Journal of Infection 79 (2019) 601–611

Contents lists available at ScienceDirect

Journal of Infection

journal homepage: www.elsevier.com/locate/jinf

Candida auris outbreak: Mortality, interventions and cost of sustaining control

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ARTICLE INFO

Article history:
Accepted 17 September 2019
Available online 23 September 2019

Keywords:
Fungal outbreaks
Emerging infections
Expense

SUMMARY

Objective: *Candida auris* has recently emerged as a global cause of multidrug resistant fungal outbreaks. An outbreak occurred at a tertiary care center in London in 2016. Transmission characteristics, interventions, patient outcomes and cost of resources are described.

Methods: Outbreak interventions included patient isolation, contact screening, single-use equipment, environmental screening and decontamination, staff education, and enhanced surveillance. Risk factors for infection were recorded. Survival probabilities of patients with *C. auris* and other *Candida* bloodstream infections (BSI) were calculated. Antifungal susceptibility and epidemiological typing were performed. Actual and opportunity costs of interventions were determined.

Results: 34 patients acquired the organism including 8 with BSI. Clinical infection was significantly associated with prolonged hospital stay, haemodialysis and antifungal therapy. Variable susceptibility to amphotericin and the triazoles was seen and isolates clustered with the South Asian strains. No significant difference was detected in the survival probabilities of *C. auris* BSI compared to other candidemias. Outbreak control cost in excess of £1 million and £58,000/month during the subsequent year.

Conclusion: *C. auris* outbreaks can be controlled by a concerted infection control strategy but can be expensive. Transmission maybe prolonged due to patient movements and unidentified transmission mechanisms.

Introduction

Candida auris is a recent addition to multidrug resistant nosocomial pathogens. Since its first description in 2009 [1,2], outbreaks have been reported across the globe [3–6]. Diabetes mellitus, critical care admission, vascular surgery, long hospital admissions and antifungal therapy are some of the described risk factors for invasive disease [7,8]. A number of concerning features distinguish this pathogen from other yeasts. Individual isolates can be resistant to multiple antifungal agents including polyenes and echinocandins while fluconazole resistance is universally reported. Most biochemical fungal identification kits are unable to accurately characterize this yeast and yield a variety of misidentifications [9]. Finally, the organism is known to persist in the healthcare environment and transmission between patients can be very rapid. Work is still needed to understand transmission characteristics and determine optimal outbreak interventions.

From July 2016 to February 2017, an outbreak of *C. auris* occurred at a large tertiary care teaching hospital in south-east London, UK, one of only three large outbreaks to be reported in the UK [8,10].

Here we describe the affected population, risk factors for clinical infection, secondary attack rates, clinical outcomes and outbreak interventions followed by a cost analysis of the resources required to halt transmission and maintain control.

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https://doi.org/10.1016/j.jinf.2019.09.007
0163-4453/

Methods

Setting: King's College Hospital is a large tertiary care teaching hospital over 3 sites in south-east London. The outbreak was limited to the main site which has 1017 beds (including 116 dedicated to acute care) and specialist services including liver transplantation, haemato-oncology, neurosurgery, vascular and cardiothoracic surgery, cardiology, renal including outpatient dialysis, complicated diabetes, Trauma Centre (66 intensive care beds in 4 units) paediatric and women's health services. The hospital largely serves a poor socioeconomic population from diverse ethnic backgrounds.

The hospital Infection Control Team is comprised of 0.4 whole time equivalent (WTE) infection control doctor, 6 WTE infection prevention and control nurses (IPCNs) and 3 WTE surveillance staff. During the outbreak period, transmission was detected in 4 adult intensive care units and 3 wards consisting of 126 beds including 11 en suite and 23 non en suite side rooms.

Design: The outbreak evaluation was ambidirectional; epidemiological and screening data was collected prospectively whereas the patient risk factors and cost analysis was done retrospectively.

Introduction into the hospital: The first case of *C. auris* managed by the hospital was a known *C. auris* colonized patient transferred for specialist healthcare in April 2016 from another hospital where an outbreak with the South Asian strain was ongoing. Infection control advice was supplied with the transfer documents and strict precautions implemented from the time of admission. No further cases were seen at that time. A second patient admitted in July 2016 grew an organism identified as *C. haemulonii* from an abdominal drain fluid sample on admission. This patient was not recognized as a carrier of *C. auris* until October 2016 when greater awareness of *C. auris* led to submission of the organism for identification to the PHE Bristol Mycology Reference Laboratory (MRLB). This patient's admissions did not overlap with the first case in time or ward location and the patient was also a healthcare transfer but from a different hospital. However, it is possible that in the absence of routine screening, other colonized patients could have served as intermediaries. The organism could also have been acquired at the referring hospital (however there was no outbreak or increased incidence reported there). The delay in identification led to a delay in isolation and exposure to a number of patients who shared common wards. Contacts were identified and screened for *C. auris*. A retrospective review of the laboratory database from 2011 to 2016 was performed to look for any *C. auris, C. haemulonii* or *C. duobushhaemulonii* isolates prior to this outbreak.

Case definitions: A *C. auris* case was defined as a patient with *C. auris* detected from any site. Immediate (close) contacts were defined as inpatients who shared a bay (partially enclosed area in a ward) with a *C. auris* case for any length of time. An episode of *C. auris* bloodstream infection (BSI) was defined as blood culture with *C. auris* from a patient with signs of clinical infection. Confirmed *C. auris* infection was defined as a case with a positive *C. auris* culture from a sterile site with clinical evidence of infection which required treatment with an antifungal. Probable *C. auris* infection was defined as a case with a positive culture from a non-sterile site accompanied by clinical evidence of infection requiring treatment with an antifungal.

Colonization was defined as *C. auris* isolation from any superficial site in a patient without clinical signs of *Candida* infection. A high-risk area was defined as a clinical area admitting patients susceptible to invasive infections with fungi, namely intensive care units, liver transplant wards, haemato-oncology and wards housing diabetic patients with chronic ulcers. A suspected case was a patient who had yeast species with which *C. auris* is commonly confused, prior to confirmation of exact identity (from any site). Central line related BSI (CLABSI) and intra-abdominal infection was defined as per CDC surveillance criteria [10].

Risk factors and outcomes of patients with clinical infections: Clinical features, risk factors (indwelling devices, ICU stay, hospital stay, haemodialysis, immunosuppression, presenting illness, antimicrobials, co-infection with MDR organisms, prior surgery) treatment and outcome details were extracted from electronic and paper-based patient records. Charlson comorbidity index (CCI) was calculated. All patients with confirmed and probable *C. auris* infections ($N = 14$) were compared with colonized patients ($N = 20$) to look for risk factor associated with developing clinical infections.

Mortality of C. auris BSI vs BSI due to other Candida spp: A retrospective cohort analysis was undertaken to determine the difference in all-cause mortality between patients with *C. auris* BSI vs those with BSI caused by any other *Candida* species and a subset analysis of *C. auris* vs *C. albicans* BSI. All patients in whom *Candida* spp. was isolated from blood cultures between January 2015 and February 2018 were identified from the hospital Laboratory Management System. Episodes were de-duplicated and each patient was only included once. Dates of deaths were retrieved from medical notes and patient administration system linked to the Office of National Statistics death registry. Univariate Kaplan–Meier curves and log-rank tests were performed using Stata v14.2.

Secondary attack rates: Immediate contacts of each newly identified patient were screened and records were kept on the results of screening. Crude secondary attack rates (CSAR) were calculated for all immediate close contacts of each positive patient.

Laboratory screening protocol, culture and typing: A screening protocol was developed to detect *C. auris* from nose, throat, axilla and groin swabs and catheterized urine. Samples were pooled and plated onto Chromogenic *Candida albicans* agar (Thermoscientific™), incubated for up to 48 h at 37 °C. Pale pink to colorless colonies were further sub-cultured onto blood agar and then identified by MALDI-TOF (Biomerieux version 4.01). If no acceptable identification was produced, then a Vitek 2 (version 7.01, Biomerieux) test was performed. Isolates identified as any of previously published misidentifications namely *C. haemulonii, C. guillermondii*, C. *famata, C. lusitaniae, R. glutinis, S. cerevisiae, C. sake* and *C. parapsilosis* [4,9] and those with no acceptable identification by either method were sent to the MRLB for definitive identification by MALDI-TOF biotyper (Bruker-Daltonics, Bremen, Germany) and PCR amplification and sequencing of the D1D2 portion of the 28S rDNA gene, and the internal transcribed spacer region 1 (ITS1) [10].

