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Containing a PER-1-producing *Pseudomonas aeruginosa* outbreak linked to sink contamination in an intensive care unit

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SUMMARY

Background: Healthcare-associated infections in intensive care units (ICUs) are associated with reservoirs such as sinks and drainage systems that sustain biofilms and disseminate Gram-negative bacteria. Between July 2021 and November 2022, an increase in PER-1-producing *Pseudomonas aeruginosa* (PaePER) was detected in the ICU of a university hospital in Uruguay.
Aim: The objective was to describe the clinical epidemiology, confirm the environmental source and assess the impact of control measures.
Methods: An outbreak investigation was conducted. Cases were ICU patients with at least one PaePER-positive sample; all other ICU patients were considered exposed. Data collection included clinical and laboratory surveillance, observation of healthcare processes and targeted environmental sampling of seven sinks. Isolates were identified by Matrix-Assisted Laser Desorption/Ionization Time-of-Flight Mass Spectrometry (MALDI-TOF MS); antimicrobial susceptibility was assessed using VITEK2 (bioMérieux), E-test and disk diffusion; extended-spectrum β-lactamase production was evaluated by double-disk synergy and bla_{PER-1} was confirmed by polymerase chain reaction. Clonal relatedness was assessed by pulsed-field gel electrophoresis (PFGE). Control measures included discontinuation of patient room sink use, relocation of medication preparation, renewal of drainage systems and scheduled decontamination with a solution of 15% acetic acid.

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Findings: Among 1221 exposed patients, 31 were affected (attack rate: 2.8%): 25 infections and six colonisation episodes. Eighty-three clinical isolates were recovered, mainly from respiratory and blood samples. Isolates showed high resistance to ceftazidime, cefepime, ceftazidime/avibactam, ceftolozane/tazobactam and amikacin, with preserved susceptibility to carbapenems and cefiderocol. PaePER was recovered from four of seven sinks. PFGE confirmed a single ST309 clone.
Conclusion: Sinks and drainage systems acted as the source of a PaePER outbreak. Targeted interventions rapidly interrupted transmission.

Introduction

Healthcare-associated infections (HAIs) are a major cause of morbidity, mortality and increased healthcare costs [1,2]. Their impact is greater when they occur as outbreaks. Intensive care units (ICUs) are particularly affected because of patient vulnerability and dependence, frequent invasive procedures, high antimicrobial exposure and the influence of environmental contamination [3].

Over the past decade, the role of the hospital environment has regained prominence as a source of HAIs and outbreaks, especially in ICUs [3]. Sanitary facilities such as sinks (designed to improve safety) may create conditions that favour transmission. Numerous experimental and observational studies have shown that sinks can act as reservoirs [4—6]. Micro-organisms, particularly those capable of readily forming biofilms and surviving in water environments, such as *Pseudomonas aeruginosa*, generated by hand hygiene (and other body fluids) colonise drains, form biofilms and migrate upwards to the strainer and bowl [7]. Water striking the strainer generates splashes that contaminate health personnel's hands and nearby surfaces, including medication preparation areas and intravenous devices [7—9]. The importance of this role has been taken into consideration in numerous guidelines with different recommendations [4,10,11].

Risk factors for sink-related infections and outbreaks include shallow bowls, water falling directly onto the strainer, high flow, short distance to patient beds, lack of physical barriers between sinks and patients and use of sinks for disposal of patient waste, antibiotics and nutritional solutions [12—14]. When these factors accumulate and colonised sinks are numerous, the risk of infection and outbreaks increases [12]. Several control strategies have been proposed, including improved cleaning and waste disposal, relocating medication preparation (away from sinks), redesign or removal of sinks (including replacing P-traps) and chemical or physical decontamination of plumbing [5,13,15,16]. These measures are often effective but usually temporary as recontamination is common [6,13]. More radical approaches, such as water-free ICUs, have also been implemented [17].

