CASE REPORT

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Outbreak of *Candida lusitaniae* bloodstream infection in neonatal intensive care unit: case series

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ABSTRACT

Introduction: Neonatal sepsis remains a significant health challenge, particularly in low- and middle-income countries (LMICs), where inadequate healthcare infrastructure and sanitation contribute to its high incidence. Sepsis is defined as life-threatening organ dysfunction resulting from a dysregulated immune response to infection. Neonates are particularly vulnerable due to immature immune systems. Candida species, especially *Candida albicans* and *Candida parapsilosis*, are common pathogens, but *Candida lusitaniae* has also emerged as a notable cause of neonatal sepsis, with increased resistance to Amphotericin B. This case series highlights an outbreak of *Candida lusitaniae* bloodstream infections in a neonatal intensive care unit (NICU), emphasizing the diagnostic and management challenges of rare fungal infections.

Case Description: In this report, we describe five neonatal sepsis cases caused by *Candida lusitaniae* in our NICU. All patients exhibited respiratory distress, pneumonia, and elevated procalcitonin levels, and were treated with Voriconazole. The neonates had varying risk factors, including prematurity, low birth weight, and asphyxia. Blood cultures identified *Candida lusitaniae* in all cases, with susceptibility testing confirming sensitivity to Voriconazole, Amphotericin B, and Flucytosine. Environmental sampling revealed contamination in the NICU, specifically from a sink, suggesting nosocomial transmission. In response, the NICU was decontaminated, infection control protocols were reinforced, and environmental surveillance was intensified. All patients improved clinically and were discharged without major complications, except for mild encephalopathy in one case.

Conclusion: This outbreak underlines the importance of timely identification and appropriate therapy in managing rare fungal infections in neonates. It also highlights the critical role of environmental sources in nosocomial infections and the need for robust infection control measures in NICUs. Strict infection prevention strategies, including enhanced cleaning, staff training, and surveillance, are essential to preventing such outbreaks in high-risk settings.

Keywords: *Candida lusitaniae*, neonatal sepsis, NICU, outbreak, voriconazole
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INTRODUCTION

Neonatal sepsis remains a major challenge in clinical practice, particularly in low- and middle-income countries (LMICs), where factors such as poor sanitation and inadequate healthcare facilities exacerbate the problem.[1] According to the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3), sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.[2] Neonates are particularly vulnerable due to their immature immune and organ systems, which impair their ability to respond effectively to infections.[3] Fleischmann-Struzek et al. reported that the incidence of sepsis is highest in neonates, with a peak at 516 per 100,000 population, significantly higher than in older children (89 per 100,000 population).[4]

In neonatal sepsis, *Candida* species are among the most common fungal pathogens, with *C. albicans* and *C. parapsilosis* being the predominant causes.[5] However, *Candida lusitaniae*, a rare pathogen, has emerged as a significant cause of sepsis, often associated with resistance to Amphotericin B.[6,7] Unlike *C. albicans*, *C. lusitaniae* does not form true hyphae but produces pseudohyphae, a characteristic linked to its antifungal resistance.[6] Sepsis caused by multidrug-resistant organisms (MDROs), including *C. lusitaniae*, is associated with higher mortality rates in neonates compared to non-MDRO infections.[8,9]

In this article, we present an outbreak of bloodstream infections caused by *C. lusitaniae* in five neonates in our NICU. The cases are described in chronological order, emphasizing the clinical presentations, diagnostic approaches, management strategies, and outcomes. This report highlights the challenges of diagnosing and treating rare fungal infections in a high-risk neonatal population and underscores the importance of timely identification and appropriate therapy, alongside robust infection control measures to prevent nosocomial outbreaks.

CASE DESCRIPTION

Our cases are summarized in **Table 1**. Below, we provide detailed descriptions of each case. These cases occurred within a short time frame, suggesting a possible outbreak of *Candida lusitaniae* sepsis in the NICU of our hospital. A timeline illustrating the admission dates, clinical events, and isolate growth findings for all patients is shown in **Figure 1**.

