



# Investigation and successful control of a *Candida auris* outbreak at a tertiary health care facility in Kenya

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## ABSTRACT

**Objectives:** This study aimed to conduct an epidemiological and genomic investigation of a hospital outbreak of *Candida auris*, and implement measures for its control.

**Methods:** We collected demographic and clinical data from medical records of patients with *C. auris* from January 2017 to June 2019 after identifying increased cases in April 2019. Point-prevalence surveys for *C. auris* colonisation were conducted in the critical care units (CCU). Antifungal susceptibility testing and genomic sequencing of isolates were performed. A bundle of infection prevention and control measures was instituted.

**Results:** Thirty-two patients with *C. auris* were identified. All patients had a history of CCU admission. A total of 283 screening swabs were obtained and 57 isolates of *C. auris* identified. Antifungal susceptibility testing was performed on 48 isolates. All but two isolates were resistant to fluconazole; one isolate was also resistant to amphotericin B. Forty-one of 46 isolate genomes were clonally related and formed a distinct genetic cluster in Clade III. *C. auris* colonisation reduced from 42% in June 2019 to 1% in August 2019, and no new hospital-acquired colonisation was identified in the subsequent 9 months.

**Conclusions:** We identified a new genetic subcluster of Clade III *C. auris*. We also show that strict implementation of infection prevention measures can lead to substantial reductions in *C. auris* transmission.

## Introduction

*Candida auris* is a multidrug-resistant pathogen that has emerged globally [1]. Although initially associated with otitis media [2,3], it has subsequently been shown to cause invasive blood stream infections [3] and difficult-to-control hospital outbreaks [4,5]. Data point to the simultaneous emergence of different clades of *C. auris* in different parts of the world [6].

Risk factors identified for *C. auris* acquisition in intensive care units (ICUs) or long-term care facilities include mechanical ventilation, presence of indwelling catheters, and prior antibiotic or antifungal use [7], all of which are frequent in critically ill patients. *C. auris* infections are associated with high mortality, with some studies reporting all-cause mortality rates exceeding 50% [8]. Contamination of patient environments and equipment in hospitals is thought to be responsible for health care-associated outbreaks [5,9].

Critical data gaps exist in understanding the *C. auris* situation in sub-Saharan Africa. The true incidence of *C. auris* is likely underreported in resource-limited settings, such as Kenya, for various reasons. These include lack of equipment and supplies for *C. auris* isolation, limited training for identification and antifungal susceptibility testing, and lack of systematic surveillance of fungal diseases. Protocols for routine *C. auris* screening do not exist in many contexts.

*C. auris* is difficult to identify, particularly in regions of the world with low diagnostic capacity. It is often misidentified as other *Candida* species [10], especially *Candida haemulonii* or *Candida duobushaemulonii*, and accurate species identification requires specialised media and techniques [11].

In April 2019, we noted an increase in cases of *C. auris* candidaemia in the ICU of a large tertiary acute care hospital in Nairobi, Kenya, and undertook an outbreak investigation to identify cases of *C. auris*, characterise *C. auris* isolates, describe patient risk factors, and mitigate

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transmission. In this report, we describe the antifungal susceptibility and genomic characterisation of *C. auris* isolates to better understand health care-associated transmission in this context and describe the interventions taken to successfully control the outbreak.

## Methods

### Setting

The Nairobi Hospital is a large tertiary private hospital in Nairobi, Kenya, which provides care for patients in the East African region. It is a 450-bed capacity hospital with 24 adult critical-care beds. The critical care unit (CCU) is divided into 16 ICUs and eight high-dependency unit (HDU) beds. Patients requiring ventilation, inotropic support, continuous haemodialysis, or more intensive monitoring are admitted to the ICU, and care is de-escalated to the HDU once there is reduced requirement for intensive support. The ICU consists of 16 single patient rooms with central monitoring, and the HDU was one large room with six beds separated by curtains and two small single patient rooms.

### Establishing a response team

A multidisciplinary *C. auris* response team was established with hospital administrative support. The team consisted of an infectious disease specialist, a clinical pathologist with training in microbiology, infection prevention and control (IPC) nurses, and physicians involved in the care of patients admitted to the CCUs. The team conducted the outbreak investigation, implemented IPC measures, and strengthened antimicrobial stewardship with carbapenem restriction and regular review of antimicrobials prescribed to patients in the CCUs.

