



Short report

The sink as a correctable source of extended-spectrum β -lactamase contamination for patients in the intensive care unit

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SUMMARY

Between December 2010 and April 2012, intensive care unit (ICU) patients in our hospital were infrequently colonized with extended-spectrum β -lactamase-positive bacteria (ESBLs). We hypothesized that these ESBLs originated from patients' room sinks, and this was prospectively investigated by weekly culturing of patients and sinks during a 20-week period. ESBLs were isolated from all 13 sinks. Four patients became colonized with ESBLs that were genetically identical to ESBLs that had previously been isolated from the sink. One of these patients died of pneumonia caused by the ESBL. Transmission from sinks to patients was stopped by integrating self-disinfecting siphons to all sinks on the ICU.

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Introduction

During 2011–2012 in the intensive care unit (ICU) of Deventer Hospital, mechanically ventilated patients were screened twice per week according to our protocol for selective gut decontamination. Infrequently, extended-spectrum β -lactamase-positive bacteria (ESBLs) were isolated in these patients' cultures, whose preceding cultures had been negative. These strains were considered to have been acquired on

the ICU, but since they belonged to different species with different antibiograms, and since there was no evident relation in time, patient-to-patient transmission was not suspected. The primary source of these ESBLs was not clear; they could have been endogenously selected by antibiotic pressure or acquired exogenously by the hands of medical personnel or medical equipment.^{1–3}

Gram-negative bacteria have been described as the cause of outbreaks on the ICU; they have been isolated from sinks, and in quantities much higher than found in other niches in patient rooms.^{4–6} In previous studies it has been shown that patients can become colonized, and even infected, by bacteria from those sinks.⁷

The aim of our study was to investigate whether patients in the ICU could have been colonized with ESBLs originating from sinks in the patient rooms, and, if this was the case, whether self-disinfecting siphons could be an effective intervention to prevent future transmissions of ESBLs.

Methods

Setting

Deventer Hospital is a 500-bed regional hospital with 21,000 admissions per year. In 2011, there were 3293 ICU patient-days and 1337 ventilation-days. The ICU contains seven single and four double patient rooms each with a sink. The two single rooms have an anteroom with an extra sink, so in total 13 sinks are present. Sinks are used for washing hands and medical instrumentation before disinfection, and flushing water that had been used for washing patients. They are cleaned with water and soap.

Environmental samples of sinks

From June until October 2011, sinks were screened once per week for 20 weeks to detect the presence of ESBLs. Culture samples were obtained by swabbing the drainage canal to a maximum depth of 4 cm with a Transwab[®] (Medical Wire & Equipment, Corsham, England).

Clinical isolates

In 2011–2012, screening cultures were routinely obtained from mechanically ventilated patients twice per week from sputum, throat and rectum, with Transwab. Sputum and rectum samples were taken from all patients present on a designated day once a month. Cultures were investigated at the medical microbiological laboratory for pathogenic or multi-resistant bacteria, including ESBLs. For our study, ESBLs that were cultured in the first 48 h of admission to the ICU were excluded, as they were probably endogenous. The first ESBL-positive isolate per patient was routinely stored at -80°C .

Microbiological methods

Sink specimens were investigated for the presence of ESBLs with Brilliance[™] ESBL Agar plates (Oxoid, Basingstoke, UK). Patient cultures were investigated following routine laboratory protocols. ESBL-suspected colonies were



Figure 1. The self-disinfecting Medizinische Hygiene-Siphon BIOREC (MoveoMed, Dresden, Germany).

identified and investigated for antibiotic resistance with Vitek-2[®] (bioMérieux, Marcy-l’Etoile, France). The presence of ESBL was confirmed by cefotaxime, ceftazidime, cefepime with and without clavulanic acid E-test[®] (AB Biodisk, Solna, Sweden). All ESBLs were stored at -80°C . Non-specific cultures of sinks were investigated with Columbia sheep blood agar (Oxoid).

