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Outbreak

An outbreak of *Serratia liquefaciens* at a rural health center in The Gambia

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Abstract

Introduction: Healthcare-associated infections (HAIs) are better documented in developed than in developing countries. There are emerging reports regarding the high frequency of HAIs in developing countries. We aimed to report an outbreak of an HAI caused by *Serratia liquefaciens* at a rural health center in The Gambia.

Methodology: Following an abrupt increase in the isolation of *S. liquefaciens* in clinical samples, laboratory and clinical consumables, as well as staff, were screened for contamination with *S. liquefaciens.* Conventional microbiological techniques and biochemical identification tests were used. A phenotypic typing was achieved using the Kirby-Bauer antibiotic susceptibility method. Strategies to control the outbreak were implemented.

Results: A total of 794 samples were processed during the outbreak; 44 (6%) grew *S. liquefaciens*. Five (25%) of the 20 suspected contaminated materials (hospital consumables and equipment) screened yielded growth of the organism. The primary source of the outbreak was hospital consumables. Three (7%) of the 44 infected children died with no other known cause than *S. liquefaciens* infection. Ninety-nine percent similarity of the antibiogram phenotypic typing suggests the isolates were from the same clonal origin. The outbreak was successfully controlled after the removal and sterilization of the respective contaminated fluids and equipment.

Conclusions: This HAI was caused by poor practice in the preparation of medications for nebulization and intravenous infusion, hygiene practices, and a lack of awareness among staff about infection control. We recommend further studies to delineate the role played by HAIs in the developing world.

Key words: Healthcare-associated infection; outbreak; *Serratia liquefaciens*; antibiotic; Gambia.

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Introduction

Healthcare-associated infections (HAIs) are considered the commonest adverse conditions threatening patients' safety worldwide [1]. The prevalence of HAIs is higher in developing countries, particularly in Africa, compared to the developed world [2-3]. HAIs cause approximately 100,000 deaths per year in developed countries such as the United States [4]. Studies have shown that contamination of health workers' protective equipment, such as gowns and gloves, with healthcare-associated pathogens is common [4-5].

The genus *Serratia* is one pathogen known to cause HAIs. *Serratia* belongs to the family Enterobacteriaceae; over time, the taxonomy of *Serratia* has evolved, with 14 currently described species belonging to the genus [6]. *Serratia* species are usually recognized as nosocomial pathogens; however, some recent studies have demonstrated that *Serratia* infection can be community acquired, with *Serratia marcescens* being implicated [7-8]. Infections with *S. liquefaciens* in healthcare setting are uncommon compared to infections with *S. marcescens*. *S. liquefaciens* colonizes hands and the respiratory, gastrointestinal, and urinary tracts [9-10]. It is a highly motile organism with swarming capacity and is commonly found in water, soil, plants, and insects [11]. The pathogenicity of *S. liquefaciens* is well established in humans, insects, and fish [11-12], and cases of fatal infection have been reported [13]. Many investigations have documented outbreaks of *S. liquefaciens* in settings of medical care, such as hemodialysis centers and neurosurgery departments [14-15]. *S. liquefaciens* often colonizes hospital instruments and consumable materials that include pressure monitoring equipment, suction pumps, oxygen concentrators, mechanical ventilators, intravenous infusion sets, intravascular catheters, hand-soap dispensers, and vials of liquid medication [16-17]. Transfusion reactions associated with contaminated blood products have also been documented [18-19]. In 2008, the European Centre for Disease Prevention and Control reported that *Serratia* species represented 2.0% of all bloodstream infections, ranking organisms from this genus as the tenth most

commonly recovered organisms from intensive care unit (ICU)-acquired bloodstream infections [20]. A case of sepsis caused by *S. liquefaciens* following a vitamin C infusion has also been reported [21]. Many reports have highlighted *S. liquefaciens* in bloodstream infections, fistulous pyoderma, and infective endocarditis [22-24]. *Serratia* species have been isolated from specimens such as blood, urine, cerebrospinal fluid, sputum, induced sputum, lung aspirate, and wound swabs [11]. High levels of antimicrobial resistance among isolates of *S. liquefaciens* complicate treatment [13].

The aim of this report was to describe an investigation and intervention conducted to terminate an outbreak of HAI caused by *S. liquefaciens* in a rural health center in The Gambia, West Africa. Thus, we highlight the potential for such outbreaks in developing countries and the interventions that can be used to address such events.