The investigation was conducted in two parts. In the initial part, we retrospectively reviewed charts and laboratory records of patients with invasive *C. auris* and searched for *C. auris* and other *Candida* species that could be misidentified as *C. auris* (e.g., *C. haemulonii* and *C. duobushaemulonii*) among patients admitted to The Nairobi Hospital between 1 January 2017 and 21 June 2019. A confirmed *C. auris* case was defined as a positive culture of *C. auris* isolated from a clinical sample (i.e., blood or other sterile site such as cerebrospinal fluid or intra-abdominal sample) in a patient admitted to the hospital between 1 January 2017 and 21 June 2019. A standardised case report form was used to extract anonymised patient data from medical records. Data collected from medical charts included patient demographics, hospitalisation history, underlying conditions, hospital procedures, treatments including antibiotics, antifungals and other drug therapies, and in-hospital all-cause mortality.

In the second part, we conducted point-prevalence surveys (PPS) in the CCU to determine the *C. auris* colonisation prevalence, with a baseline PPS conducted at the beginning of May 2019. Monthly PPSs were conducted over a 4-month period (from end of May 2019 to August 2019) to identify trends in *C. auris* colonisation rates. In addition, we screened all patients at admission to the CCU from May 2019 to March 2020. Patients were swabbed within 24 hours of admission unless admitted over the weekend or on a holiday when swabs were collected on the following business day. Patients not colonised with *C. auris* at admission were prospectively screened for *C. auris* every 2 weeks until discharge. The results were used to identify *C. auris* at admission, monitor its spread in the CCU, and implement contact precautions for patients with positive tests.

### Sampling method

A composite axillary and groin swab was taken to detect *C. auris* colonisation. Environmental screening involved sampling high-touch surfaces, as defined by the Centers for Disease Control and Prevention (CDC) environmental checklist for terminal cleaning (e.g., bed rails, bedside table, handles) [12], using a sponge in the CCU.

### IPC interventions

Patients with *C. auris* were placed in private rooms in the CCU with contact precautions. The IPC team directly observed environmental cleaning to identify gaps, followed by training of the housekeeping teams on effective routine and terminal cleaning of all patient rooms in the CCU from end of June 2019 to end of August 2019. Cleaning staff training included use of appropriate disinfectant types for *C. auris*, including composition and dilution, recommended disinfectant-surface contact time, and cleaning sequence. The effectiveness of environmental cleaning was assessed using Glo Germ™ (DMA International) and a fluorescent marker. High-touch surfaces in ICU rooms were marked with Glo Germ™ before terminal cleaning. After cleaning, the IPC staff calculated the proportion of high-touch surfaces from which Glo Germ™ had been successfully removed during the cleaning, and provided feedback to the environmental cleaning staff for immediate remediation and learning. Cleaning teams would repeat the terminal cleaning until all of the Glo Germ™ was removed.

### Laboratory methods

All samples were collected and immediately transported in salt dextrose broth to the laboratory; these were stored in a refrigerator at 4°C until processing. Primary cultures on Sabouraud dextrose agar (SDA) were initiated on the same day, with the enrichment broth incubated at 40°C for 5 days to increase diagnostic yield [13]. Plating on SDA was done after 5 days, or once broth turbidity was noted.

### Species confirmation

Any yeast growth was identified using VITEK 2 software version 8.01 (bioMérieux), and all colonies suspected to be *C. auris* were stocked. A total of 57 isolates were sent to the CDC Mycotic Diseases Branch laboratory in Atlanta, USA for identification, antifungal susceptibility testing, and whole genome sequencing (WGS). Species confirmation was done using Bruker MALDI-TOF.

