Fisher, and R. P. Wenzel.** 1981. Contaminated breast milk: a source of Klebsiella bacteremia in a newborn intensive care unit. Rev. Infect. Dis. **3:**716–720.
6. **Gaillot, O., C. Maruéjouls, E., Abachin, F. Lecuru, G. Arlet, M. Simonet, and P. Berche.** 1998. Nosocomial outbreak of *Klebsiella pneumoniae* producing SHV-5 extended-spectrum β-lactamase, originating from a contaminated ultrasonography coupling gel. J. Clin. Microbiol. **36:**1357–1360.
7. **Gonzalez-Vertiz, A., D. Alcantar-Curiel, M. Cuauhtli, C. Daza, C. Gayosso, G. Solache, C. Horta, F. Mejia, J. I. Santos, and C. Alpuche-Aranda.** 2001. Multiresistant extended-spectrum β-lactamase-producing *Klebsiella pneumoniae* causing an outbreak of nosocomial bloodstream infection. Infect. Control Hosp. Epidemiol. **22:**723–725.
8. **Kiddy, K., E. Josse, and N. Griffin.** 1987. An outbreak of serious Klebsiella infections related to food blenders. J. Hosp. Infect. **9:**191–193.
9. **Lalitha, M. K., J. Kenneth, A. K. Jana, M. V. Jesudason, K. A. Kuruvilla, K. Jacobson, I. Kühn, and G. Kronvall.** 1999. Identification of an IV-dextrose solution as the source of an outbreak of Klebsiella pneumoniae sepsis in a newborn nursery. J. Hosp. Infect. **43:**70–72.
10. **Lin, M.-F., M.-L. Huang, and S.-H. Lai.** 2003. Risk factors in the acquisition of extended-spectrum β-lactamase *Klebsiella pneumoniae*: a case-control study in a district teaching hospital in Taiwan. J. Hosp. Infect. **53:**39–45.
11. **Lucet, J.-C., D. Decré, A. Fichelle, M.-L. Joly-Guillou, M. Pernet, C. Deblangy, M.-J. Kosmann, and B. Régnier.** 1999. Control of a prolonged outbreak of extended-spectrum β-lactamase-producing Enterobacteriaceae in a university hospital. Clin. Infect. Dis. **29:**1411–1418.

12. **Martinez-Aguilar, G., C. M. Alpuche-Arande, C. Anaya, D. Alcantar-Curiel, C. Gayosso, C. Daza, C Mijares, J. C. Tinoco, and J. I. Santos.** 2001. Outbreak of nosocomial sepsis and pneumonia in a newborn intensive care unit by multiresistant extended spectrum β-lactamase-produciing *Klebsiella pneumoniae*: high impact on mortality. Infect. Control Hosp. Epidemiol. **22:**725–728.
13. **Pagani, L., M. Perilli, R. Migliavacca, F. Luzarro, and G. Amicosante.** 2000. Extended-spectrum TEM- and SHV-type β-lactamase-producing Klebsiella pneumoniae strains causing outbreaks in intensive care units in Italy. Eur. J. Clin. Microbiol. Infect. Dis. **19:**765–772.
14. **Patterson, D. L., and V. L. Yu.** 1999. Extended-spectrum β-lactamases: a call for improved detection and control. Clin. Infect. Dis. **29:**1419–1422.
15. **Peña, C., M. Pujol, C. Ardanuy, A. Ricart, R. Pallarés, J. Liñares, J. Ariza, and F. Gudiol.** 2001. An outbreak of hospital-acquired Klebsiella pneumoniae bacteraemia, including strains producing extended-spectrum β-lactamase. J. Hosp. Infect. **47:**53–59.
16. **Pessoa-Silva, C. L., B. Meurer Moreira, V. Cãmara Almeida, B. Flannery, M. C. Almeida Lins, J. L. Mello Sampaio, L. Martins Teixeira, L. E. Vaz Miranda, L. W. Riley, and J. L. Gerberding.** 2003. Extended-spectrum β-lactamase-producing *Klebsiella pneumoniae* in a neonatal intensive care unit: risk factors for infection and colonization. J. Hosp. Infect. **53:**198–206.
17. **Piroth, L., H. Aubé, J.-M. Doise, and M. Vincent-Martin.** 1998. Spread of extended spectrum β-lactamase producing *Klebsiella pneumoniae*: are β-lactamase inhibitors of therapeutic value? Clin. Infect. Dis. **27:**76–80.
18. **Podschun, R., and U. Ullmann.** 1998. *Klebsiella* spp. as nosocomial pathogens: epidemiology, taxonomy, typing methods, and pathogenicity factors. Clin. Microbiol. Rev. **11:**589–603.
19. **Schiappa, D. A., M. K. Hayden, M. G. Matushek, F. N. Hashemi, J. Sullivan, K. Y. Smith, D. Miyashiro, J. P. Quinn, R. A. Weinstein, and G. M. Trenholme.** 1996. Ceftazidime-resistant *Klebsiella pneumoniae* and *Escherichia coli* bloodstream infection: a case-control and molecular epidemiologic investigation. J. Infect. Dis. **174:**529–536.
20. **Siu, L. K., P.-L. Lu, P.-R. Hsueh, F. M. Lin, S.-C. Chang, K.-T. Luh, M. Ho, and C.-Y. Lee.** 1999. Bacteremia due to extended-spectrum β-lactamase-producing *Escherichia coli* and *Klebsiella pneumoniae* in a pediatric oncology ward: clinical features and identification of different plasmids carrying both SHV-5 and TEM-1 genes. J. Clin. Microbiol. **37:**4020–4027.
21. **Stobberingh, E. E., J. Arends, J. A. A. Hoogkamp-Korstanje, W. H. F. Goessens, M. R. Visser, A. G. M. Buiting, Y. J. Debets-Ossenkopp, R. J. van Ketel, M. L. van Ogtrop, L. J. M. Sabbe, G. P. Voorn, H. L. J. Winter, and J. H. van Zeijl.** 1999. Occurence of extended-spectrum beta-lactamases (ESBL) in Dutch hospitals. Infection **27:**348–354.
22. **Su, L.-H., H.-S. Leu, Y.-P. Chiu, J.-H. Chia, A.-J. Kuo, C.-F. Sun, T.-Y. Lin, T.-L. Wu, and the Infection Control Group.** 2000. Molecular investigation of two clusters of hospital-acquired bacteraemia caused by multi-resistant *Klebsiella pneumoniae* using pulsed-field gel electrophoresis and infrequent restriction site PCR. J. Hosp. Infect. **46:**110–117.
23. **Szabó, D., Z. Filetóth, J. Szentandrássy, M. Némedi, E. Tóth, C. Jeney, G. Kispál, and F. Rozgonyi.** 1999. Molecular epidemiology of a cluster of cases due to *Klebsiella pneumoniae* producing SHV-5 extended-spectrum β-lactamase in the premature intensive care unit of a Hungarian hospital. J. Clin. Microbiol. **37:**4167–4169.
24. **van der Zee, A., N. Steer, E. Thijssen, J. Nelson, A. van 't Veen, and A. Buiting.** 2003. Use of multienzyme multiplex PCR amplified fragment length polymorphism typing in analysis of outbreaks of multiresistant *Klebsiella pneumoniae* in an intensive are unit. J. Clin. Microbiol. **41:**798–802.
25. **Vauterin, L., and P. Vauterin.** 1992. Computer-aided objective comparison of electrophoresis patterns for grouping and identification of micro-organisms. Eur. Microbiol. **1:**37–42.