Hospital environmental colonisation has emerged as a key survival strategy for micro-organisms. Although *P. aeruginosa* is considered an opportunistic pathogen, it is frequently associated with HAIs and outbreaks in ICUs and is among the leading causes of mortality related to antimicrobial resistance [18]. It persistently colonises moist environments, forms robust biofilms and exhibits multiple intrinsic and acquired resistance mechanisms, often resulting in multi-drug-resistant (MDR) or difficult-to-treat-resistant *P*seudomonas *aeruginosa* (DTR-PA) [19]. These traits are characteristic of high-risk clones (e.g. ST235, ST111 and ST233), and ST309 has recently emerged as one such clone [19]. Accordingly, water-related ST309 *P. aeruginosa* outbreaks are increasingly reported [5,6]. In *P. aeruginosa*, high-level resistance to broad-spectrum cephalosporins is often associated with the production of extended-spectrum β-lactamases (ESBLs), predominantly of the GES type, but also PER, VEB and SHV [20]. Their detection is important to differentiate them from non-transferable resistance mechanisms as this distinction has relevant implications for infection control and prevention of dissemination [21].

From July 2021 to November 2022, an outbreak of PER-1-producing *Pseudomonas aeruginosa* (PaePER), belonging to ST309, occurred in the ICU of a university hospital in Uruguay. The microbiological and molecular characteristics of this outbreak have been previously reported [22]. In this study, we describe the clinical and epidemiological features of the outbreak, investigate its environmental source and summarise the control measures implemented.

Methods

Design and definitions

We conducted an outbreak investigation within the framework of the hospital's Infection Prevention and Control Programme (IPCP). A case was defined as any patient in the ICU of the university hospital 'Hospital de Clínicas Dr. Manuel Quintela' (HC) with at least one microbiological sample positive for PaePER. Exposed patients were all others admitted to the same ICU during the study period (July 2021—November 2022). A mixed methodology was used, combining microbiological investigations in clinical and environmental samples, data analysis, staff interviews, review of healthcare processes and direct observation of infrastructure.

Setting and baseline infection control measures

HC is a 320-bed tertiary university hospital. The adult medical-surgical ICU of HC (HC-ICU) involves 23 beds in five areas. Twenty beds are in single rooms, each with a small sink and an adjacent medication preparation surface on the same countertop (approximately 1 m in length); three beds are in an open area with a shared sink. In recent years, the incidence of multi-drug-resistant Gram-negative rods (MDR-GNRs) has increased markedly [23,24], and many *P. aeruginosa* isolates now meet criteria for DTR-PA. Metallo-β-lactamase-producing *P. aeruginosa* (e.g.,

VIM-2) and strains coproducing VIM-2 and PER-1 had been detected previously [21,25], but *P. aeruginosa* producing only PER-1 had not been reported before this outbreak [22].

As part of routine policy, the ICU performed weekly screening for intestinal carriage of MDR-GNRs (carbapenemase-producing *Enterobacterales* or non-fermenteing GNR and colistin-resistant *Acinetobacter baumannii*) and targeted admission screening for patients transferred from other facilities. Screening included species identification and phenotypic carbapenemase detection but not ESBL screening; therefore, PaePER was not detected through routine colonisation surveillance. However, ESBL production is routinely investigated in all *P. aeruginosa* isolated from clinical samples using the double-disk synergy test with ceftazidime, amoxicillin—clavulanate and cefepime. No specific measures targeting water systems were being implemented before the outbreak.

Outbreak investigation

Data collection

Patient data were obtained anonymously from electronic medical records, IPCP outbreak reports and the microbiology laboratory database. Variables included sex, age, ICU length of stay, status at hospital discharge (dead in ICU, dead after ICU discharge and alive), patients categorised as infected or colonised, days from ICU admission to the first PaePER isolation, number and type of samples and susceptibility patterns. Structural features of patient rooms and sinks were documented. Healthcare processes, especially medication preparation and handling of intravenous devices, were observed and explored in staff interviews.