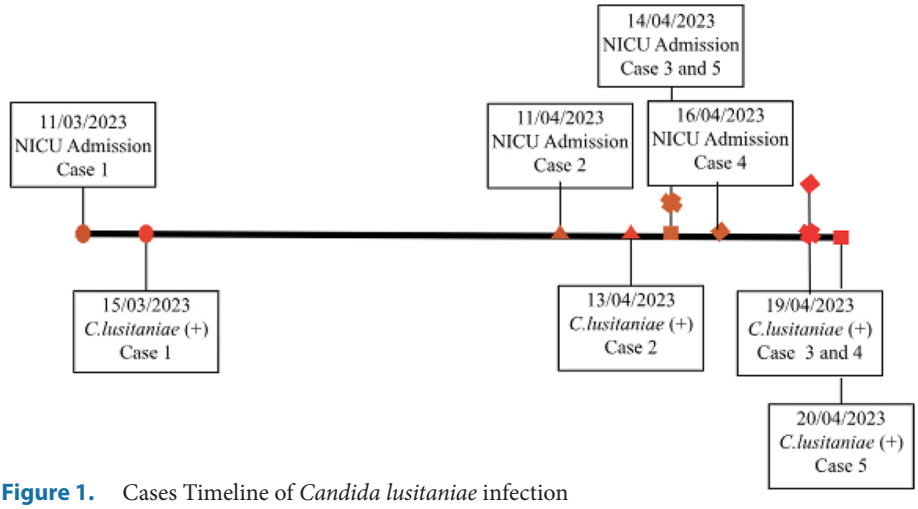

Figure 1. Cases Timeline of *Candida lusitaniae* infection

Case 1

The first patient was a male preterm neonate born at 29–30 weeks of gestation via caesarean section due to severe pre-eclampsia. He weighed 1680 grams and presented with low birth weight and severe asphyxia (APGAR score 1-1-3). Upon admission to the Level III NICU, he experienced apnea, flaccidity, and pale-bluish skin. Laboratory findings revealed anemia (hemoglobin 7.55 g/dL), thrombocytopenia (platelet count 147 × 10^3/µL), and elevated procalcitonin levels (80.30 ng/mL). Imaging showed pneumonia and Grade I necrotizing enterocolitis (NEC). On the 5th day of admission to NICU, blood cultures identified *Candida lusitaniae*. The isolate was sensitive to Voriconazole, Amphotericin B, and Flucytosine. The patient received Voriconazole for 21 days and did not receive any concurrent antibiotic therapy during the fungal infection episode. He improved clinically and was discharged, but follow-up revealed mild diffuse encephalopathy on EEG.

Case 2

The second patient was a male late-preterm neonate born at 36 weeks of gestation via cesarean section due to maternal Bartholin abscess and ruptured vaginal varices requiring general anesthesia. He weighed 2400 grams and was admitted on the 1st day of life to the Level III NICU with respiratory distress, frequent desaturation, cyanosis, and rhonchi on both lungs. His laboratory results showed leukopenia (white blood cell count 3.4 × 10^3/µL) and significantly elevated procalcitonin levels (48.14 ng/mL). Imaging revealed pneumonia. The patient received Meropenem for 9 days based on local pneumonia protocol after sputum cultures isolated *Enterobacter cloacae*. Subsequently, on the 3rd day of admission, blood cultures revealed *Candida lusitaniae*. Voriconazole was initiated and continued for 21 days. The patient showed significant clinical improvement and was discharged in good condition. This case was unique within our cohort, representing the only instance of a confirmed mixed infection involving both *Candida lusitaniae* and *Enterobacter cloacae*. The co-infection highlights the potential for concurrent fungal and bacterial pathogens in neonatal sepsis, underscoring the importance of comprehensive microbiological workups in critically ill neonates.

Case 3

The third patient was a male term neonate born at 37 weeks of gestation via cesarean section following premature rupture of membranes. He weighed 3200 grams and was admitted to the Level III NICU on the 1st day of life with respiratory distress and increased work of breathing. Laboratory findings included leukopenia (white blood cell count 2.6 × 10^3/µL) and elevated procalcitonin levels (7.78 ng/mL). Imaging revealed pneumonia, and no brain edema was observed on head ultrasound. On the 6th day of admission to NICU, blood cultures identified *Candida lusitaniae*. The patient was treated with Voriconazole for 14 days. He did not receive concurrent antibiotic therapy at the time of positive fungal culture. The patient recovered without complications and was discharged in good condition.