Antifungal susceptibility testing was done on Vitek 2 (version 7.01, Biomerieux) and confirmed by the broth microdilution using CLSI methodology at the MRLB when possible. Interpretation was by CLSI criteria for *C. albicans* [11].

Organism shedding in the environment was determined by placing 5 settle plates of SDA within 3 m from patient beds. The location of plates was dependent on availability of suitable spaces which would be undisturbed during the course of healthcare hence were variable with room layouts but always included the windowsill. One set of plates during the day was replaced with fresh plates in the same position for overnight exposure to complete 24 h. Environmental surfaces were sampled by sterile swabs moistened in sterile saline and rolled over a $10cm^2$ surface area and then plated onto Brilliance™ Candida agar. Plates were incubated and colonies were Gram stained. All yeasts were identified as above and numbers added to determine *C. auris* 24 h environmental colony counts (ECC).

Surveillance data and statistical methods: All *C. auris* cases were recorded on Microsoft Excel as a line list which was updated daily. Epidemic curve and time lines were updated regularly. Hand hygiene and environmental cleanliness scores were collated and reviewed regularly.

Outbreak interventions:

(a) Early identification and isolation: Upon suspicion of a possible outbreak (case ID 3 onwards), active screening of immediate contacts of all new positive cases was started (one set for 3 consecutive days). From October 2016, an admission and weekly screening program was introduced at the four adult intensive care units as the risk of invasive infections was deemed highest in this clinical cohort. In other clinical areas where *C. auris* cases were admitted, one off screening of the whole ward (three samples over three days per patient) was undertaken at random time points and as the last known *C. auris* cases were discharged, to document possible undetected colonization in the ward. In addition, in all high-risk areas, passive surveillance was performed by identifying all yeast isolates from clinical samples to species level. Patients were placed in mandatory single room isolation if confirmed or suspected *C. auris* colonies were identified. Contacts were kept in isolation until one set of screening swabs was negative. A further two screening sets were taken for contacts although isolation was not always possible. All confirmed cases were placed in isolation rooms for all subsequent readmissions.

(b) PPE and disinfection: Surfaces in all affected areas were cleaned with 1000 ppm active chlorine or chlorine based wipes. Cleaning frequency was increased in affected areas to three times per day. For terminal disinfection, a deep clean with chlorine containing agents and curtain change was followed by 35% hydrogen peroxide vapor based fumigation. If fumigation was not possible due to ward or room permeability to gas, moveable equipment was fumigated in an alternate room. A policy for skin decolonization with 2% aqueous chlorhexidine wipes existed in all ICUs before the outbreak. This was extended to include all cases outside ICUs as well. This procedure takes between 15 and 25 min per patient. Chlorhexidine dressings for IV lines (Biopatch®, Ethicon) (4% in alcohol) were in use in the Trust selectively for MRSA colonized high risk patients. Following identification of the outbreak, Biopatch® was used for all patients in the adult ICUs. Disposable pillows and blood pressure cuffs were introduced in the affected wards where these were not already in use. Plastic single use long sleeve gowns were used for the clinical care of all patients with confirmed or suspected *C. auris* and their contacts (until one negative screen). Masks and visors were recommended only if performing an exposure prone procedure.

(c) Guidance, training and communication: A local infection control guidance document was developed based on published guidance from Public Health England [12], informal discussions with other affected hospitals worldwide and the available scientific literature [8,13,14]. *C auris* communication bulletins, hospital wide email alerts, computer screen savers and posters were issued to raise awareness. In affected areas, IPC practitioners undertook regular training sessions for clinical staff. Daily infection control meetings were held in the affected areas to ensure results were communicated early and patient's movements were planned for in advance. A hospital wide outbreak control meeting was held weekly with public health and senior hospital management representation to discuss new developments and ensure management strategies were implemented. Printed guidance was prepared for patients, relatives and carers to enable them to understand the organism. *C. auris* care plan and IPC checklists were produced. If cases were transferred to another hospital, transfer documents included information on presence of colonization. If colonization was detected after discharge, patient's GP was informed of the result and given an information sheet to guide further management. Other healthcare facilities receiving cases or contacts under investigation were informed by telephone and on discharge letters. An evening symposium was held with external invitees to encourage open sharing of experiences.

Cost analysis: Financial impact of the outbreak was calculated for actual and opportunity costs of additional length of stay, antifungal treatment, laboratory screening, disposables, additional cleaning, and staff time spent in managing the outbreak.

The burden of *C. auris* 'Additional Length of Stay (ALoS)' was calculated for 23/26 cases detected during the outbreak by matching controls closest to the cases in time. Three cases were excluded (case ID 1 was known to be positive before admission as a healthcare transfer from another hospital and confirmation of positive results for two patients (cases 20 and 24) arrived after they were discharged). The 10 cases detected post outbreak during the follow up period were excluded from this analysis.

Matched controls were extracted from the patient administration system (PiMS) using an SQL query for each of the 23 positive cases. Ten inpatients closest to the outbreak start date were matched on primary admission diagnosis code, gender and age. Where less than ten controls were found in the last five years, the age interval was increased marginally to facilitate this. Seven cases (2, 8, 11, 12, 15, 19 and 22) were excluded as they had a shorter length of stay post *C. auris* detection than their control group average length of stay.

Additional length of stay (ALoS) was calculated as follows:

- For 9 cases (ID 3, 4, 7, 10, 14, 16, 21, 25, and 26) where the matched controls mean inpatient days was less than the case admission to first positive date – the ALoS was the number of days from first positive date to discharge date.

  i.e. If Control Avg <Adm to +ve date: ALoS = +ve date to discharge.

- For 7 cases (ID 5, 6, 9, 13, 17, 18, and 23) where the matched controls mean of inpatient days was more than the case admission to first positive date – the ALoS was the total number of inpatient days minus the controls mean length of stay.

  i.e. If Control Avg >Adm to +ve date: ALoS = Total LoS-Control Avg

ALoS days for 12 cases who were admitted to ICU post positive or diagnosed in ICU was also calculated to apportion their stay in ICU. Two patients had 6 days stay on HDU which was included in ward stay costs.

Cost of managing the outbreak from August 2016 to Feb 2017 and ongoing costs over the one year follow up period (March 2017–Feb 2018) were calculated separately (details in Supplementary data).

Results

Outbreak extent: The retrospective electronic review of the laboratory database revealed no *Candida auris* amongst clinical samples in the laboratory database. A single sample from 2015 identified as *C. haemulonii* was confirmed as such by the MRLB suggesting that *C. auris* was a new introduction into the hospital. The first patient managed in the hospital was admitted in April 2016. No further cases were seen until July 2016 following which there were 25 other cases to mid-January 2017. The outbreak was declared over in February 2017 when no new cases had been identified for 6 weeks. After Feb 2017, although the outbreak was over, one patient (case ID 3) remained in hospital and 11 positive cases were