Environmental sampling

Targeted environmental sampling was performed in seven ICU sinks (where more cases were detected) and the associated nearby clean utility areas: four from rooms of patients with PaePER isolates and three in clean utility areas. Using sterile swabs premoistened with Luria—Bertani (LB) broth (Oxoid), four sites per sink were sampled: faucet interior, junction between basin and strainer, basin surface and rubber gasket near the P-trap (Figure 1).

Microbiological methods and molecular typing

Clinical samples were processed in the hospital microbiology laboratory according to standard protocols and interpreted as infection or colonisation by the ICU medical team. Environmental samples were processed in a research laboratory. Swabs were incubated in LB broth (18—24 h, 37 °C) and plated on MacConkey agar with ceftazidime and amikacin and on MacConkey with ceftazidime alone.

Species identification was performed by MALDI-TOF MS and antimicrobial susceptibility testing by VITEK2®. ESBL production was assessed by double-disk synergy test using ceftazidime, amoxicillin—clavulanate and cefepime. For the first clinical isolate from the first 25 patients, additional testing included disk diffusion for cefiderocol (FDC) and E-test for ceftolozane/tazobactam (C/T) and ceftazidime/avibactam (CZA). Results were interpreted according to European Committee on Antimicrobial Susceptibility Testing (EUCAST) 2022 criteria. In this subgroup, the presence of $bla_{\text{PER-1}}$ was confirmed by polymerase chain reaction. Clonal relatedness among clinical and environmental isolates was evaluated by pulsed-field gel electrophoresis (PFGE) after *SpeI* digestion [22]. PFGE analyses were performed in multiple runs, and gel images were acquired independently. Banding patterns were normalised using BioNumerics software (Applied Maths, bioMérieux) and subsequently interpreted according to Tenover criteria, with profiles showing differences $\leq$3 bands considered indistinguishable or closely related [26].

Control measures

Control measures were designed based on the hypothesis of sink contamination as the main source and on observed care activities that could facilitate transmission (Figure 2). Standard infection prevention practices (hand hygiene, glove use, intravenous device handling, environmental cleaning and disinfection) were reinforced.

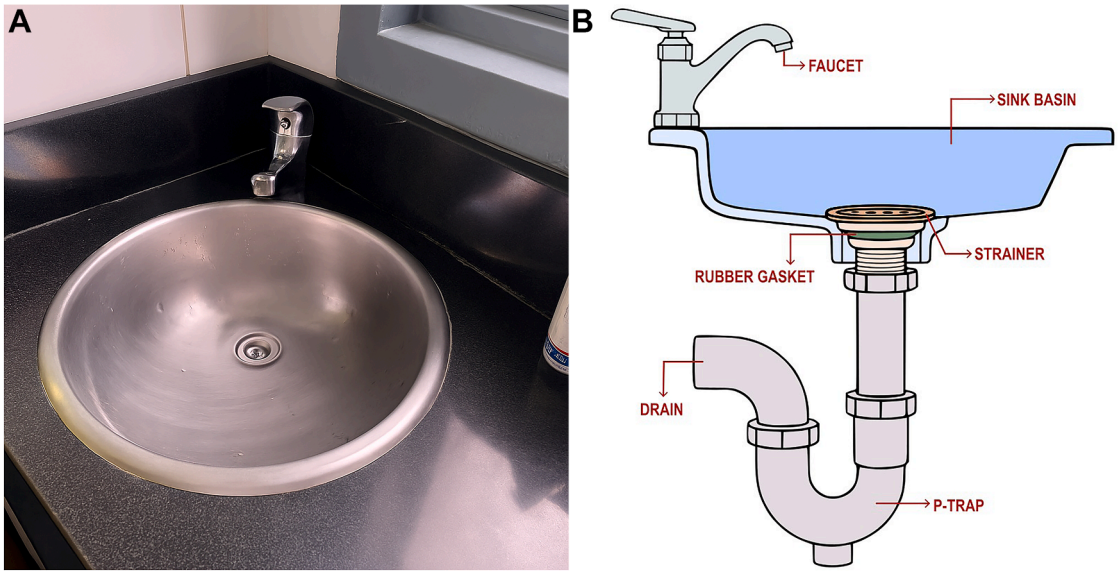

Figure 1. A. Standard sink in the ICU patient's room. B. Scheme of a sink showing environmental sample sites. ICU, intensive care unit.