Molecular characterization

All ESBL-positive patient isolates were genetically typed with amplified fragment length polymorphism (AFLP).⁸ For every newly identified ESBL-positive patient from June 2011 ($N = 9$), the isolated strains ($N = 10$) were compared with all ESBL-positive strains that had been isolated from the sink of that room. If the ESBL strains of patient and sink were identical on species level and the resistance pattern showed no more than two differences in resistance pattern, the strains from the sink were also investigated with AFLP.

Intervention

In April 2012, all 13 siphons from sinks in the ICU patient rooms and five siphons from sinks at other locations where medical workers wash their hands frequently (two toilets, the medication room, the scullery room and the staff room) were replaced by Medizinische Hygiene-Siphon BIOREC (MoveoMed, Dresden, Germany) (Figure 1). These siphons prevent the formation of a biofilm by means of permanent physical disinfection (heating and ultrasound), electromagnetic cleansing and antibacterial coating.

In order to monitor the effect of the intervention, all 18 sinks were sampled for the presence of ESBL one, two, three, four, six, and eight months after the intervention. During month 8, samples were cultured non-selectively to determine the whole microbial flora present in the sinks.

Table 1

Extended-spectrum β -lactamase-suspected bacterial isolates found in screening cultures ($N = 20$) obtained weekly from June until October 2011 from 13 sinks in Deventer Hospital ICU

Bacterial species isolated from sink	ICU patient rooms												
	R1	R2	R2A	R3	R4	R4A	R5	R6	R7	R8	R9	R10	R11
<i>Citrobacter freundii</i>	3	6	4	5	3	14	0	0	1	4	2	1	1
<i>Citrobacter amalonaticus</i>	0	1	0	0	0	0	0	0	0	0	0	0	0
<i>Enterobacter aerogenes</i>	0	0	0	0	0	0	2	0	0	0	0	0	0
<i>Enterobacter amnigenus</i>	0	0	0	0	0	0	0	0	0	0	0	1	0
<i>Enterobacter cloacae</i>	17	15	15	8	8	2	19	10	23	17	10	8	15
<i>Escherichia coli</i>	2	5	1	6	6	0	0	0	1	0	8	5	0
<i>Escherichia hermannii</i>	0	0	0	0	0	0	1	6	0	0	0	0	0
<i>Klebsiella oxytoca</i>	7	1	0	3	17	1	4	4	5	0	3	10	0
<i>Klebsiella ozaenae</i>	3	0	0	0	0	0	0	0	0	0	0	0	0
<i>Klebsiella pneumoniae</i>	0	7	1	11	0	0	1	0	3	0	0	1	6
<i>Kluyvera species</i>	0	0	0	0	1	2	0	0	0	0	0	0	0
<i>Raoultella planticola</i>	0	9	0	1	0	0	0	0	0	0	7	0	5
<i>Serratia marcescens</i>	0	1	0	10	0	0	0	0	0	0	0	0	0

ICU, intensive care unit; R, room; A, anteroom.

Strains were resistant to third-generation cephalosporins and were subsequently investigated for the presence of extended-spectrum β -lactamase phenotype. *Enterobacter cloacae* was the predominant species.

Clinical course

Medical records for all ESBL-positive patients were scrutinized to assess whether colonization with ESBL resulted in infection.

Results

Sinks

Every patient room sink contained one or more ESBLs on at least one time-point; 247 out of 260 cultures (95%) were positive (Table 1). *E. cloacae* was the predominant strain. ESBLs were also isolated from sinks of one toilet, scullery room and staff room.

After the intervention, ESBLs have not been found in control cultures of any ICU sink. Non-selective cultures eight months after the intervention showed no growth in 11 out of 18 sinks. Positive cultures contained small amounts of coagulase staphylococci and *Bacillus* spp.

Clinical isolates

From January 2011 to March 2012, 18 patients became colonized with 23 ESBL-positive strains during their stay on the ICU. Three patients were colonized with two ESBLs and one patient by three ESBLs (Figure 2).