Methodology

Study area and patient population

This study was conducted at the Basse Health Centre (BHC) in Upper River Region (URR) of eastern Gambia. The health center serves a population of over 200,000, among whom those under five years of age account for 20% of the population. A population-based surveillance study of the burden of pneumococcal disease and a study of pneumonia etiology started in May 2008 and August 2009, respectively. The etiology study ended in March 2011, while the surveillance study is ongoing. The Medical Research Council (MRC) laboratory at the MRC Basse field station processes samples to detect pathogens for many ongoing studies that included the etiology and surveillance studies. Over 20,000 samples were processed at MRC laboratory in Basse from these two studies prior to the onset of the outbreak. The first isolate in the outbreak was identified on 30 June 2010. One isolate of *S. marscens*, and no isolates of *S. liquefaciens* were detected prior to June 2010.

Description of the outbreak

In mid-August 2010, staff at the Basse MRC laboratory became concerned about the frequent isolation of *S. liquefaciens* from clinical samples, particularly invasive samples such as blood. The first, second, and third *S. liquefaciens* isolates from blood were isolated 30 June, 25 July, and 1 August. Three isolates from nasopharyngeal aspirates (NPAs) were isolated on August 18 (Table 1). When the eighth isolate of *S. liquefaciens* was isolated, the laboratory conducted a thorough investigation to determine if there was a source of contamination in the laboratory. All laboratory equipment (incubators, suction tube, BACTEC 9050, class II safety cabinet, centrifuges, and fridges) and consumable reagents such as distilled water, normal saline, and prepared bacterial culture media were inoculated for microbiology bacterial growth using conventional bacterial culture on fluid and solid media. The outcome of the investigation of the laboratory sources revealed an absence of *S. liquefaciens*. When the twelfth isolate was detected, on 22 August 2010, the investigation was expanded to the clinical setting at the health center.

General investigation to establish corrective and preventive actions

Following the notification of relevant stakeholders, a committee including clinicians, microbiologist, and a nurse was formed to address the problem. The major task was to investigate and identify the source of the outbreak. First, the procedure room where intravenous infusions were prepared and administered was inspected. Also, the administration of infusion fluid, drug suspension vials, and nebulized salbutamol fluid were observed. Additionally, the committee monitored procedures for the collection of venous blood and the inoculation of blood culture bottles and sample processing in the laboratory.

All infusion fluid, liquid medication, sterilizing agents, LabGuard-hand soap, and equipment used for patient care was swabbed and cultured for bacterial

Table 1. Detection pattern for the first to sixteenth isolates of *Serratia liquefaciens*.

Case	Date	No. of Sample	Type of sample	Diagnosis
1	June 30, 2010	1	Blood	Pneumonia
2	July 25, 2010	1	Blood	Pneumonia
3	August 1, 2010	1	Blood	Pneumonia
4–6	August 18, 2010	3	NPA	Pneumonia
7–8	August 19, 2010	2	NPA-1, IS-1	Pneumonia
9–11	August 20, 2010	3	NPA-2, IS-1	Pneumonia
12	August 22, 2010	1	Blood	Pneumonia
13–16	August 23, 2010	4	NPA-3, IS-1	Pneumonia

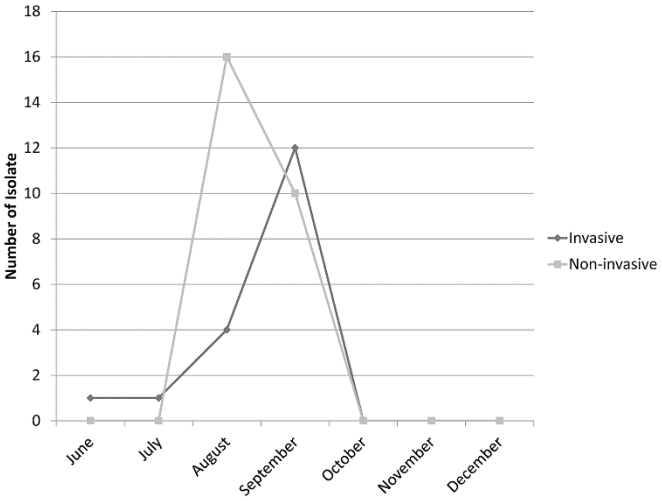
LA: lung aspirate; NPA: nasopharyngeal aspirate; IS: induced sputum.

isolation and identification. In addition, the hands of staff (nurses/phlebotomist, field and laboratory personnel) attending to patients and handling and processing samples were swabbed and cultured. Antimicrobial susceptibility tests were conducted on *S. liquefaciens* isolates from contaminated items, and the results were compared with antimicrobial susceptibility results obtained from isolates from the original clinical samples for phenotypic comparison.