### Antifungal susceptibility testing

Antifungal susceptibility testing was performed as outlined by the Clinical and Laboratory Standards Institute (CLSI) guidelines [14]. Custom prepared microdilution plates (Trek Diagnostic Systems) were used for the echinocandins (i.e., anidulafungin, caspofungin, and micafungin) and the azole fluconazole. Interpretive breakpoints for *C. auris* were defined on the basis of a combination of breakpoints that have been established for other closely related *Candida* species, epidemiological cutoff values, and the biphasic distribution of minimum inhibitory concentrations between the isolates with and without known mutations associated with antifungal resistance [14]. Resistance to anidulafungin and micafungin was set at  $\geq 4$  µg/ml, caspofungin at  $\geq 2$  µg/ml, and fluconazole at  $\geq 32$  µg/ml. ETEST (bioMérieux) was used for the polyene amphotericin B, and resistance was set at  $\geq 2$  µg/ml. Isolates were considered resistant to a class if resistance was documented for at least one drug within that class.

### Paired-end whole genome sequencing

In the preparation of WGS, DNA was extracted using the Quick-DNA™ (ZR) Fungal/Bacterial Miniprep Kit (Zymo Research, Irvine, CA, USA) according to the manufacturer's instructions. Genomic libraries were constructed and barcoded using the NEBNext Ultra™ DNA Library Prep Kit for Illumina (New England Biolabs, Ipswich, MA, USA) by following the manufacturer's instructions. Genomic libraries were sequenced on the HiSeq 2500 platform (Illumina, San Diego, CA, USA).

## Single-nucleotide polymorphism (SNP) analysis

Paired-end sequences that had at least 50× coverage were used for downstream analyses. FastQC (<https://www.bioinformatics.babraham.ac.uk/projects/fastqc/>) and PRINSEQ (PREprocessing and INformation of SEquence data) 8 were used to assess the quality of read data and perform read filtering. Read data were aligned against a previously described *C. auris* reference genome assembly (GenBank accession PEKT00000000.29) using the Burrows-Wheeler Alignment (BWA) tool. SNP variants were identified using SAMtools 11 and filtered using the publicly available SNP analysis pipeline NASP (<https://github.com/TGenNorth/NASP>) to remove positions that had <10× coverage, <90% variant allele calls, or that were identified by Nucmer as being within duplicated regions in the reference genome. Phylogenetic analysis was performed on SNP matrices using Molecular Evolutionary Genetics Analysis (MEGA X).

## Data analysis

We describe demographic features, movements within and outside the hospital, underlying conditions, treatment received during admission, and *Candida* culture information for patients with colonised or confirmed cases of *C. auris*. We calculated the proportion of colonisation swabs that were positive for *C. auris* and the proportion of isolates that were resistant to antifungals. Analysis was conducted using R version 4.2.3 (R Foundation for Statistical Computing).

## Ethics

Ethical approval was granted by The Nairobi Hospital Ethics Committee. Environmental screening was conducted as part of the hospital infection prevention procedures. Patients or their next of kin provided consent for the collection of screening swabs.

## Results

### Outbreak investigation

During the outbreak investigation, we identified 32 patients with invasive specimens positive for *C. auris*, all cases of candidaemia, between January 2017 and May 2019 (Figure 1). The median age was 55 years (interquartile range, 43–65); 59% were male, and all were of African descent. The in-hospital all-cause mortality rate was 64% for the 28 patients whose outcomes were available.

The most common reasons for ICU admission were sepsis (50%), pneumonia (34%), post-surgical care (25%), and stroke or other neurologic diagnosis (25%). Underlying comorbidities included hypertension (38%), diabetes mellitus (25%), malignancies (22%), and cerebrovascular events (19%). Two patients had HIV infection.

Nineteen percent of the patients reported treatment at a facility outside the country in the last 12 months, 19% had received health care at another facility in the country in the last 12 months, and 31% had been admitted to The Nairobi Hospital in the last 90 days.

Most (97%) patients had a central venous catheter inserted before the *C. auris* culture, and 44% had an acute dialysis catheter; 69% were on mechanical ventilation at the time of *C. auris* specimen collection. Most patients had a urinary catheter (97%) and a nasogastric tube (84%); 91% were receiving total parenteral nutrition; 69% had received steroids during this admission; and 75% had received blood product transfusions.

More than half (61%) of the patients had a positive culture for another multidrug-resistant organism during the admission when they also had *C. auris*. Specifically, six patients had methicillin-resistant *Staphylococcus aureus*, four had carbapenem-resistant *Acinetobacter baumannii*, and three had carbapenem-resistant *Pseudomonas aeruginosa*.