a. Discontinuation of patient room sink use, promoting hand hygiene using alcohol-based hand rub.
b. Installation of new handwashing sinks located away from patient areas and critical zones.
c. Relocation of medication preparation to clean utility rooms, outside patient rooms and away from sinks.
d. Replacement of drainage systems in clean utility rooms where hand hygiene and medication preparation are performed.
e. Scheduled cleaning of sinks and drainage systems using a commercial solution (70% ethanol) with 15% acetic acid was performed twice weekly. This involved flooding the sinks and drainage system for 10 minutes using a distal drain shut-off valve, followed by thorough rinsing with running water. Cleaning was performed in the absence of personnel, with ventilation using an air extractor and with personal protective equipment (mask, eye protection, and gloves).

Figure 2. Infection control measures implemented for outbreak containment.

Outcome

The primary outcome was the detection of new cases with PaePER isolates. The postintervention follow-up period was six months.

Statistical analysis

Categorical variables were expressed as absolute and relative frequencies and quantitative variables as median and interquartile range (IQR). Incidence was expressed in number of cases in absolute frequency and in incidence density expressed as cases per 1000 patient-days. Lethality (the number of deceased patients from the outbreak/total patients in the outbreak) and attack (total patients in the outbreak/total exposed patients in the period of the outbreak) rates were calculated. Data were analysed using SPSS Statistics v25 (IBM).

Results

Outbreak description

From July 2021 to November 2022, 31 patients in the HC-ICU were colonised or infected with PaePER among 1221 exposed (attack rate: 2.8%) (Figure 3). The incidence density in the period (the number of cases/summatory 1000 patient-days of the period) was 3.32 episodes per 1000 patient-days (31/9332*1000).

Among the 31 patients included, 20 were male and median age was 53 years (IQR: 15). Twenty-five patients were considered infected and treated accordingly; six were classified as colonised. Median ICU stay was 42 days (IQR: 41.5), and median time from ICU admission to the first PaePER isolation was 17 days (IQR: 13.5). Thirteen patients died in the ICU, three after ICU discharge and 15 were discharged alive, corresponding to a lethality of 51.6% (16/31).

Microbiological findings

Eighty-three PaePER isolates were obtained from the 31 patients: 56 from respiratory samples, 14 from blood, four from catheter tips, four from urine, two from soft tissue, two from cerebrospinal fluid and one from a surgical wound. Isolates showed high levels of resistance to amikacin (77/83; 92,8%), cefepime (82/83; 98,8%), ceftazidime (83/83, 100%) and piperacillin/tazobactam (77/83; 92,8%) and remained mostly susceptible to meropenem (76/83; 91,6%), imipenem (70/83; 84,3%) and ciprofloxacin (62/83; 74,7%). In the subgroup of the first clinical isolates tested by extended methods (25 patients), all were susceptible to FDC and resistant to C/T; 5 of 25 were