Bacteriology

Isolation of *S. liquefaciens* from clinical samples that included blood and lung aspirate samples was achieved by incubating inoculated blood culture bottles (BD BACTEC Peds Plus – Maryland, USA) in an automated blood culture machine (BACTEC 9050 – Maryland, USA) followed by subculture on solid media (blood, chocolate, and MacConkey agars). Nasoparngeal aspirate and induced sputum samples were also cultured on the three solid agar media and incubated overnight at 37°C. Identification of the organism was achieved using standard biochemical techniques including analytical profile index 20 non-enteric and 20 enteric (API 20NE and API 20E; BioMerieux – Macy-I'Etoile, France). Antimicrobial susceptibility testing was performed on all the isolates using the Kirby-Bauer disk diffusion method, following the 2010 guidelines of the Clinical and Laboratory Standards Institute (CLSI). The control organism used was *Escherichia coli* ATCC 25922.

Figure 1. Distribution of *S. liquefaciens* during and post-outbreak.



Results

S. liquefaciens was isolated from 44 children during the 10 weeks (30 June–16 September 2010) of the outbreak (Figure 1). The affected children were all seen at the outpatient department. Of the 794 clinical

Table 2. Screened hospital consumables and equipment from health center outpatient department and Medical Research Council laboratory for bacterial growth.

S/No.	Consumable and equipment	Result
	Health Center Outpatient Department	
1	Normal saline (infusion, 0.85% – in use)	*S. liquefaciens* (> 25 × 10^5 organisms/mL)
2	Normal saline in syringe	*S. liquefaciens* (> 25 × 10^5 organisms/mL)
3	Ventolin fluid for nebulization	*S. liquefaciens* (> 25 × 10^5 organisms/mL)
4	Methylated spirit (in use)	*S. liquefaciens* (> 25 × 10^5 organisms/mL)
5	Oxygen concentrator	*S. liquefaciens* (> 25 × 10^5 organisms/mL)
6	Normal saline (infusion, 0.85% – unused stock)	No bacterial growth
7	Methylated spirit (unused stock)	No bacterial growth
8	Totacide disinfectant	No bacterial growth
9	LabGuard liquid soap	No bacterial growth
10	BACTEC blood culture bottle	No bacterial growth
11	Conventional blood culture bottle	No bacterial growth
12	Glucose (intravenous infusion)	No bacterial growth
13	Benzypenicillin	No bacterial growth
14	Suction tube connected to vacuum	Bacillus species (< 20 × 10^5 organisms/mL)
15	Suction tube connected to mucus extractor	*Bacillus* species, *Viridans streptococcus*, and coagulase-negative staphylococci (< 20×10^5 organisms/mL)
	Basse MRC Laboratory	
16	Sterile distilled water – stock and in use	No bacterial growth
17	Sterile normal saline – stock and in use	No bacterial growth
18	Methylated spirit	No bacterial growth
19	BACTEC culture bottle top	No bacterial growth
20	Conventional blood culture bottle top	Coagulase-negative staphylococci (< 20 x 10^5 organisms/mL)

samples processed for bacterial culture during the outbreak, 44 (6%) yielded *S. liquefaciens*; these samples included blood (14; 32%), lung aspirate (4; 9%), nasopharyngeal aspirate (22; 50%), and induced sputum (4; 9%). Twenty clinical items used in the collection and preparation of clinical samples were screened for bacterial growth (Table 2). Microbiological investigation of clinical materials and equipment revealed that 5/20 (25%) were contaminated with *S. liquefaciens*, and 3/20 (15.3%) were contaminated with other environmental bacteria, such as coagulase-negative *Staphylococcus*, *Streptococcus viridians*, and *Bacillus* species (Table 2). The primary sources of contamination were the saline used for preparation of intravenous medication, salbutamol fluid used to prepare solutions for nebulization, and surgical spirit used for cleansing skin before venepuncture. Repeated use of single bottles and vials for multiple patients was identified as a sub-standard practice that likely contributed to the outbreak. Eight of 15 swabs of the hands of clinical staff at the pediatric outpatient department (OPD) and laboratory staff grew coagulase-negative staphylococci. *S. liquefaciens* was not isolated from the hands of any staff member. Three of the 44 infected patients (7%) died, and *S. liquefaciens* was isolated from the blood of the three patients who died (3/18 [17%]) (Figure 2). Interestingly, the impact of control measures introduced was excellent in that there were no cases of *S. liquefaciens* isolated in the laboratory in October, November, and December 2010 (post-outbreak) and thereafter.