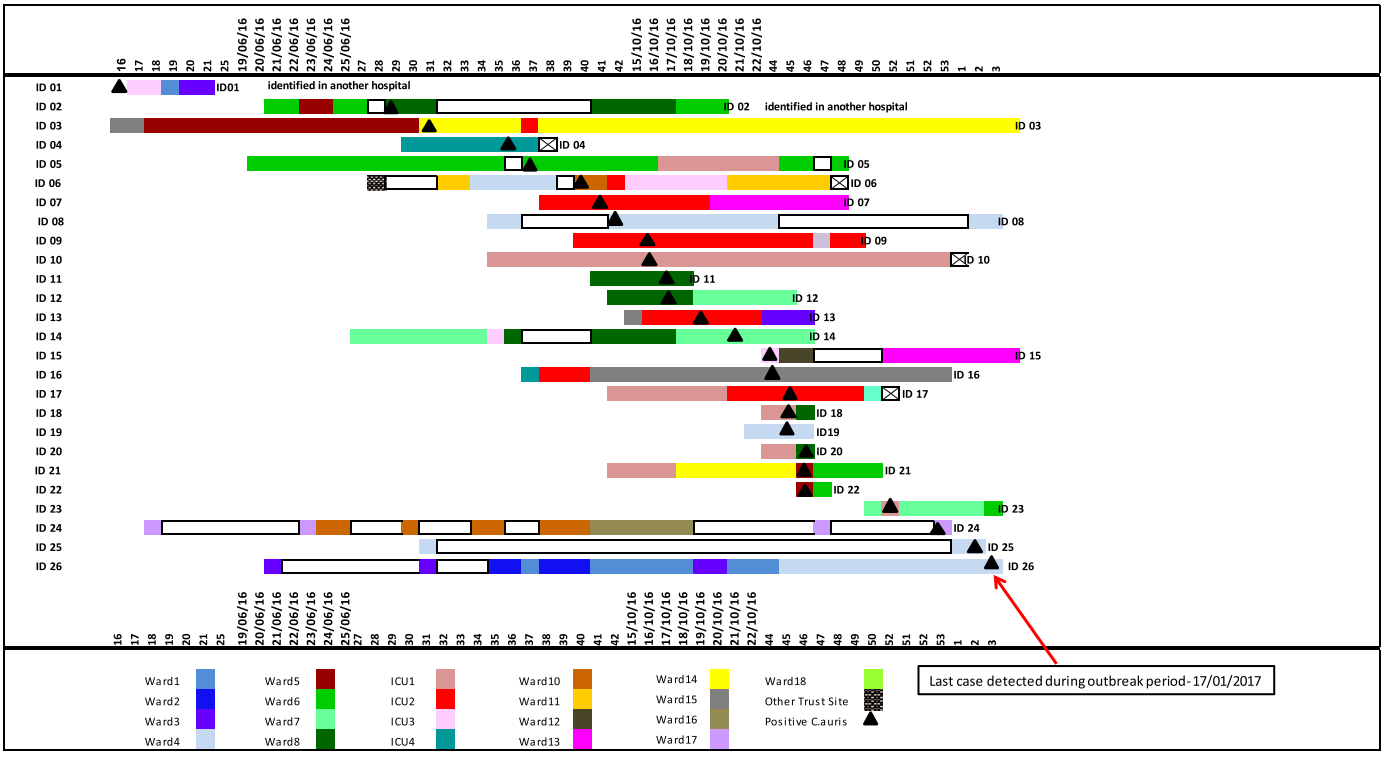

Fig. 1A. Time line outbreak (May 2016–Feb 2017).
This graph represents locations of affected patients during their hospital stay before and after acquiring C. auris. Y axis has patient numbers and x axis has time (weeks of the year). Weeks 26 and 43 are expanded to show details. Transmission was detected in 4 ICUs and 3 wards (wards 4, 5, and 8). Other wards are shown as they were part of the patient tracing exercise. Blank bars indicate patient was not admitted in the hospital during that time.

readmitted (case IDs 5, 7, 8, 9, 14, 19, and 22–26). Occasional transmission events were seen and 2 patients were detected on readmission screening having had contact with known cases in previous admissions or in previous wards (case IDs 29 and 33). Overall ten new cases were identified over this period. Figs. 1A and 1B represent the time lines of affected patients during and after the outbreak respectively. Fig. 2 shows the epidemic curve of the outbreak and one year follow-up period.

Clinical infections and risk factors: Fourteen patients were clinically infected during the outbreak and follow up period (including 8 patients (9 episodes) with BSI). The commonest risk factors for acquisition of *C. auris* were antimicrobial (100%) and antifungal (85.7%) treatment within the past 30d, surgery within past 30d (71.4%), presence of CVC (78.6%) and urinary catheter (78.6%) at the time of diagnosis. Source of BSI was identified as central line associated (CLABSI) in 5 patients, intra-abdominal in 2 patients and urinary catheter associated in one patient. One patient (case ID 10) had two episodes of *C. auris* BSI but there were no metastatic complications. Risk factors, treatment, root causes and outcomes are listed in Table 1 and Supplementary information Table S1 and Fig. S1. Risk factors for clinical infection are compared in Table 2. Significant risk factors include ITU stay ($p = 0.03$), long hospital stay ($p = 0.004$), presence of CVC ($p = 0.013$), haemodialysis ($p = 0.042$) and prior antifungal therapy ($p < 0.001$). Age, immunosuppression, mortality and other factors were not associated with significance.

Mortality: All-cause 30d mortality following *C. auris* BSI was 12.5% and for all clinically infected patients was 14.3%. In one patient (case ID 5) clinical infection was identified 14 months after initial colonization and was listed as the immediate cause of death. However no significant difference was found between survival probabilities of patients with *C. auris* BSI when compared to those with BSI caused by *C. albicans* nor when compared to all *Candida spp*. BSI combined Fig. 4. Most clinically infected patients were treated with an echinocandin, though amphotericin and voriconazole were also used as per sensitivity of isolates (see SupplementTable S1).

Environmental surveillance and secondary attack rates: C. auris was detected from 2 samples around one patient only (bed rails and dining trolley) of a total of 48 environmental swabs collected from high touch point areas. ECC were available for 11 cases and *C. auris* was detected in the settle plates of 10 patients (range 0–19, median 1colony). During the outbreak and follow up period 233 immediate contacts were identified, 55.8% were screened and 9 were positive giving an average CSAR of 6.9%. ECC and CSAR for the immediate close contacts of each patient can be seen in Fig. 3.

Antifungal susceptibility data was available for 54 isolates from 34 patients. Uniform resistance to fluconazole was observed ($N = 54$, MIC range 8–>64.0 mg/L), whereas susceptibility to other antifungals was variable (amphotericin $N = 47$, MIC range 0.25–8.0 mg/L; anidulafungin $N = 41$, MIC range 0.03–1 mg/L; voriconazole $N = 47$, MIC range 0.06–>16 mg/L flucytosine $N = 32$, MIC range <0.12–>64 mg/L; caspofungin $N = 11$, MIC range 0.125–>32; itraconazole $N = 5$, MIC range 0.03–0.25) . Nineteen patients had isolates resistant to at least two of the tested classes of antifungals (details in Table 3).

All isolates clustered with the Indian/Malaysia/Pakistani/Kuwaiti lineage of *C. auris* (Fig 5).

Outbreak control: Most infection control precautions were implemented at the start of the outbreak though as awareness of the outbreak increased; implementation of interventions and awareness of hygiene and cleaning measures became more widespread than at the start. The monthly hand hygiene compliance for the hospital as a whole was above 90% (range 90.6–94.7%, mean 93%) from May 2016 to February 2017. However hand hygiene compliance in the ICUs was 85.8% and 86.8% in May and June 2016. From