All of the patients had received antibiotics during the current admission, with the most common antibiotic being a carbapenem (84%). Half (50%) had received an antifungal before the isolation of *C. auris*, with the most common antifungal being an echinocandin (Table 1).

### *C. auris* colonisation

A total of 283 screening swabs from 234 patients were processed from May to August 2019.

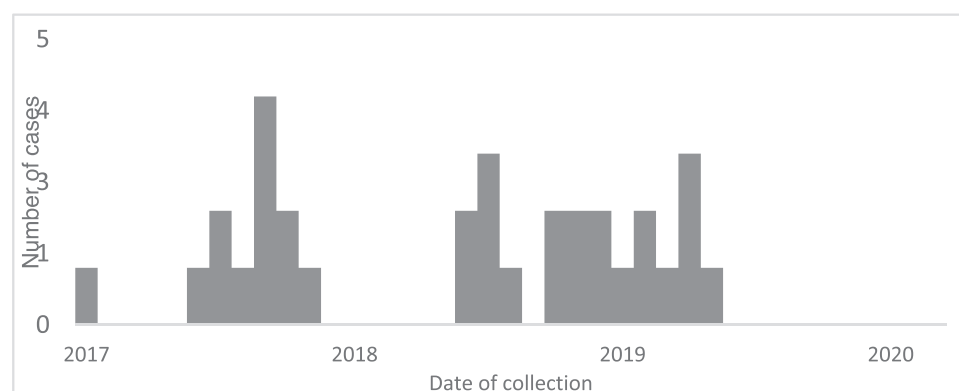
Positivity for *C. auris* was found for 20% (18/88) of PPS swabs and 7% (13/180) of admission screening swabs. The PPS results showed a rapid decrease in colonization; 43% (6/14) in May, 22% (12/54) in June, 9% (9/98) in July, and 1% (1/70) in August. No new *C. auris* invasive infections were identified from June 2019 to March 2020 (Figure 2).

### Environmental cleaning

The IPC team observed that ineffective disinfectants were used for cleaning and disinfection of floors, touch screens, surfaces of the medical equipment and trays, bed rails, trolleys, chairs, sinks, and other surfaces in rooms. Proper dilution and use of disinfectants with a sporicidal effect for *C. auris* were reinforced to clean rooms and high-touch surfaces. From the last week of June 2019 to the last week of August 2019, a total of 153 high-touch surfaces had Glo Germ™ application (17 surfaces each week). The proportion of surfaces with total Glo Germ™ elimination improved from 29% to 93%, and the number of times that cleaning had to be repeated to achieve perfect elimination of Glo Germ™ reduced from five to zero.

### Species confirmed by CDC

A total of 57 isolates were confirmed as *C. auris*, with 41 from skin swabs, six from blood, three from sputum, three from patient environment swabs, two from urine, and one each from cerebrospinal fluid

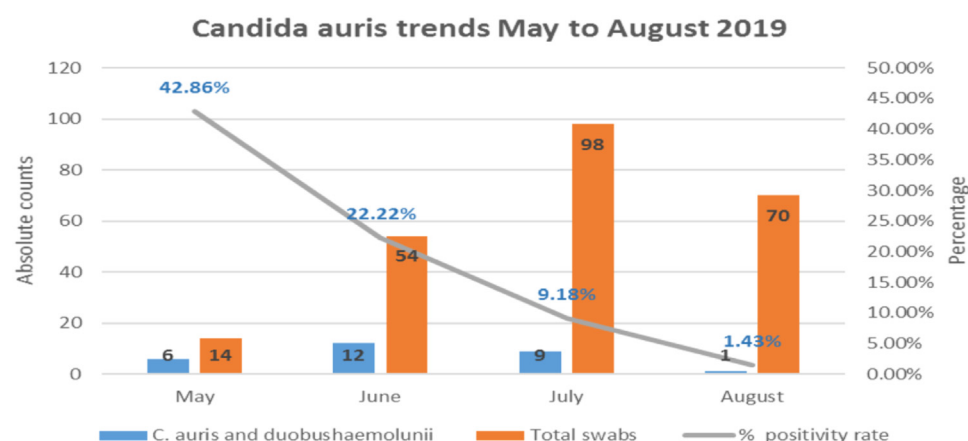


**Figure 1.** Epidemic curve of invasive *Candida auris* cases at a tertiary care hospital from 2017 to 2019, Nairobi, Kenya.