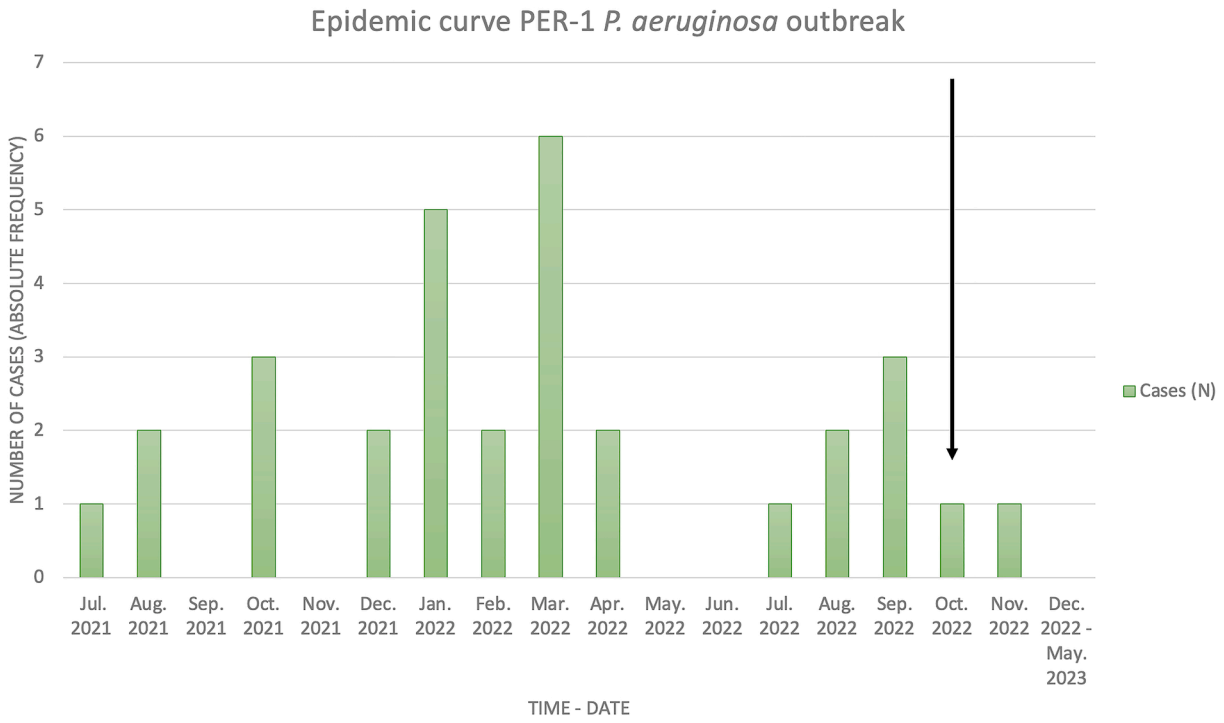

Figure 3. Epidemic curve. Dates (in months) correspond to the first isolate of each patient. The number of cases is expressed in absolute frequency. Black arrow: the moment of initiation of control measures (14th October 2022).

resistant to CZA. All clinical isolates were ESBL positive by double-disk synergy, and bla_{PER-1} was confirmed in the tested subgroup.

A subset of five isolates previously analysed by whole-genome sequencing (WGS) in a previous work from our group revealed the presence of several antibiotic resistance genes, including bla_{PER-1} (associated with high-level resistance to broad-spectrum cephalosporins) and *aac*(6′)-II (associated with amikacin resistance), among others. Additionally, biofilm formation assays of selected isolates revealed that they were strong biofilm producers [22].

Environmental investigation

From 28 sink samples, 60 isolates of MDR-GNR were recovered; nine corresponded to PaePER. Four of seven sinks yielded PaePER: two in patient rooms, one in a clean utility room and one in a common area. Six positive samples came from the rubber gasket near the P-trap, two from the strainer body and one from the junction between basin and strainer; samples that came from faucets were negative. Environmental PaePER isolates had susceptibility profiles similar to the clinical isolates, with high minimum inhibitory concentrations of ceftazidime, cefepime and amikacin and retained susceptibility to meropenem, imipenem and ciprofloxacin (except one ciprofloxacin-resistant isolate). All were ESBL positive and carried bla_{PER-1}.

PFGE analysis

PFGE comparison of 25 clinical and nine environmental isolates revealed closely related subclusters of isolates displaying indistinguishable or closely related banding patterns ($\leq$3 band differences), consistent with a clonal outbreak. Band variations likely reflect micro-evolution over time and may be explained by point mutations, insertions or deletions or recombination events occurring during clonal persistence. Notably, isolates recovered from sinks displayed banding patterns indistinguishable from or closely related to those of clinical isolates, suggesting a shared clonal origin (Figure 4).

Previously, the subset of five clinical isolates analysed by WGS showed that all belonged to ST309, consistent with the clonal relatedness observed by PFGE [22].