The antimicrobial susceptibility patterns of the isolates obtained from clinical samples, hospital consumables, and equipment were very similar, suggesting they were all related to one original source (Table 3), but high throughput methods such as pulsed-field gel electrophoresis (PFGE) or random amplified polymorphic DNA (RAPD) with high power discriminatory outcome were not available to determine clonal similarity.

Discussion

Healthy individuals rarely become infected by the genus *Serratia*, but hospitalized patients may be colonized or infected by *S. marcescens* [7,25]. Occasionally, *S. liquefaciens* is isolated from clinical specimens but its pathogenic role is often unclear [6]. *S. liquefaciens* causes infections in immunocompromised hosts [13], such as neonates [26], and in those with indwelling/introduced foreign bodies/liquids, *e.g.*, intravenous/intra-arterial (IV/IA) lines, endotracheal tubes, multiple use vials [16]; thus, the entry routes are ingestion, injection, and catheterization. Our investigation of an outbreak of *S. liquefaciens* infection in a rural African health center revealed expected sources and modes of transmission; various solutions used in patient care were contaminated and there was poor practice in the use of these solutions.

Figure 2. Type of samples processed during outbreak.

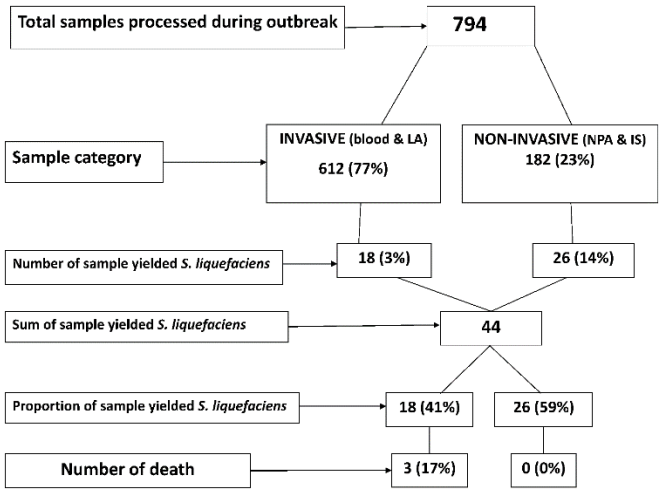


Table 3. Comparison of antibiotic susceptibility profile of the *Serratia liquefaciens* isolates from human (n = 44) and contaminated health clinical materials (n = 5).

Antibiotic (concentration)	Human (invasive and non-invasive) isolates (n = 44)		Consumables and equipment isolates (n = 5)	
	R (%)	S (%)	R (%)	S (%)
Ampicillin (10 µg)	44 (100)	0 (0)	5 (100)	0 (0)
Chloramphenicol (30 µg)	0 (0)	44 (100)	0 (0)	5 (100)
Cotrimoxazole (30 µg)	0 (0)	44 (100)	0 (0)	5 (100)
Tetracycline (30 µg)	5 (11)	39 (89)	0 (0)	5(100)
Gentamicin (30 µg)	0 (0)	44 (100)	0 (0)	5 (100)
Ciprofloxacin (30 µg)	0 (0)	44 (100)	0 (0)	5 (100)
Cefotaxime (30 µg)	0 (0)	44 (100)	0 (0)	5 (100)
Nalidixic acid (30 µg)	0 (0)	44 (100)	0 (0)	5 (100)

R: resistant; S: sensitive.

Review of the three deaths recorded in this study by the attending clinician revealed that these deaths were likely attributed to infection with *S. liquefaciens*. In 2011, a study conducted in Greece made a similar fatal case report [13]. Again, our findings corroborate those of a report of a 10-year population-based study in Canberra, Australia, which showed *Serratia* species bacteremia to be more common than what was known, with a high proportion (47%) of episodes [27]. Antibiogram phenotyping of isolates obtained from clinical samples, hospital consumables, and equipment showed 99% similarity, suggesting isolates were from a common source and probably of the same clonal origin. One of the limitations of this study was the unavailability of genotypic characterizations such as PFGE or RAPD assays, which may be used to confirm clonal similarity of isolates.