**Table 1**Patient characteristics, health care use, and outcomes of patients with *Candida auris* at a tertiary health care facility in Kenya.

Characteristic	Patients with <i>C. auris</i> (n = 32)
Age, median (interquartile range), years	55 (43-65)
Sex, male	19 (59%)
Race, African	32 (100%)
In-hospital all-cause mortality	18 (64%)
History of ICU admission	32 (100%)
<b>Reason for ICU admission</b>	
Sepsis	16 (50%)
Pneumonia	11 (34%)
Post-surgical	8 (25%)
Other infection	4 (13%)
Trauma	2 (6%)
Cardiac reason	4 (13%)
Stroke/neurologic issue	8 (25%)
Transplantation	2 (6%)
Other	15 (53%)
<b>Underlying conditions</b>	
Hypertension	12 (38%)
Diabetes mellitus	8 (25%)
Malignancies	7 (22%)
Cerebrovascular accident	6 (19%)
Surgical wound	6 (19%)
Chronic kidney disease	4 (13%)
Decubitus ulcer	4 (13%)
Chronic pulmonary disease	3 (9%)
Congestive heart failure	3 (9%)
Pregnancy	2 (6%)
HIV infection	1 (3%)
Transplantation	1 (3%)
Other	25 (78%)
Overnight hospital stay outside Kenya in the last 12 months	6 (19%)
Received health care at another county or subcounty in Kenya in the last 12 months	3 (9%)
Admission to this hospital in the last 90 days	10 (31%)
<b>Activities of daily living</b>	
Eating with tube or TPN	21 (66%)
Wheelchair-bound or bedridden	21 (66%)
Incontinent	15 (50%)
<b>Multidrug-resistant organism diagnosed during current admission</b>	
CRE	1 (6%)
CRPA	3 (18%)
CRAB	4 (24%)
VRE	1 (6%)
MRSA	6 (33%)
<b>Hospital procedures</b>	
Central venous catheter	31 (97%)
Acute dialysis catheter	14 (44%)
Endotracheal tube	21 (66%)
Mechanical ventilation	22 (69%)
Urinary catheter	31 (97%)
Nasogastric tube	27 (84%)
Total parenteral nutrition	29 (91%)
Blood transfusion	24 (75%)
<b>Medications</b>	
Corticosteroids	22 (69%)
Transfused with blood products	24 (75%)
Antibiotics	32 (100%)
Carbapenem	27 (84%)
<b>Antifungal medication before <i>C. auris</i> diagnosis</b>	16 (50%)
Fluconazole	6 (19%)
Voriconazole	6 (19%)
Echinocandin	10 (31%)
Liposomal amphotericin B	1 (3%)
<b>Antifungal medical after <i>C. auris</i> diagnosis</b>	19 (59%)
Fluconazole	3 (9%)
Voriconazole	3 (9%)
Echinocandin	16 (50%)
Liposomal amphotericin B	6 (19%)

CRAB, Carbapenem-resistant *Acinetobacter baumannii*; CRE, Carbapenem-resistant Enterobacteriaceae; CRPA, Carbapenem-resistant *Pseudomonas aeruginosa*; ICU, intensive care unit; MRSA, Methicillin-resistant *Staphylococcus aureus*; TPN, Total parenteral nutrition; VRE, Vancomycin-resistant Enterococcus.



**Figure 2.** *Candida auris* colonization on point-prevalence surveys.

**Table 2**  
Antifungal susceptibility profile of *Candida auris* isolates.

Antifungal	Minimum inhibitory concentration	Number of isolates	No susceptible	% Susceptible	% Resistant
Fluconazole	≥32	48	2	4	96.00
Amphotericin B	≥2	48	47	97.9	2.1
Anidulafungin	≥4	48	48	100	0.00
Caspofungin	≥2	48	48	100	0.00
Micafungin	≥4	48	48	100	0.00

and peritoneal fluid. Of the 57 isolates identified as *C. auris* by MALDI-TOF, 44 had been accurately identified by VITEK as *C. auris*, 10 as *C. duobushaemulonii*, one as *Candida dubliniensis*, one as *Candida lusitanae*, and one as *Candida glabrata*.