Case 4

The forth patient was a male term neonate born at 40 weeks of gestation after prolonged labor, oligohydramnios, and maternal anemia. He weighed 3000 grams and was admitted on the 1st day of life to the Level II NICU with moderate asphyxia (APGAR score 4-5-6-7). On the 4th day of life, he developed abdominal distension and passed black-colored stool. Laboratory findings showed anemia (hemoglobin 16.5 g/dL), elevated procalcitonin levels (49.43 ng/mL), and imaging suggestive of pneumonia On the 4th day of admission to NICU, blood cultures identified *Candida lusitaniae*. The patient was treated exclusively with Voriconazole for 14 days and showed clinical improvement. He was discharged in good condition.

Case 5

The fifth patient was a female term neonate born at 37–38 weeks of gestation via cesarean section due to placenta previa. She weighed 2150 grams and presented with low birth weight and mild asphyxia. She was admitted to the NICU on the 3rd day of life with abdominal distension and black-colored stool. Laboratory results showed anemia (hemoglobin 7.8 g/dL), thrombocytopenia (platelet count 269 × 10^3/µL), and markedly elevated procalcitonin levels (141.42 ng/mL). Imaging showed pneumonia. On the 6th day of admission to NICU, blood cultures identified *Candida lusitaniae*. The patient received only Voriconazole for 14 days without any concurrent antibiotic therapy and showed significant clinical improvement, leading to discharge.

Table 1. **Neonatal sepsis caused by *Candida lusitaniae* case summaries**

	Case 1	Case 2	Case 3	Case 4	Case 5
History					
Maternal Diagnosis	G2P1001; GA 29-30 weeks; severe pre-eclampsia	G2P1001; GA 36 weeks; Bartholin abscess; vaginal varices rupture	G2P1001; GA 37 weeks; PROM	G1P0000; GA 40 weeks; prolonged labour; oligohydramnios; anemia	G4P2012; GA 37-38 weeks; placenta previa
Delivery Mode and Location	Sectio caesarean; hospital-outborn	Sectio caesarean; hospital-inborn	Sectio caesarean; hospital-outborn	Spontaneous delivery; hospital-outborn	Sectio caesarean; hospital-outborn
Sex	Male	Male	Male	Male	Female
Neonate Risks Factor	Prematurity (GA 29-30 weeks; low-birth weight (1680 grams);	Prematurity (GA 36 weeks); fetal distress; history of general anaesthesia during delivery	No risk factor	No risk factor	Low-birth weight (2150 grams)
Present Illness					
Age	4 days	1 day	1 day	1 day	3 days
Clinical Presentation	Admitted to Level III NICU due to frequent apnoea; marked respiratory distress; AS Score 5-6-8	Admitted to Level III NICU due to apnoea after delivery; flaccid; pale-bluish skin; AS Score 1-1-3;	Admitted to Level III NICU due to respiratory distress; frequent desaturation; cyanosis; rhonchi observed at both lungs; AS score 5-6-7	Admitted to Level III NICU due to respiratory distress with greenish amniotic fluid; increased work of breathing and retraction observed; AS Score 7-8-8	Admitted to Level II NICU with the history of born with moderate asphyxia (AS score 4-5-6-7); distended abdomen on DOL 4 with black stool
Laboratory Findings (Initial Diagnostic Laboratory Workup)					
WBC (10^3/µL)	7.55	3.4	2.6	18.34	11.32
HGB (g/dL)	15.2	12.2	13.4	16.5	7.8
HCT (%)	43.7	34.3	36.6	44.4	20.5
PLT (10^3/µL)	147	211	276	154	269
Procalcitonin (ng/mL)	80.30	48.14	7.78	49.43	141.42
Imaging	Babygram revealed pneumonia and Grade I NEC	Chest X-ray revealed pneumonia	Babygram revealed pneumonia; head USG indicates no brain oedema observed	Babygram suggesting pneumonia	Babygram revealed pneumonia
Blood Cultures and Susceptibility	*Candida lusitaniae* isolated on DOL 8; sensitive Voriconazole, Amphotericin B, Flucytosine	• *Candida lusitaniae* isolated on DOL 3; sensitive Voriconazole, Amphotericin B, Flucytosine • *Enterobacter cloacae* (Sputum culture from ETT)	*Candida lusitaniae* isolated on DOL 8 (3rd blood culture); sensitive Voriconazole, Amphotericin B, Flucytosine	*Candida lusitaniae* isolated on DOL 3; sensitive Voriconazole, Amphotericin B, Flucytosine	*Candida lusitaniae* isolated on DOL 9 (2nd blood culture); sensitive Voriconazole, Amphotericin B, Flucytosine