In this investigation, the original source of contamination of the pediatric OPD items by *S. liquefaciens* is not clear. Nonetheless, our findings suggest that contamination happened in the pediatric OPD. The question about the source of the contamination has no straightforward answer, but it was certain that the source was neither the commercial 0.85% saline nor the methylated spirit because only the in-use quantities of these agents were contaminated and not the unused stock, with evidenced sterility of the unused stock. Our study also established that the contamination was not from the Basse MRC laboratory. Also, the contamination was not from the hands of the healthcare staff (Table 4), reinforcing the bactericidal effect of hand washing with soap (LabGuard) against microbial agents such as *S. liquefaciens*. The confirmed sources of contamination included the salbutamol fluid, single vials of which were used for nebulization of multiple patients. Oxygen concentrators were also contaminated with *S. liquefaciens*, and these machines were often shared among patients. Normal saline infusion fluid was contaminated, most likely due to multiple puncture of the bottles in order to prepare dosages of medication or volumes for resuscitation. Our hypothesis is that either an asymptomatic carrier or a symptomatic individual contaminated the inhaling fluid and the inhaling devices because there was a phenotypic similarity of the strains isolated from blood, lung aspirate, and induced sputum compared with strains from contaminated items. We also observed poor aseptic technique that most likely contributed to the contamination of the normal saline infusion and methylated spirit, as it was likely that *S. liquefaciens* was inoculated into the infusion bottle due to multiple punctures. Also, an asymptomatic carrier may have generated droplets either by coughing or sneezing during sample collection, thus inoculating uncovered disinfecting agents such as methylated spirit, which later was used to disinfect a patient's skin prior to sample collection but instead caused contamination of the skin. Contamination of methylated spirit is a strong possibility because the first three *S. liquefaciens* were first isolated from blood at on different days (30 June, 25 July, and 1 August 2014), while the first set of isolates from NPAs and induced sputum was detected on 18 and 19 August 2014, respectively. Similar information highlighted on the table that showed demographic detail of the infected individuals (Table 5).

Table 4. Bacterial growth result from screened health workers' hands.

Staff ID Code	Result
A	Coagulase-negative staphylococci (< 20 ×10^5 organisms/mL)
B	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
C	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
D	No bacterial growth
E	No bacterial growth
F	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
G	No bacterial growth
H	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
I	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
J	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
K	No bacterial growth
L	No bacterial growth
M	No bacterial growth
N	Coagulase-negative staphylococci (< 20 × 10^5 organisms/mL)
O	No bacterial growth

Table 5. Demographic detail of patient whose sample yielded *Serratia liquefaciens*.