#### Antifungal susceptibility testing

A total of 48 isolates were tested, as nine duplicate patient samples were not tested. All but two isolates were resistant to fluconazole, one of which was also resistant to amphotericin B (Table 2).

#### Single-nucleotide polymorphism analysis

All isolates from this study clustered with Clade III (Figure 3). Of the 106 isolates included in the phylogenetic analysis, 45 were from the current study and 61 were previously published and included 38 from another facility in Kenya, six from South Africa, and 17 from the United States [15]. Thirty-six isolates from this study and one isolate from the United States formed a distinct subcluster different from previously published isolates from Kenya; isolates from this subcluster were highly clonal and differed from each other by no more than 23 SNPs. Five isolates clustered with the previously published Clade III Kenyan strains [15], while five were related to strains identified in the United States.

#### Discussion

We report an outbreak of *C. auris* in a Kenyan hospital, which was caused by Clade III *C. auris*. This outbreak was successfully controlled within a few months through implementation of a strict bundle of IPC interventions.

Control of *C. auris* outbreaks is often complicated and requires a multifaceted approach. Schelenz et al. [4] report an ongoing outbreak several months after it was first identified despite instituting various IPC measures. Setting up a multipronged approach that involves strong institutional support, clear leadership, a broad set of specialists (including infectious disease physicians, microbiologists, IPC nurses, and CCU doctors and nurses), cleaning, and a range of support staff involved in the

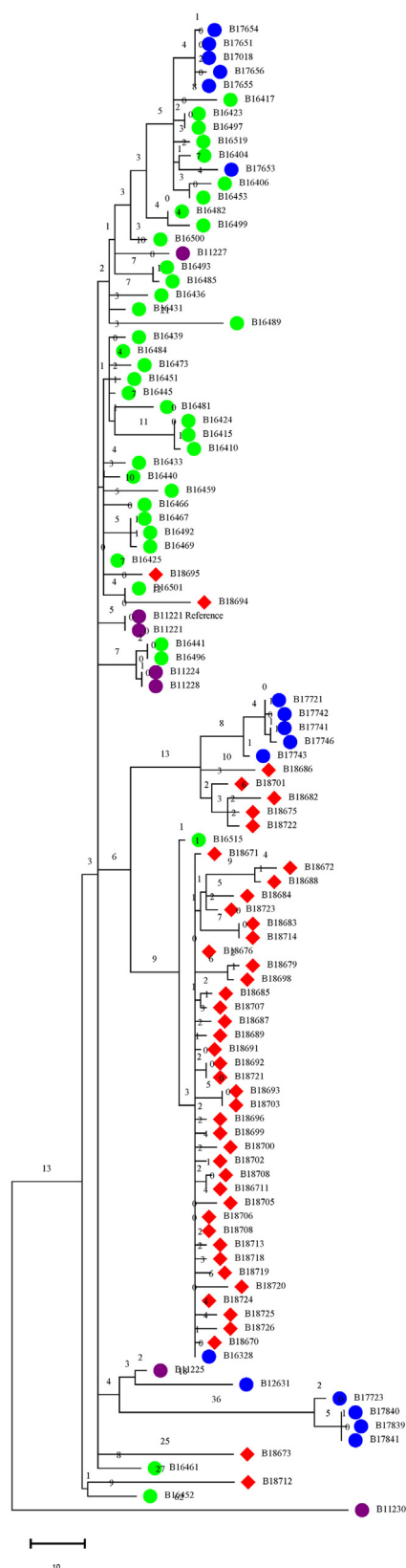
care of patients in the CCU may be key to the control of *C. auris* outbreaks.