Molecular characterization

The 23 ESBLs belonged to 11 unique AFLP genotypes; 19 ESBLs belonged to seven smaller AFLP clusters ($N = 2-4$); the remaining four showed a unique AFLP pattern (see Figure 2).

Four patients (10, 12, 14, and 17) were colonized by ESBLs that had been isolated from the sink before the patients were admitted to the ICU (Figure 2), so it was concluded that these strains had been transmitted from sink to patient.

Clinical course

Seventeen of the 18 positive patients were colonized with ESBL without causing an infection.

One patient (patient 17) became infected. This patient was admitted to the ICU for 10 days and screening cultures remained negative for ESBL; the patient's clinical condition worsened again and 10 days after discharge from the ICU the patient developed pneumosepsis, for which readmission to an ICU in another hospital was necessary. Antibiotic therapy with ceftriaxone was started and one day later, due to further deterioration, this was replaced with moxifloxacin. On the third day, the patient died. An ESBL-positive *E. cloacae* (AFLP type F) was isolated from sputum and urine. Most probably, ESBL colonization had occurred at the end of the first ICU admission period and remained unnoticed at that time. Three other patients (nos. 6, 14, and 18) who became colonized with *E. cloacae* of the same genotype (AFLP-type F) remained uninfected.

Discussion

This study showed that ESBLs were present in every sink in the ICU during a 20-week screening period, and therefore sinks may act as a source of infection. In four ICU patients it was incontrovertibly shown that these bacteria had been transmitted from sink to patient, as sinks had been colonized with identical ESBLs before the patient was admitted to the ICU room. One of these patients died from an infection with an ESBL-positive *E. cloacae* originating from the sink. These transmissions were stopped effectively by integrating self-disinfecting siphons in the 18 sinks on the ICU; during the study period no other infection prevention measures were implemented. In a follow-up period of eight months, no ESBLs were found in ICU sinks.

During 2011–2012, another 14 patients were colonized with ESBLs during their admission to the ICU. Although no new ESBL