Patient ID	Date of isolation	Age in months	Sex	Diagnosis	Sample type
1	30-Jun-10	4	F	Pneumonia	Blood
2	25-Jul-10	6	M	Pneumonia	Blood
3	01-Aug-10	9	M	Pneumonia	Blood
4	22-Aug-10	13	M	Pneumonia	Blood
5	26-Aug-10	4 days	F	Pneumonia	Blood
6	31-Aug-10	10	M	Pneumonia	Blood
7	03-Sep-10	6	F	Meningitis	Blood
8	06-Sep-10	9	F	Pneumonia	Blood
9	07-Sep-10	60	M	Pneumonia	LA
1	07-Sep-10	21	F	Pneumonia	LA
11	11-Sep-10	3	F	Septicaemia	Blood
12	13-Sep-10	18	M	Pneumonia	Blood
13	13-Sep-10	5	F	Pneumonia	Blood
14	14-Sep-10	11	F	Pneumonia	Blood
15	15-Sep-10	23	F	Pneumonia	LA
16	15-Sep-10	3 days	F	Septicaemia	Blood
17	15-Sep-10	42	M	Pneumonia	LA
18	15-Sep-10	26	M	Pneumonia	Blood
19	18-Aug-10	3	F	Pneumonia	NPA
20	18-Aug-10	14	M	Pneumonia	NPA
21	18-Aug-10	7	M	Pneumonia	NPA
22	19-Aug-10	24	M	Pneumonia	NPA
23	19-Aug-10	10	M	Pneumonia	IS
24	20-Aug-10	7	F	Pneumonia	NPA
25	20-Aug-10	17	M	Pneumonia	NPA
26	20-Aug-10	9	M	Pneumonia	IS
27	23-Aug-10	58	F	Pneumonia	IS
28	23-Aug-10	9	M	Pneumonia	NPA
29	23-Aug-10	8	M	Pneumonia	NPA
30	23-Aug-10	58	F	Pneumonia	NPA
31	24-Aug-10	33	M	Pneumonia	NPA
32	24-Aug-10	34	M	Pneumonia	NPA
33	25-Aug-10	8	M	Pneumonia	NPA
34	27-Aug-10	6	M	Pneumonia	NPA
35	01-Sep-10	15	M	Pneumonia	NPA
36	14-Sep-10	3	F	Pneumonia	NPA
37	14-Sep-10	17	M	Pneumonia	IS
38	12-Sep-10	12	M	Pneumonia	NPA
39	13-Sep-10	20	M	Pneumonia	NPA
40	14-Sep-10	4	M	Pneumonia	NPA
41	14-Sep-10	6	M	Pneumonia	NPA
42	14-Sep-10	4	M	Pneumonia	NPA
43	15-Sep-10	13	M	Pneumonia	NPA
44	16-Sep-10	6	F	Pneumonia	NPA

LA: lung aspirate; NPA: nasopharyngeal aspirate; IS: induced sputum.

This investigation confirmed the reservoir for *S. liquefaciens* in saline preparations, infusion fluid, liquid medications (particularly multi-use vials/containers), inhalation fluid, and ventilation and mechanical equipment, as shown in table 2. Our findings are similar to those observed in previous studies [16,28].

A number of corrective actions were taken to eradicate the spread of *S. liquefaciens*. We removed and destroyed contaminated saline, nebulizers, suction tubing, methylated spirit bottles, salbutamol solutions, and syringes. We disinfected all equipment, surfaces, oxygen concentrators, and nebulizing devices with 10% sodium hypochlorite. In addition, we autoclaved all heat-proof, reusable medical devices. In order to ensure complete eradication of *S. liquefaciens*, we re-sampled fresh normal saline, sabutamol fluid, sterilized equipment, and disinfected surfaces by swabbing for bacterial culture and isolation; the result obtained yielded no bacteria growth. Finally, we informed all staff about the likely source of the outbreak. Following the corrective actions, no further cases of *S. liquefaciens* have been detected in the MRC Basse laboratory.

We established preventive measures that included introduction of mandatory weekly general cleansing of the clinical area, and strongly encouraged strict hygiene such as washing of hands and arms with liquid soap and the use of cleansers containing 70% alcohol before every procedure in which contamination may occur (*e.g.*, before touching a patient, refilling saline reservoirs). We also introduced a strict policy that one syringe and one needle be used for any one patient receiving IV therapy, enforced the use of one nebulizing mask and chamber and one set of tubing per patient, and recommended that nebulizing procedures be performed in a separate identified area at the OPD or on the ward. In order to sustain the preventive measures, we introduced an infection control committee that comprises senior clinical and laboratory staff. The role of the committee is to address infection control issues and institute strategies to prevent reoccurrence of environmental microbial agents contaminating healthcare devices, equipment, and consumables.

Conclusions

Our study demonstrates that keen observation is important in order to identify outbreaks of HAI, and that HAIs and contamination can be avoided by adhering strictly to hygienic precautions. Our study also showed that *S. liquefaciens*, an environmental contaminant, has the potential to contaminate hospital consumables, devices, and equipment and ultimately contaminate and infect humans in a low-income country. We recommend strict aseptic procedure in the preparation of fluids for patient care, continuous cleansing and disinfecting of the healthcare environment, along with training and re-training of health workers. Also, we recommend that research to document the magnitude HAIs in low-income countries be encouraged. Thorough investigations will raise awareness among policymakers and strengthen political will to support initiatives that can prevent long stays in hospital that often stem from HAIs. In this way, unnecessary healthcare cost and unwarranted morbidity and mortality in healthcare facilities in low-income countries can be averted.

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Authors' contributions

UNI conceived and designed the study. UNI, AK, ML, and JM performed the laboratory investigation. UNI and GAM wrote the manuscript.

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