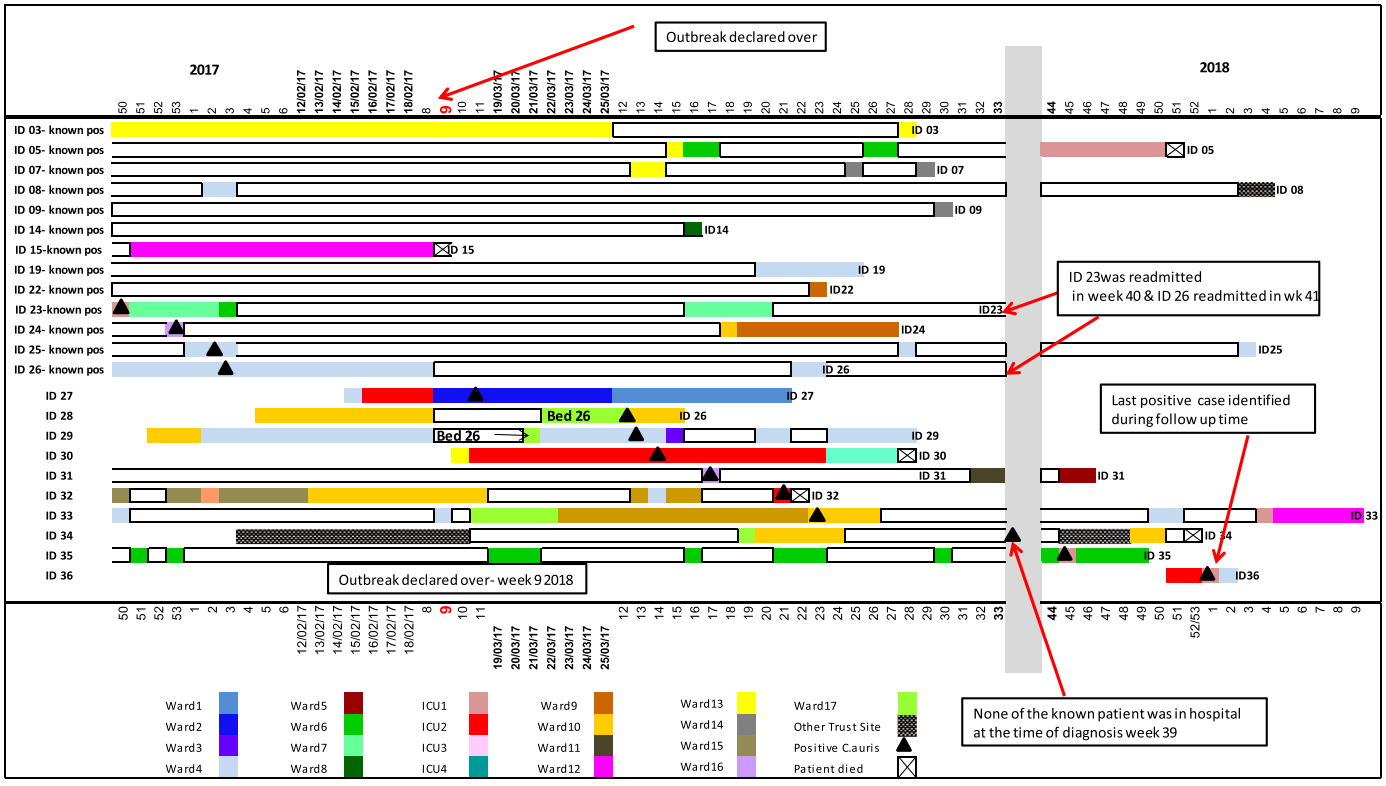

Fig. 1B. Timeline post outbreak (March 2017–Feb 2018).
This graph represents locations of affected patients during the one year follow up period before and after acquiring C. auris. Y axis has patient numbers and x axis has time (weeks of the year). Weeks 7and 12 are expanded to show details and weeks 34–43 are compressed to fit in the allotted space. Transmission was detected sporadically in various parts of the hospital. All wards which were part of the patient tracing exercise are shown. Blank bars indicate patient was not admitted in the hospital during that time.

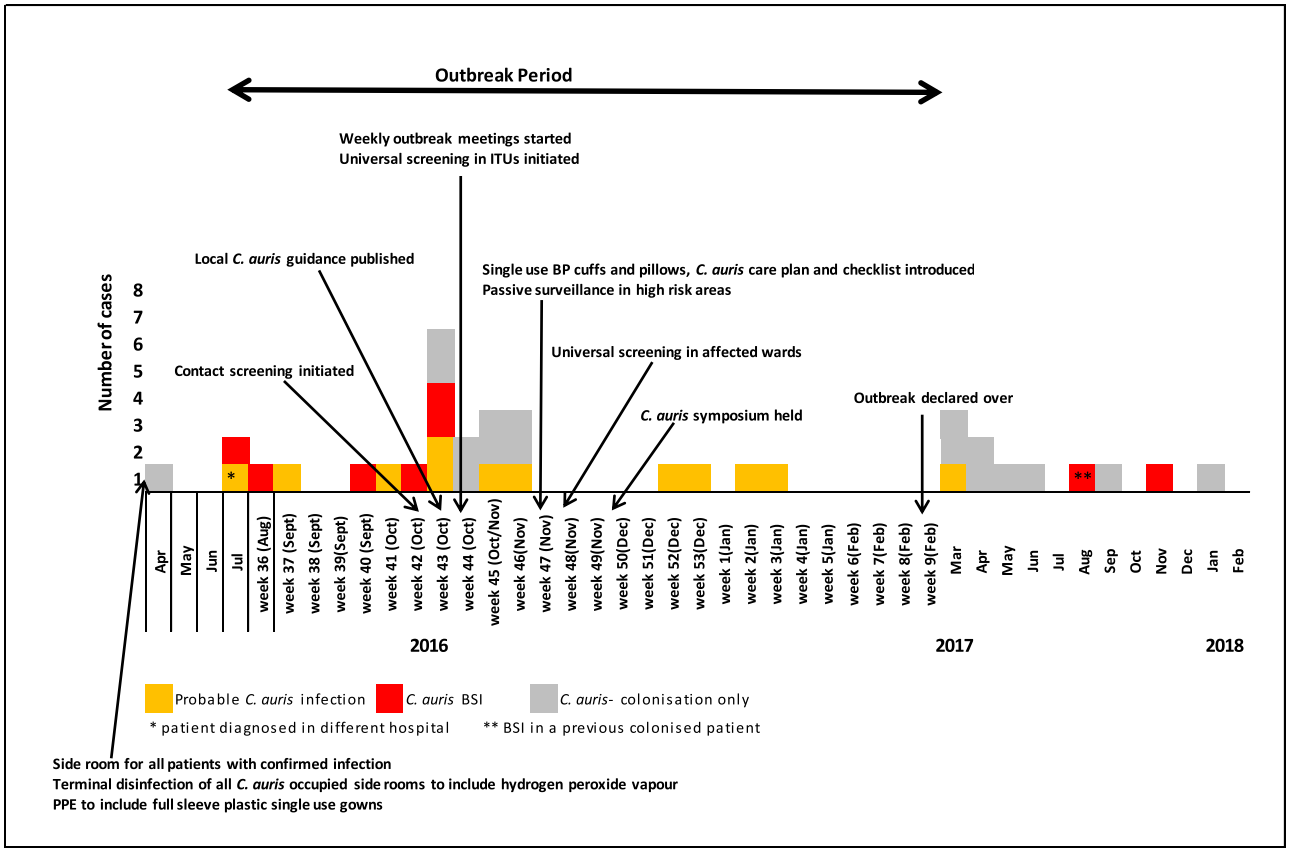

Fig. 2. Epidemic curve of *C. auris* cases from April 2016 to February 2018.

Table 1
Risk factors and outcomes of patients with *C.auris* clinical infections.

	Confirmed infection $n=8$ (%)	Probable infections $n=6$ (%)	Total $n=14$ (%)
Risk factors			
Median age in years (range)	55 (23–85)	64.5 (27–75)	57.5 (23–85)
Male	3 (37.5)	6 (100)	9 (64.3)
APACHE II score, median (range) $n=8$*	18.5 (14–28)	22, 26	21 (14–28)
Previous antimicrobial treatment (< 30 days)	8 (100)	6 (100)	14 (100)
CVC	8 (100)	3 (50)	11 (78.6)
Urinary catheter	6 (75.0)	5 (83.0)	11 (78.6)
Any previous surgery (< 30 days)	5 (62.5)	5 (83.3)	10 (71.4)
Previous gastro surgery (< 30 days)	4 (50.0)	4 (66.7)	9 (64.3)
Diabetes	4 (50.0)	1 (16.7)	5 (35.7)
Transplant	3 (37.5)	2 (33.3)	5 (35.7)
Nutrition (PEG/NG/TPN**)	5 (62.5)	3 (50.0)	8 (57.1)
Mechanical ventilation	4 (50.0)	1 (16.7)	5 (35.7)
Haemodialysis	5 (62.5)	2 (33.3)	7 (50.0)
Neutropenia ($<1.5 \times 10^9$/L)	1 (12.5)	0 (0)	1 (7.1)
Previous candida spp (<30 days)	6 (75.0)	2 (33.3)	8 (57.1)
Previous antifungal treatment (30 days)	6 (75.0)	6 (100)	12 (85.7)
Anidulafungin and then Fluconazole (or vice versa)	3 (60.0)	1 (16.7)	4 (36.4)
Anidulafungin	2 (40.0)	2 (33.3)	4 (36.4)
Fluconazole	1 (16.7)	2 (33.3)	3 (25.0)
Nystatin	0 (0.0)	1 (16.7)	1 (9.1)
Antifungal exposure in days, median	9.5 (3–30)	7.5 (4–30)	8.5 (3–30)
Therapeutic measures			
CVC removal	8 (100)	–	
Antifungal treatment			
Started in days	3 days (0–6)	0 (2), 9, 12 and 40	
Treatment			
Anidulafungin	8 (100)	6 (100)	
Amphotericin	3 (62.5)		
Voriconazole	2 (25.0)		
Micafungin	2 (25.0)		
Caspofungin	1 (12.5)		
30 day all-cause mortality	1 (12.5)	0	1 (7.2%)
60 day all cause mortality	2 (25.0)	3 (21.4)	

* APACHE II score was only available for 8 patients (6 BSI and 2 probable cases).
** TPN only 2 patients.

Table 2
Risk factors for developing clinical infection.