Risk factors, observed practices and targeted interventions

Several risk factors for sink-related transmission were identified in the ICU: shallow sinks (12 cm) with water falling directly onto the strainer, use of patient room sinks for disposal of hygiene water and other fluids, contamination with multiple MDR-GNRs and the absence of programmed disinfection, visible splashes reaching the working surface on the same countertop and lack of physical barriers and a distance $<$2 m between sinks and beds. Staff interviews and observation revealed that medication preparation and handling of intravenous devices were performed on surfaces adjacent to patient room sinks and that patient fluids and solutions used for care and medication preparation were routinely discarded in these sinks.

These findings guided the targeted interventions: relocation of medication preparation and device handling to clean utility rooms away from contaminating splashes from sinks, elimination of patient room sinks, prohibition of fluid disposal in room sinks, renewal of drainage systems in clean areas and incorporation of these sinks into the acetic acid cleaning schedule.

Outcome: impact of control measures

After implementation of control measures in October 2022, only one additional PaePER isolate was detected in November 2022. No further clinical isolates were identified during the subsequent six months, and the outbreak was considered terminated.

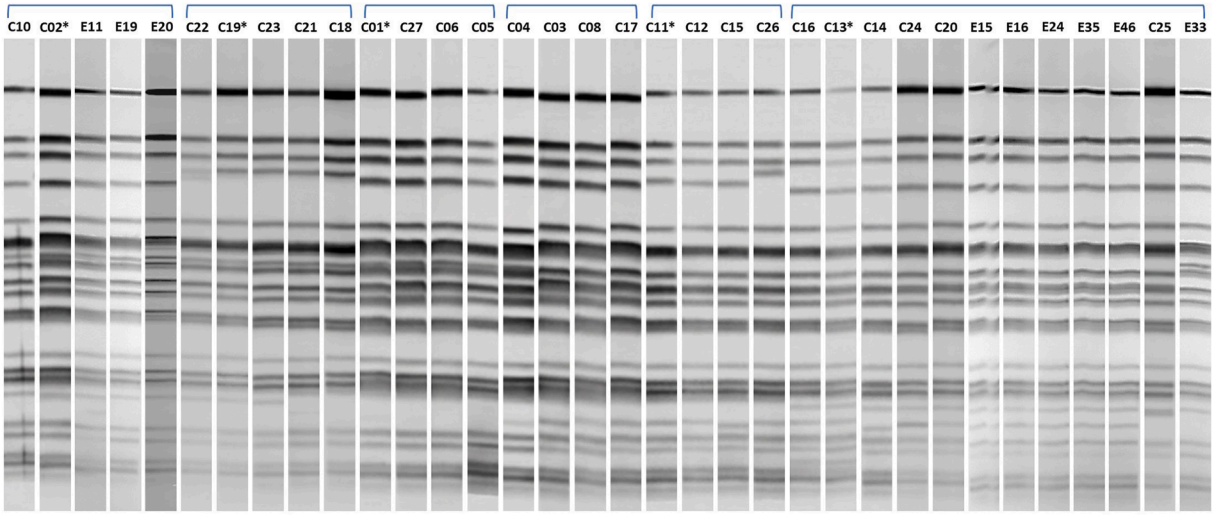

Figure 4. PFGE banding patterns of clinical and environmental *Pseudomonas aeruginosa* isolates. Isolate identifiers are indicated above each lane, with 'C' and 'E' denoting clinical and environmental (sink) isolates, respectively. Brackets group closely related profiles, and asterisks mark isolates subjected to whole-genome sequencing. PFGE, pulsed-field gel electrophoresis.

Discussion

We describe a low-incidence outbreak of PER-1-producing *P. aeruginosa* in an ICU, with cases scattered over two years and months without new detections. The unusual ESBL mechanism, which had not previously been seen alone in our ICU, together with continuous assessment of ESBL production in every *P. aeruginosa* isolate in the clinical laboratory, enabled recognition of the outbreak, despite its low density.