Previous studies have identified outbreaks of *C. auris* in CCUs that were difficult to control because of colonisation and persistence in patient environments. Eyre et al. describe an outbreak of *C. auris* in a neurosciences ICU that was traced to reusable axillary temperature monitoring probes [9]. In a single hospital trust in London, an outbreak of *C. auris* persisted over several months despite the implementation of stringent IPC measures. The persistence was thought to be due to low-level environmental contamination [4]. Dbeibo et al. [16] report an outbreak of *C. auris* in multiple units of a large hospital that resulted from environmental contamination from a patient colonised with *C. auris*.

Implementing IPC measures was associated with the decline in the proportion of patients colonised with *C. auris* in serial PPS during the outbreak. In our outbreak investigation, we identified gaps in the IPC procedures that included suboptimal environmental cleaning, where cleaning included the use of disinfectants that were ineffective against *C. auris*. IPC measures have been identified as gaps in multiple settings. In an assessment of IPC in post-acute settings with a *C. auris* outbreak in California, Karmarkar et al. found significant gaps in the implementation of hand hygiene, transmission-based precautions, and environmental cleaning [17]. We implemented various strategies to strengthen IPC, including screening and isolation of colonised patients at the CCUs, improvement of environmental hygiene by using disinfectants with sporicidal effect for *C. auris*, and training of environmental cleaning staff to ensure cleaning of all relevant surfaces. These measures have been shown to be effective in controlling *C. auris* outbreaks in various settings, including in long-term care and nursing facilities and transplant centres [18,19]. In other facilities, removal of contaminated hospital equipment has led to control of outbreaks [9].

IPC measures may be easy to implement in resource-rich settings where there is less pressure for in-patient space and human resources. However, resource-limited settings can implement many IPC measures that do not require a significant increase in resources, such as improvement in hand hygiene and cohorting of patients as opposed to isolating patients in individual rooms, which may be unfeasible because of space limitations. Leveraging existing nursing staff to oversee IPC activities in units where it is not possible to have well-staffed stand-alone IPC committees may be a useful strategy where staffing challenges exist.





**Figure 3.** Maximum parsimony phylogenetic tree showing genetic relationships among isolates. Numbers next to the branches show the number of SNPs that differentiate nodes on the tree. SNPs were identified using the Northern Arizona SNP Pipeline against the reference, as described previously [16]. Phylogenetic analysis was performed using Molecular Evolutionary Genetics Analysis (MEGA). Isolates are color-coded according to patients' geographic origin: red, Kenya, current investigation; green, Kenya, previous study; blue, USA; purple, South Africa [15]. SNPs, single-nucleotide polymorphisms.

In our retrospective review of invasive *C. auris* infections, all patients had been admitted to the CCU, and most of these patients had a central venous catheter, had received mechanical ventilation, and had prior use of a carbapenem-class antibiotic, all of which have been identified as risk factors for *C. auris* candidaemia and colonisation [7]. Antimicrobial stewardship initiatives aimed at improving antibiotic prescribing practices and reducing unnecessary broad-spectrum antibiotic use are critical in reducing the risk of *C. auris* colonisation and infection [20].

Our results underscore how hospital outbreaks may remain unidentified, as *C. auris* is a difficult organism to identify, particularly in facilities using older identification platforms that may not incorporate *C. auris* in their databases [11]. In our facility, we identified 13 closely related *Candida* species that had been misidentified by VITEK 2. As part of the outbreak investigation, we enhanced the laboratory's capacity to identify *C. auris* from non-sterile samples by training laboratory personnel and providing the supplies required for *C. auris* culture, identification, and antifungal susceptibility testing. Many laboratories in the country and region do not have these measures as part of the standard operating procedures for isolation, identification, and antifungal susceptibility testing. This means that *C. auris* may often be missed or misidentified, and multiple silent outbreaks may be ongoing. Strengthening of laboratory capacity will be critical in combating the emergence and spread of *C. auris* in Kenya and other similar settings.