Variable	Infection ($N=14$)	Colonization ($N=20$)	p-value
Male	8 (57.1%)	14 (70%)	0.726
Age group			
23–35 years	2 (14.3%)	0 (0%)	
36–55 years	4 (28.6%)	3 (15.0%)	
56–75 years	6 (42.9%)	13 (65%)	
76–95 years	2 (14.3%)	4 (20%)	0.245
ICU stay	12 (85.7%)	9 (45%)	0.030
Charlson comorbidity index			
0–2	3 (21.4%)	2 (10.0%)	
3–4	3 (21.4%)	5 (25.0%)	
≥5	8 (57.1%)	13 (65.0%)	0.779
Duration in hospital after colonization (>30 days)	10 (71.4%)	4 (20.0%)	0.004
Diabetes mellitus	5 (35.7%)	13 (65.0%)	0.092
Malignancy	1 (7.1%)	1(5%)	1.000
Mortality (days from first detection)			
30 days	1 (7.1%)	1(5%)	1.000
90 days	3 (21.4%)	1(5%)	0.179
Mortality (days from clinical infection)			
30 days	2 (14.3%)		
90 days	4 (28.6%)		
Immunosuppression	3 (21.4%)	2 (10.0%)	0.328
Haemodialysis dialysis	7 (50.0%)	2 (10%)	0.017
Broad-spectrum antibiotics	11 (78.6%)	16 (80.0%)	1.000
Presence of CVC	12 (85.7%)	8 (40.0%)	0.013
Presence of urinary catheter	9 (64.3%)	14 (70.0%)	0.726
Antifungal therapy up to 30 days before detection	11 (78.6%)	3 (15%)	<0.001
Coinfection with other MDR organisms	5 (35.7%)	6 (30%)	0.726
Surgery	10 (71.4%)	15 (75%)	0.816
Organ transplant	3 (21.4%)	3 (21.4%)	0.672

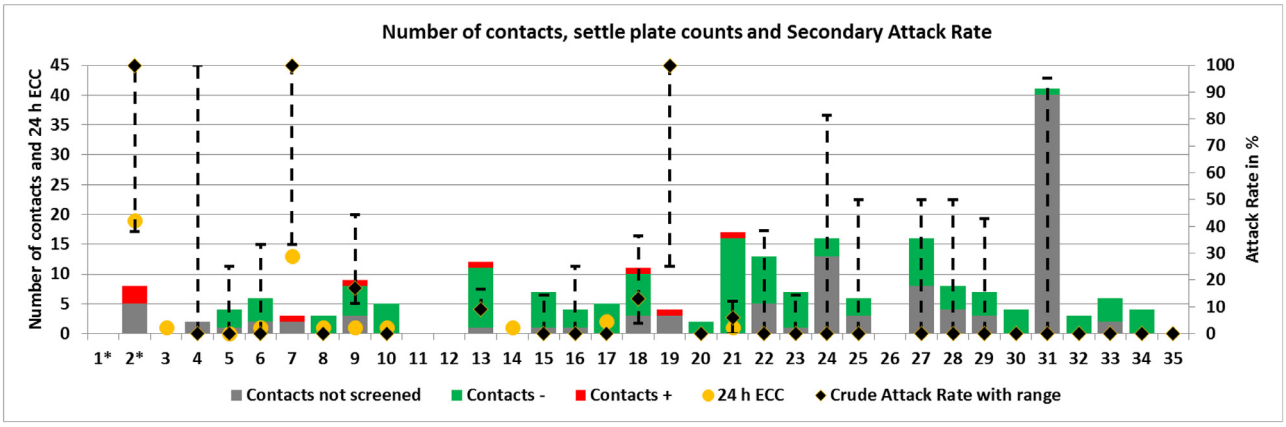

Fig. 3. 24 h settle plate counts, contacts and crude secondary attack rates.
This figure shows *C. auris* case numbers on the x axis, number of contacts on the left y axis and crude secondary attack rates on the right *y* axis. Dotted lines indicate range of attack rates (minimum if none of the unscreened patients would be positive and maximum if all of the unscreened would be positive. Settle plate colony counts were available for 11 patients only.

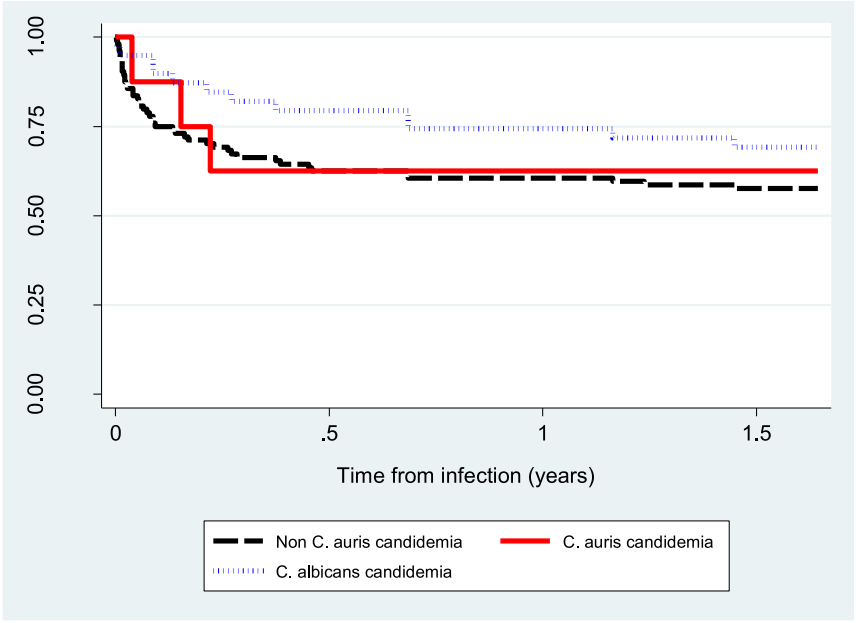

Fig. 4. Kaplan Meier curve showing survival probability of *C. auris* candidemia vs *C. albicans* candidemia vs all non *C. auris* candidemias.

July 2016 to Feb 2017 the compliance improved (range 94.4–98.7%, mean 97%).

Cost analysis: Table 4 lists the costs associated with managing the outbreak. The total is estimated to be £1014848.2 (€1217817.84) for the outbreak period. This did not account for the opportunity cost of cancelled operations, extra consumables in the operating theatres, screening costs before a formal test was introduced in the lab and non IPC staff time outside of formal meetings. In addition, there was a substantial cost associated with isolation and IPC precautions taken for patients who were awaiting confirmation of *C. auris* in the initial part of the outbreak but were ultimately negative. Although bed closure was avoided as much as possible, there were a few occasions when cases could not be isolated, hence adjacent beds were blocked. Indication for bed closure was not available on retrospective analysis and therefore could not directly be attributed to *C. auris*. Keeping these additional costs in mind, the actual total would be in excess of the calculated cost. ALoS contributed to almost half the calculated total, followed by the cost of screening and personnel time spent managing the outbreak. After the outbreak was controlled, management of known cases and the screening protocol was continued. This incurred an additional £58045.4 (€69645.5) per month.

Discussion and conclusion

The lack of early identification and isolation of case ID 2 upon readmission appears to be the initiating factor in this outbreak. No other patient was known to be a healthcare transfer from a hospital with a known outbreak or healthcare tourist from abroad.

The commonest source of *C. auris* BSI were thought to be CVC (5/8 patients; 62.5%) Previous reports have described *C. auris* CLABSI to be between 11.1% and 92.1% [8,15]. The first 3 CLABSI were diagnosed before biopatches became routine by the end of Nov 2016. One colonized patient developed BSI due to inappropriate presence of a PICC line when it was not being used. The final CLABSI was in a patient who had multiple admissions but never in an ITU (case ID 35). Risk factors for development of clinical infection (Table 2) are in keeping with the observations that most BSI

Table 3
MIC values of isolates identified in the outbreak.