The epidemiological, microbiological and environmental findings support sinks and their drainage systems as the main reservoir and source of transmission as it has been well described previously [6,27,28]. PaePER was repeatedly isolated from sink components, particularly rubber gaskets, and clinical and environmental isolates were closely related by PFGE and shared the same resistance profile. The clone belonged to ST309, an emerging high-risk lineage. As reported previously by our group, the isolates studied presented strong biofilm-forming capacity, likely favouring persistence in wet environments and facilitating ongoing transmission [22]. PER-1 is an uncommon ESBL with a distinctive susceptibility profile, notably conferring resistance to CZA and C/T, as seen in our isolates. These agents are not widely available in our setting, while the frequent use of ceftazidime in the ICU may contribute to selective pressure favouring the emergence of this resistance mechanism [20,29].

The outbreak occurred in the context of the COVID-19 pandemic when, in 2021, the ICU expanded from 11 to 23 beds and new staff members were incorporated. Additionally, as reported previously, during this period, surveillance activities focussed on SARS-CoV-2 and carbapenemase-producing organisms, which may have delayed recognition of the event [30].

The control strategy targeted both the environmental source and care processes associated with it. Eliminating patient room sinks, relocating medication preparation, renewing drainage systems and instituting regular acetic acid treatment of sinks and drains (together with reinforcement of standard infection prevention measures) rapidly interrupted transmission, consistent with what is well established for effective outbreak control [3,5,13,15,16]. Such horizontal measures may also reduce the burden of other MDR organisms present in the same reservoirs, including carbapenemase-producing *Enterobacterales*, and are relatively low cost compared with the consequences of ICU outbreaks [16,31].

This investigation has several limitations. Clinical records could not be fully reviewed because data were anonymised, preventing assessment of individual risk factors and attribution of mortality to PaePER infection. Environmental sampling did not include all sinks in the unit, although control measures were applied in the whole ICU. Sinks were not resampled after the outbreak ended; this decision reflected the known tendency of sinks to become recontaminated [6,13] and the recognition that the main objectives are interruption of transmission and reduction of bacterial load rather than complete eradication of micro-organisms from environmental reservoirs.

In conclusion, this outbreak underscores the importance of considering environmental reservoirs, particularly sinks and their drainage systems, as potential sources of ICU outbreaks. Detailed analysis of the micro-organism involved, infection patterns and unit-level structural and procedural risk factors was essential to identify the source and design effective interventions. Active assessment of resistance mechanisms in the laboratory enabled detection of this low-incidence event. Integrating routine structural review and cleaning of sinks and drains, together with ongoing laboratory-based surveillance, into standard infection prevention practice may help prevent recurrence of similar outbreaks and reduce the burden of MDR organisms in ICUs.

CRediT authorship contribution statement

L. Araújo Pírez: Writing — original draft, Visualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **R. Papa-Ezdra:** Writing — original draft, Project administration, Methodology, Investigation, Formal analysis, Conceptualization. **M. Outeda:** Writing — original draft, Visualization, Methodology, Investigation, Data curation. **R. Guzmán:** Writing — original draft, Validation, Software, Methodology, Formal analysis, Data curation. **N. Hernández:** Methodology, Investigation. **G. Méndez:** Methodology, Investigation. **P. Gadea:** Validation, Methodology, Investigation. **M. Moreira:** Methodology, Investigation. **A. Inchausti:** Investigation. **G. Burghi:** Validation, Investigation. **V. Seija:** Visualization, Validation, Investigation. **R. Vignoli:** Writing — review & editing, Visualization, Validation, Supervision, Methodology, Formal analysis, Conceptualization. **H. Albornoz Da Silva:** Writing — review & editing, Validation, Supervision, Methodology, Investigation, Formal analysis, Conceptualization.

Ethics statement

The study was approved by the Hospital de Clínicas Ethics Committee (resolution 104-24). Data were provided to the investigators in an anonymized form and were handled and reported in accordance with institutional requirements.

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Conflict of interest statement

We declare no conflict of interest regarding this investigation.

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