Resistance to fluconazole was high, and one isolate that was resistant to fluconazole was also resistant to amphotericin B. Resistance of *C. auris* to multiple antifungal agents has been variously described. A study in Kuwait describing antifungal susceptibility of 56 *C. auris* isolates found that all isolates were resistant to fluconazole and 19.6% were also resistant to voriconazole and amphotericin B, with one isolate being resistant to caspofungin [21]. Similar observations have been made in South Africa, where 91% of *C. auris* isolates were found to be resistant to at least one antifungal class [22], with one recent report of an outbreak of *C. auris* in a neonatal unit in which 6% of the isolates were resistant to amphotericin B and 2.3% of isolates were resistant to two antifungal agents [23]. Fluconazole is the most readily available systemic antifungal in Kenya and in many resource-limited settings, as amphotericin B is expensive and its associated toxicity makes administration difficult, particularly in settings with limited resources for monitoring for toxicity. In addition, echinocandins are expensive and only available in the larger and mostly privately owned hospital settings. Newer antifungal agents with potential activity against multidrug-resistant *C. auris*, such as manogepix [24], are not yet available. This makes local transmission of *C. auris* particularly concerning given the inability to appropriately offer treatment to patients with invasive *C. auris*, and makes surveillance and prevention even more crucial in such settings.

WGS was used to investigate transmission within and between the facilities. Previously, isolates of Clade I and Clade III were identified in another health care facility in Kenya [15]; however, only Clade III isolates were detected in our study. Although the two facilities with *C. auris* cases were located in the same city (one described herein and one from a previous study), each facility had its own unique subcluster of *C. auris*, suggesting ongoing transmission within these facilities and limited exchange of patients between the facilities. Specifically, no more than 23 SNPs differentiated isolates from the current outbreak subcluster, which is consistent with recent transmission [25]. Notably, two isolates from the current facility clustered with the subcluster of isolates from another facility, indicating that occasional exchange between the facilities did occur. However, no transmission of the introduced isolates was observed in our study. It is also notable that several isolates from the United States clustered with the subclusters from each of the facilities; however, no travel information was available for the US patients to determine whether they had recent travels to Kenya. These WGS results demonstrate that transmissions occurring within the facilities were likely the main contributors to the outbreaks.

Our study is subject to several limitations. First, because the *C. auris* outbreak was not immediately recognised, we likely underestimate the

scale of this outbreak given the lack of screening. Second, not all *C. auris* isolates were available for WGS. Finally, the intensive sampling and IPC measures implemented to control this outbreak might not be feasible in other settings with fewer resources.

In conclusion, we demonstrate a new genetic cluster of *C. auris* causing an outbreak in the CCUs of a hospital in Kenya. We also show that strict implementation of a strict IPC program can lead to rapid control of *C. auris* outbreaks in resource-limited countries.

### Declarations of competing interest

L.A.O. has received research grants from ViiV Healthcare and Gilead Sciences, and serves on a scientific advisory board for GSK. The remaining authors report no conflicts of interest.

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### Ethical approval

Ethical approval was granted by The Nairobi Hospital Ethics Committee. Environmental screening was conducted as part of the hospital infection prevention procedures. Patients or their next of kin provided consent for collection of screening swabs.

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### Author contributions

LAO - conceptualization, data collection, formal analysis, investigation, methodology, project oversight, writing-original draft, writing-review and editing; AK - Data collection, formal analysis, investigation, methodology, writing - review and editing; RN - Data collection, methodology, visualization, writing - review and editing; CM - Data collection, methodology, writing - review and editing; EB - Formal analysis, methodology, writing - review and editing; LG - Formal analysis, writing - review and writing; EB - Investigation, methodology, writing - review and editing; JO - data collection, writing - review and editing; RN - data collection, writing - review and editing; MN - data collection, writing - review and editing; RCB - data collection, writing - review and editing; AL - formal analysis, visualization, writing - review and editing; ML - writing - review and editing; MT - Data collection, formal analysis, investigation, methodology, visualization, writing - review and editing

### Disclaimer

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the US Centers for Disease Control and Prevention. Use of trade names and commercial sources is for identification only and does not imply endorsement by the US Department of Health and Human Services or the US Centers for Disease Control and Prevention.

### Note

This activity was reviewed by US Centers for Disease Control and Prevention (CDC) and was conducted consistent with applicable federal law and CDC policy (e.g., 45 C.F.R. part 46, 21 C.F.R. part 56; 42 U.S.C. §241(d); 5 U.S.C. §552a; 44 U.S.C. §3501 et seq).

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