	PHE ID**	Site	Date	Minimum inhibitory concentrations in mg/L (MIC interpretation)						
				AMB	FZL	VOR	AFN	FCY	IZL	CFN
2*	–	Pus	07/10/2016	1 (S)	32 (R)	0.5 (I)	–	–	–	1.5(R)
3	33	Blood	28/07/2106	2 (R)	32 (R)	0.125 (S)	–	8 (I)	–	–
4	64	Blood	29/08/2016	1 (S)	>64 (R)	0.5 (I)	0.06 (S)	4 (I)	0.125	–
5	66	Urine	20/10/2016	–	>64 (R)	–	–	>64(R)	–	–
		Swab	20/10/2016	1 (S)	>64 (R)	0.5 (I)	0.25 (S)	32 (R)	–	0.125 (S)
		Swab	01/11/2016	1 (S)	64 (R)	0.25 (I)	0.25 (S)	8 (I)	–	–
		Swab	24/11/2017	1 (S)	>64 (R)	0.5 (I)	0.25 (S)	–	–	–
6	80	Blood	27/09/2016	0.25(S)	64 (R)	0.25 (I)	–	1(S)	–	>32 (R)
		Nose	16/10/2016	1 (S)	16 (R)	0.25 (I)	0.25 (S)	<0.125 (S)	0.25 (S)	
7	81	Blood	03/10/2016	1 (S)	32 (R)	0.125 (S)	0.06 (S)	32 (R)	–	0.25 (S)
		Urine	06/10/2016	–	32 (R)	–	–	32 (R)	–	–
		Groin	15/10/2016	1 (S)	64 (R)	0.5 (I)	0.25 (S)	64 (R)	–	0.125 (S)
		Urine	24/10/2016	2 (R)	64 (R)	0.5 (I)	0.25 (S)	4 (I)	–	0.125 (S)
		Swab	22/06/2017	1 (S)	>64 (R)	0.5 (I)	0.125 (S)	–	–	–
8	91	Tissue	14/10/2016	1 (S)	32 (R)	0.25 (I)	0.25 (S)	<0.125 (S)	0.125 (S)	
9	92	Urine	16/10/2016	1 (S)	32 (R)	0.125 (S)	0.125 (S)	<0.125(S)	–	–
		Blood	28/10/2016	1 (S)	64 (R)	0.125 (S)	0.125 (S)	<0.125(S)	<0.03 (S)	0.5 (I)
10	87	Blood	16/10/2016	1 (S)	32 (R)	0.25 (I)	0.125 (S)	32 (R)	–	–
		Swab	18/10/2016	1 (S)	64 (R)	0. 5 (I)	0.25 (S)	16 (I)	–	0.125 (S)
		Blood	21/10/2016	1 (S)	64 (R)	0.5 (I)	0.125 (S)	32(R)	–	0.25 (S)
		Urine	09/11/2016	–	>64 (R)	0.5 (I)	0. 5 (I)	–	–	–
		Blood	18/11/2016	2 (R)	>64 (R)	0.25 (I)	0. 125 (S)	–	–	–
11	97	Swab	17/10/2016	1 (S)	64 (R)	0. 5 (I)	0.125 (S)	16 (I)	–	0.25 (S)
12	93	Urine	17/10/2016	–	>64 (R)	–	–	32 (R)	–	–
		Urine	20/10/2016	–	>64 (R)	–	–	32 (R)	–	–
13	88	Swab	19/10/2016	1 (S)	32 (R)	0.5 (I)	0.25 (S)	–	–	–
14	110	Urine	21/10/2016	2 (R)	>64 (R)	0.25 (I)	<0.015 (S)	<0.125 (S)	–	–
15	98	Urine	24/10/2016	2 (R)	>64 (R)	0.25 (I)	<0.015(S)	4 (I)	–	–
16	101	Swab	25/10/2016	2 (R)	16 (R)	0.125 (S)	0.25 (S)	<0.125 (S)	–	–
17	99	Swab	02/11/2016	2 (R)	32 (R)	0.125 (S)	0.5 (I)	0.25 (S)	–	–
18	113	Swab	02/11/2016	2 (R)	64 (R)	0.25 (I)	0.125 (S)	8 (I)	–	–
19	121	Swab	04/11/2016	2 (R)	32 (R)	0.25 (I)	1.0 (R)	–	–	–
20	119	Swab	06/11/2016	2 (R)	>64 (R)	1.0 (R)	0.25 (S)	–	–	–
21	120	Swab	12/11/2016	2 (R)	>64 (R)	1.0 (R)	0.5 (I)	–	–	–
22	116	Swab	11/11/2016	2 (R)	64 (R)	0.5 (I)	–	–	–	–
23	148	Swab	21/12/2016	1 (S)	8 (R)	0.125 (S)	0.5 (I)	–	–	–
		Urine	22/12/2016	–	8 (R)	–	–	<0.125 (S)	–	–
24	150	Urine	28/12/2016	–	32 (R)	–	–	16 (I)	–	–
		Swab	05/05/2017	1 (S)	32 (R)	0.25 (I)	0.25 (S)	–	–	–
		Urine	01/06/2017	1 (S)	>64 (R)	0.25 (I)	0.125 (S)	–	–	–
25	158	Urine	09/01/2017	1 (S)	>64 (R)	–	0.125 (S)	8 (I)	–	–
26	160	Swab	17/01/2017	1 (S)	16 (R)	0.06 (S)	0.03 (S)	–	–	–
27*	183	Swab	17/03/2017	1 (S)	64 (R)	0.25 (I)	–	8 (I)	–	–
28*	187	Tissue	28/03/2017	2 (R)	32 (R)	0.12 (S)	–	1 (S)	–	–
29*	186	Bone	30/03/2017	8 (R)	32 (R)	0.12 (S)	–	1 (S)	–	–
30	188	Sputum	07/04/2017	–	–	–	–	–	–	–
31	191	Swab	05/04/2017	1 (S)	16 (R)	0.25 (I)	0.06 (S)	–	–	–
		Blood	08/08/2017	1 (S)	64 (R)	0.5(I)	0.125 (S)	–	0.06 (S)	–
32	192	Swab	20/05/2017	1 (S)	32 (R)	0.125 (S)	0.06 (S)	–	–	–
33	194	Tissue	05/06/2017	0.5 (S)	32 (R)	0.125 (S)	0.125 (S)	–	–	–
34	201	Urine	20/09/2017	1 (S)	>64 (R)	>16 (R)	0.25 (S)	8 (I)	–	1.0 (R)
35	205	Swab	1 0/11/2017	1 (S)	>64 (R)	0.5(I)	0.125 (S)	–	–	–
		Blood	11/11/2017	1 (S)	64 (R)	0.25 (I)	0.06 (S)	–	–	–
		Blood	12/11/2017	1 (S)	64 (R)	0.25 (I)	0.06 (S)	–	–	–
36	207	Swab	06/01/2018	1 (S)	32 (R)	0.5(I)	0.25 (S)	–	–	–

Interpretation of MIC values *R* = resistant, *I* = intermediate/susceptible (dose dependent), *S* = susceptible.
AFN = Anidulafungin; NYS = Nystatin; FZL = Fluconazole; AMB = Amphotericin B; FCY = Flucytosine; VOR = Voriconazole; CFN = Caspofungin; ICZ = Itraconazole.
Swab refers to screening sample.
* Tested by Vitek 2 only.
** Reference to isolates depicted in phylogenetic analysis Fig. 5.

were CVC related. Similar to a previous study from India [7] prior antifungal therapy corelated strongly with development of infection even though most patients received anidulafungin to which the isolates were largely sensitive. Some patients did receive fluconazole and would possibly have influenced our findings. Mortality and age were not different between the two groups as has been previously observed in a different study [15]. Unfortunately, due to the low number of patients, multivariable analysis could not be done.

We followed a strict isolation and cohorting policy which has been previously recommended [8,16]. Published literature suggests that hospital surfaces can be contaminated as a result of exposure to colonized patients [5]. The settle plate results in our study suggest there is dispersal of *C. auris* in the patient's vicinity not just at high touch point areas but in corners of the room such as windowsills. We postulate that this risk may increase during high turbulence activities. We also observed two patients (Fig. 3; cases ID 2 and 7) had a high ECC and high CSAR. Although numbers stud-

Table 4
Cost breakdown of measures taken to manage the outbreak during and after active transmission.