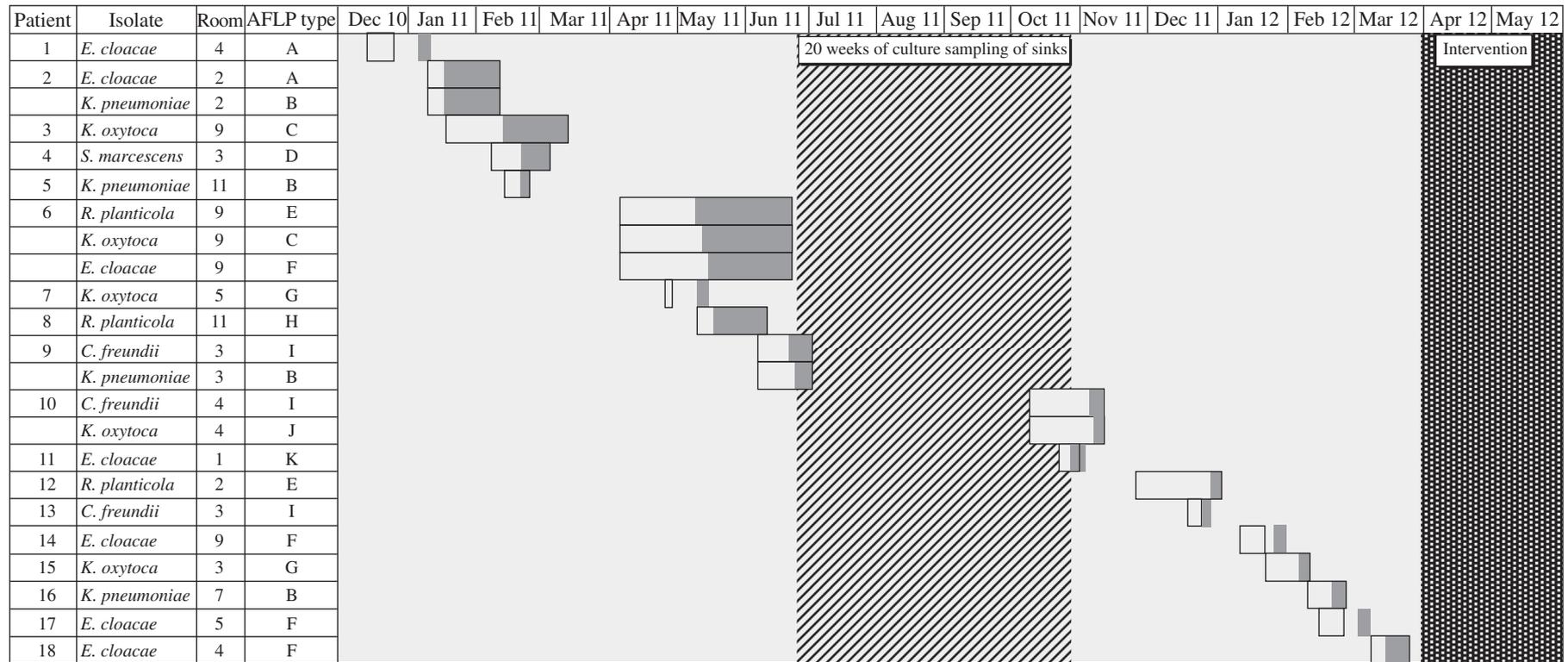


Figure 2. Chronological overview of extended-spectrum β -lactamase (ESBL) colonizations of patients in Deventer Hospital intensive care unit (ICU). Eighteen patients became colonized with 23 different strains. From June until October 2011 patient room sinks were screened on a weekly basis. At the end of March 2012, self-disinfecting siphons were integrated in all sinks, after which no new ESBL-positive patients were observed. Horizontal bars: duration of admission to the ICU; within each bar the darker grey tint indicates the period from the first ESBL-positive culture. In some instances (patients 1, 7, 13, and 17) ESBL positivity was determined from clinical specimens after discharge from the ICU. AFLP, amplified fragment length polymorphism.

colonizations were found after the intervention, it is very likely that some of these patients had also been colonized by strains originating from the sinks. Bacteria may be transmitted from a sink to the patient in various ways. When the tap water is running, water drops may splash out of the sink on to nearby objects that are used for/by the patient (e.g. toothbrush, shaver, medicines, etc.). Furthermore, nursing staff may carry ESBLs when water from the sink or its vicinity touches their hands.^{1,2}

In retrospect, colonization of ICU patients with various ESBLs had occurred for a number of years. There are several reasons why a common source for these colonizations had gone unnoticed. First, a great diversity of ESBLs (different species; different resistance patterns within species) had been isolated in the ICU. Second, new colonizations occurred randomly and without apparent epidemic peaks. Third, in our hospital genotyping of ESBLs is only performed in epidemic situations and with strains with an identical antibiogram. Genetic typing enabled us to demonstrate transmission of bacteria from sinks to patients.

In several studies, attempts have been made to stop transmission of bacteria from sink to patient without long-lasting success. Because bacteria in a biofilm have a reduced sensitivity to chlorine and other disinfectants, flushing sinks with a chlorine solution cannot be completely effective.^{9,10} The effect is only temporary, because remaining viable bacteria can multiply again to the original number of colony-forming units. Substitution of sinks and siphons by new ones has been described as a possible intervention.⁴ This too is not a permanent solution, because washing hands and discarding of wash water will bring new (ESBL-positive) bacteria into the siphon and a new biofilm will be formed.

In the ICU of our hospital, the introduction of 18 self-disinfecting siphons was shown to be a successful intervention. These self-disinfecting siphons prevent the formation of a biofilm in the sink by means of permanent physical disinfection (heating and ultrasound), electromagnetic cleansing, and antibacterial coating. It is very likely that this cost-effective intervention also results in a reduction of transmission of other, especially Gram-negative, bacteria from sink to patient.

Conflict of interest statement

None declared.

Funding sources

None.

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