Item		Cost incurred during outbreak (July 2016–Feb 2017)	Cost per month of continuing beyond outbreak calculated during the period March 2017–Feb 2018	
Additional length of stay		525,760	N/A	Actual
Screening*	Cost of tests	260,780	49,300	Actual
	Time to collect screening	9204	1740	Opportunity
Staff costs	MDT meetings	6279.97		Opportunity
	IPCN	34,145		Opportunity
	Surveillance and data collection	23,040	619	Opportunity
	IPC doctor time	26,250	71.83	Opportunity
	Microbiology junior doctor time	13,477	39.34	Opportunity
Antifungal treatment		74349.38	2598.67	Actual
Cleaning**	Hydrogen peroxide fogging	5876	644.10	Opportunity
Consumables	Biopatches	16,660	2380	Actual
	Disposable BP cuffs	89.10	8.57	Actual
	Disposable pillows	207.48	22.61	Actual
	Full sleeve plastic gowns	14509.92	920.56	Actual
	Gloves	3627.48	230.14	Actual
	Disposable curtains	821.95	89.57	Actual
Total		£1014848.20	58045.39	
		€1217817.84	€69654.47	

* *Lower limit as actual screeing started before test codes were changed.*
** Cost of deep cleaning and enhanced cleaning schedule N/A as Trust has a block contract.
Total actual = £896,805 (€ 1,076,166).
Total opportunity=£118,272 (€141926.4) MDT: multi-disciplinary team meeting.

ied are low and there are limitations inherent in the methodology, this data suggests there may be a variation in organism dispersal amongst patients and possibly a link between environmental shedding and secondary attack rates with the limitation that some transmission events could not be explained by close contact alone. More work is needed to substantiate this hypothesis and determine risk factors for high dispersal. Other studies have detected *C. auris* from air sampling and screens from healthcare workers though their significance in transmission is not yet established [5,8]. We did not screen HCWs as the outbreak was controlled fairly rapidly without this intervention. Hence, we recommend early identification, strict contact precautions and high priority for isolation when managing *C. auris* patients. Heightened awareness as a result of hospital wide communication, enhancement of preventive strategies and early detection of infection could have prevented further transmission. In vitro evidence for the efficacy of chlorhexidine wipes in reducing *C. auris*colonization is limited; however, it has been shown to be effective when used as part of an integrated outbreak control program [8]. Twenty-six out of 36 (72.2%) patients can be linked to other patients (through timeline), which suggests that the presence of colonized patients is a major factor in transmission. Although we did not extensively sample common use equipment for *C. auris*, the outbreak was brought under control by introducing single use equipment wherever possible and decontamination where single use was not an option.

In this study, mortality associated with *C. auris* BSI was not higher than that of other prevalent yeasts. This should be interpreted alongside our all cause 30d mortality rate (12.5%) for BSI which is lower than other studies (range 17–60%) [15–19]. Resource poor settings have reported differences in the *C. auris* lineage involved, and antifungal availability and prescribing practices may account for this difference. Limitations include low numbers of BSI cases and limited data on the controls which did not allow us to control or describe differences for other potential confounders on all-cause mortality comparisons. Death registries would not identify patients who died abroad.

Antifungal susceptibility of the isolates from this outbreak was similar to that observed in a previous UK outbreak of the same strain with anidulafungin the most reliable empiric agent [8]. The variable susceptibility to amphotericin is of particular concern and has been observed in previous outbreaks of the South Asian strain and otherwise [7,20]. The mechanism for this variation is not full but hypotheses include biofilm tolerance [21]

The first two British outbreaks have reported 50 cases (9 *C. auris* BSI) over 16 months [8] and 70 cases over 2.5 years [19], respectively. A recent Spanish outbreak reported transmission over more than 10 months with greater than 140 affected cases including 41 BSI [16]. Other outbreaks have used different methodology making direct comparison difficult. In the present report, outbreak duration of 7 months appears to be shorter, with fewer affected cases than other reports even when new cases seen over the next year are accounted for. This was made possible by aggressive implementation of all available interventions which did have associated cost implications. When compared to the overall reported costs of recent MDR organism outbreaks in Europe (€1.1m over 10 months for an outbreak of CPE [22] and €1,222,376 for an outbreak of *Clostridium difficile* infections over 12 months [23] the cost was broadly comparable. Additional length of stay was the single largest cost but reasons for this could not be evaluated in the present study. Compared to other reports, the cost of screening amounted to a substantial proportion, largely because new infrastructure and personnel were needed to introduce the test in the laboratory where it was performed. Screening was also the largest ongoing cost after the outbreak was declared over.

Recent studies demonstrated that isolates corresponding to three different clonal lineages (S. Asian, S. African and Japanese/Korean) have been introduced into the UK [24]. Isolates from the current outbreak cluster closely with other strains detected in South Asia, making this the second reported outbreak in the UK caused by the South Asian clade (Fig. 5). It is possible that our outbreak was sustained by fresh introductions of *C. auris*: at least one patient presented with a deep infection with the organism directly on admission.

In the one year follow up period, new cases continued to be detected sporadically at low frequency in different clinical areas (see Fig. 1B) but were mostly linked to ward contact with known positive patients. Case ID 36 did not have a clear link to any known patient, was admitted in two wards with previous cases but could also represent a novel introduction. However, due to the extremely clonal nature of the S. Asian clade isolates [13] we were not able

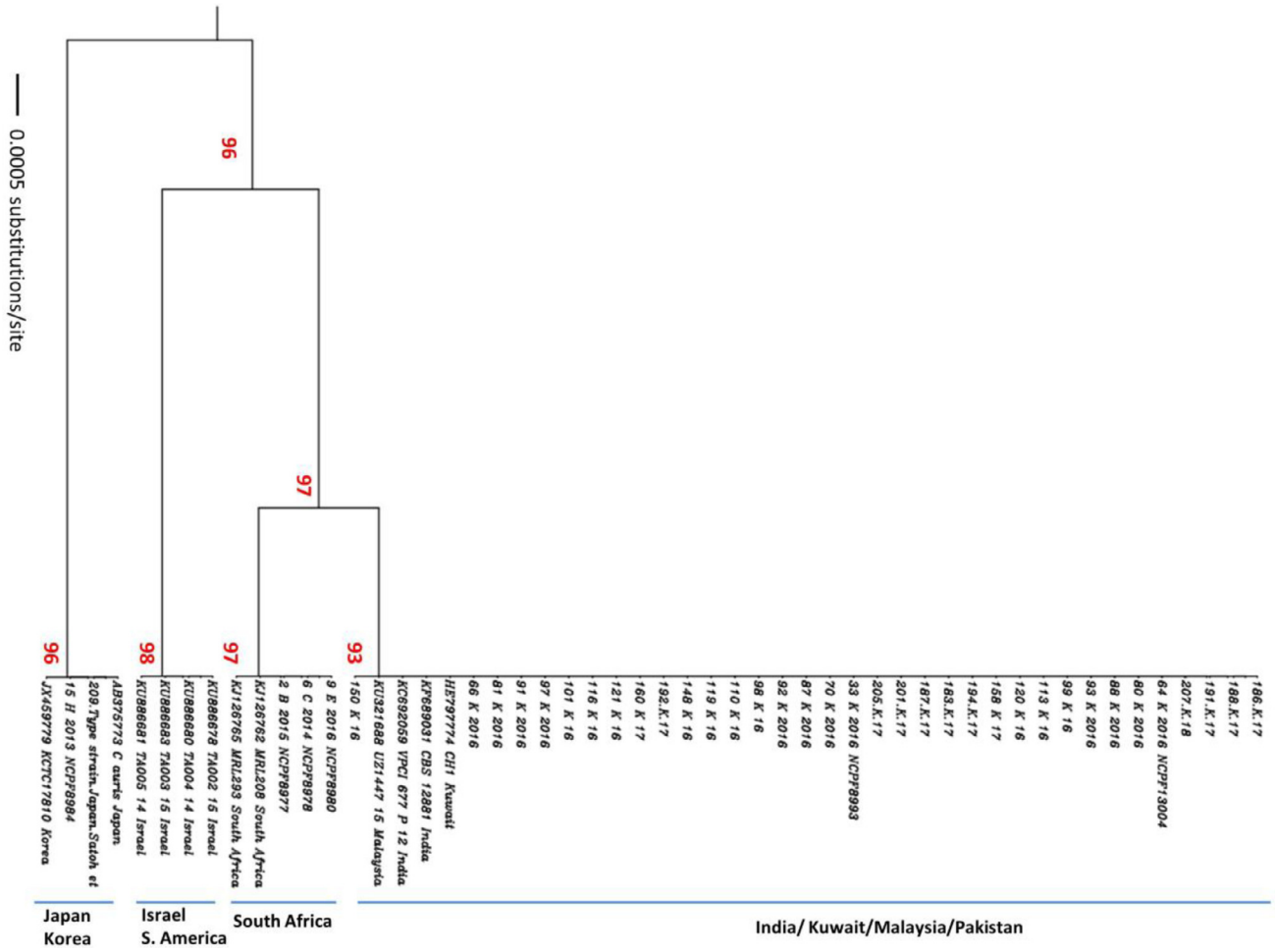

Fig. 5. Phylogenetic tree corresponding to the concatenated D1/D2 and ITS1 datasets of reference *C. auris* isolates from the 4 clonal lineages and strains from the current outbreak (red arrows). Trees were constructed using the UPGMA method, with bootstrap values above 90% shown below branches.
Refer Table 2 for case IDs.

to formally prove this hypothesis by conventional typing methodologies. Whole genome sequencing of the isolates is underway to determine the genetic relatedness of the isolates.

Some patients could have been potentially detected earlier if active screening was practiced in the wards they were initially admitted to. After the outbreak was declared over, screening was continued to provide reassurance on transmission control and enable rapid isolation and control measures for any new cases. Hospitals without an outbreak in a low prevalence area may wish to consider passive surveillance only. Recent reports describing clonally identical BSI over 17 months (Venezuela) [17], a prevalence of 5.3% amongst all fungal BSI from intensive care units in India [7] and other transmission events across the world [5,13,25] suggest prevalence maybe high in certain parts of the world and is likely to increase.

In conclusion, controlling an outbreak of *C. auris* requires a multipronged approach which can be expensive over the short term. Implementation of hospital wide communication, early detection by active screening, mandatory isolation and aggressive disinfection at an early stage may help reduce transmission and morbid complications of the disease.

Declaration of Competing Interest

All authors report no conflicts of interest pertaining to this article.

CRediT authorship contribution statement

Surabhi K. Taori: Conceptualization, Data curation, Formal analysis, Investigation, Project administration, Writing - original draft. **Kirstin Khonyongwa:** Data curation, Formal analysis, Investigation, Writing - original draft. **Iain Hayden:** Data curation, Formal analysis. **GID Dushyanthie AD Athukorala:** Data curation, Project administration, Writing - review & editing. **Andrew Letters:** Data curation, Writing - review & editing. **Amanda Fife:** Writing - review & editing. **Nergish Desai:** Data curation, Formal analysis, Project administration, Writing - original draft. **Andrew M. Borman:** Data curation, Formal analysis, Writing - review & editing.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethics

Formal ethics committee approval was not required for this report due to the nature of the article.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jinf.2019.09.007.

References

1. Oh BJ, Oh BJ, Shin JH, Kim MN, Sung H, Lee K, Joo MY, et al. Biofilm formation and genotyping of Candida haemulonii, Candida *pseudohaemulonii*, and a proposed new species (Candida auris) isolates from Korea. *Med Mycol* 2011;**49**(1):98–102.
2. Satoh K, Makimura K, Hasumi Y, Nishiyama Y, Uchida K, Yamaguchi H. Candida auris sp. nov., a novel ascomycetous yeast isolated from the external ear canal of an inpatient in a Japanese hospital. *Microbiol Immunol* 2009;**53**(1):41–4.
3. PHE. *Guidance for the laboratory investigation, management and infection prevention and control for cases of Candida auris*. England PH, editor. London, UK: Public Health England; 2017.
4. Chowdhary A, Sharma C, Meis JF. Candida auris: a rapidly emerging cause of hospital-acquired multidrug-resistant fungal infections globally. *PLoS Pathog* 2017;**13**(5):e1006290.
5. Biswal M, Rudramurthy SM, Jain N, Shamanth AS, Sharma D, Jain K, et al. Controlling a possible outbreak of Candida auris infection: lessons learnt from multiple interventions. *J Hosp Infect* 2017;**97**(4):363–70.
6. Al-Siyabi T, Al Busaidi I, Balkhair A, Al-Muharrmi Z, Al-Salti M, Al'Adawi B. First report of Candida auris in Oman: clinical and microbiological description of five candidemia cases. *J Infect* 2017;**75**(4):373–6.
7. Rudramurthy SM, Chakrabarti A, Paul RA, Sood P, Kaur H, Capoor MR, et al. Candida auris candidaemia in Indian ICUs: analysis of risk factors. *J Antimicrob Chemother* 2017;**72**(6):1794–801.
8. Schelenz S, Hagen F, Rhodes JL, Abdolrasouli A, Chowdhary A, Hall A, et al. First hospital outbreak of the globally emerging Candida auris in a European hospital. *Antimicrob Resist Infect Control* 2016;**5**:35.
9. Mizusawa M, Miller M, Green R, Lee R, Durante M, Perkins R, et al. Can multidrug-resistant Candida auris be reliably identified in clinical microbiology laboratories? *J Clin Microbiol* 2017;**55**(2):638–40.
10. CDC. *National Healthcare Safety Network (NHSN) HAI checklists*; 2019. Available from: *https://www.cdc.gov/nhsn/hai-checklists/index.html*.
11. Szekely A, Borman AM, Johnson EM. Candida auris isolates of the Southern Asian and South African lineages exhibit different phenotypic and antifungal susceptibility profiles in vitro. *J Clin Microbiol* 2019;**57**(5):e02055–18.
12. PHE. *Guidance for the laboratory investigation, management and infection prevention and control for cases of* Candida auris. England PH, editor. London: PHE Publications Gateway; 2016.
13. Magobo RE, Corcoran C, Seetharam S, Govender NP. *Candida auris*-associated candidemia, South Africa. *Emerg Infect Dis* 2014;**20**(7):1250–1.
14. Chowdhary A, Kumar A, Sharma C, Prakash A, Agarwal K, Babu R, et al. Multidrug-resistant endemic clonal strain of Candida auris in India. *Eur J Clin Microbiol Infect Dis* 2014;**33**(6):919–26.
15. Sayeed MA, Farooqi J, Jabeen K, Awan S, Mahmood SF. Clinical spectrum and factors impacting outcome of Candida auris: a single center study from Pakistan. *BMC Infect Dis* 2019;**19**(1):384.
16. Ruiz-Gaitan A, Moret AM, Tasias-Pitarch M, Aleixandre-Lopez AI, Martinez–Morel H, Calabuig E, et al. An outbreak due to Candida auris with prolonged colonisation and candidaemia in a tertiary care European hospital. *Mycoses* 2018;**61**(7):498–505.
17. Calvo B, Melo AS, Perozo-Mena A, Hernandez M, Francisco EC, Hagen F, et al. First report of Candida auris in America: clinical and microbiological aspects of 18 episodes of candidemia. *J Infect* 2016;**73**(4):369–74.
18. Lee WG, JH Shin, Uh Y, Kang MG, Kim SH, Park KH, et al. First three reported cases of nosocomial fungemia caused by Candida auris. *J Clin Microbiol* 2011;**49**(9):3139–42.
19. Eyre DW, Sheppard AE, Madder H, Moir I, Moroney R, Quan TP, et al. A Candida auris outbreak and its control in an intensive care setting. *N Engl J Med* 2018;**379**(14):1322–31.
20. Escandon P, Chow NA, Caceres DH, Gade L, Berkow EL, Armstrong P, et al. Molecular epidemiology of Candida auris in Colombia reveals a highly related, countrywide colonization with regional patterns in amphotericin B resistance. *Clin Infect Dis* 2019;**68**(1):15–21.
21. Kean R, Ramage G. Combined antifungal resistance and biofilm tolerance: the global threat of Candida auris. *mSphere* 2019;**4**(4):e00458–19.
22. Otter JA, Burgess P, Davies F, Mookerjee S, Singleton J, Gilchrist M, et al. Counting the cost of an outbreak of carbapenemase-producing enterobacteriaceae: an economic evaluation from a hospital perspective. *Clin Microbiol Infect* 2017;**23**(3):188–96.
23. van Beurden YH, Bomers MK, van der Werff SD, Pompe E, Spiering S, Vandenbroucke-Grauls C, et al. Cost analysis of an outbreak of clostridium difficile infection ribotype 027 in a Dutch tertiary care centre. *J Hosp Infect* 2017;**95**(4):421–5.
24. Borman AM, Szekely A, Johnson EM. Isolates of the emerging pathogen Candida auris present in the UK have several geographic origins. *Med Mycol* 2017;**55**(5):563–7.
25. Belkin A, Gazit Z, Keller N, Ben-Ami R, Wieder-Finesod A, Novikov A, et al. Candida auris infection leading to nosocomial transmission, Israel, 2017. *Emerg Infect Dis* 2018;**24**(4):801–4.