	Case 1	Case 2	Case 3	Case 4	Case 5
Parenteral Nutrition; External Devices	Yes; PICC	Yes; PICC and ETT	Yes; PICC and ETT	Yes; IV Line	Yes; IV Line
Antibiotics Usage	Ampicillin, Gentamicin (2 days); Cefoperazone Sulbactam, Amikacin (3 days); Meropenem (9 days)	Ampicillin, Gentamicin (3 days); Cefoperazone Sulbactam, Amikacin (3 days); Meropenem (9 days)	Ampicillin, Gentamicin (6 days); Cefoperazone Sulbactam, Amikacin (4 days)	Ampicillin, Gentamicin (4 days); Cefoperazone Sulbactam, Amikacin (2 days)	Ampicillin, Gentamicin (4 days); Cefoperazone Sulbactam, Amikacin (4 days); Metronidazole (5 days)
Antifungal Therapy	Voriconazole (21 days)	Voriconazole (21 days)	Voriconazole (14 days)	Voriconazole (14 days)	Voriconazole (14 days)
Laboratory Findings (Final Evaluative Laboratory Workup After Antifungal Therapy)					
WBC (10^3/µL)	8.27	16.09	22.65	15.39	12.9
HGB (g/dL)	10.6	10.5	13.8	14.6	13.2
HCT (%)	32.2	28.5	32.6	39.5	36.6
PLT (10^3/µL)	114	529	577	473	193
Procalcitonin (ng/mL)	0.33	0.07	0.16	1.51	1.81
Outcome	Clinically improved, discharged	Clinically improved, discharged	Clinically improved, discharged	Clinically improved, discharged	Clinically improved, discharged

Abbreviation: AS, APGAR Score; DOL, day of life; GA, gestational age; NEC, necrotizing enterocolitis

The clustering of these cases in time and the identification of *Candida lusitaniae* in all five neonates strongly suggested a nosocomial outbreak in the NICU. In all five cases, *Candida lusitaniae* was identified between the 3rd and 6th day of hospitalization, indicating acquisition during NICU stay. Antifungal susceptibility testing confirmed that all isolates were sensitive to Voriconazole, Amphotericin B, and Flucytosine. All patients responded well to antifungal treatment and showed clinical and laboratory improvement. Voriconazole was the only antifungal used in all five cases, and it was the sole antimicrobial therapy in four of them. Only one patient (Case 2) had a documented co-infection with *Enterobacter cloacae* and received concurrent antibiotic therapy. Follow-up revealed no complications except for mild diffuse encephalopathy in the first patient.

To further investigate the source of the outbreak, extensive environmental sampling was conducted throughout the neonatal intensive care unit (NICU). Samples were collected from various high-touch and high-risk locations, including medical equipment, incubators, intravenous preparation areas, and handwashing facilities. Notably, *Candida lusitaniae* was isolated from one of the sinks, implicating it as a potential reservoir for the pathogen. This finding was pivotal in elucidating the outbreak's transmission dynamics and underscored the critical role of environmental sources in nosocomial infections.

To contain the outbreak, immediate and comprehensive measures were implemented. The NICU was temporarily closed for ten days to allow for thorough decontamination of the entire unit. During this time, all equipment and high-contact surfaces were meticulously cleaned and disinfected, and the implicated wash basin was decontaminated. Stricter protocols for environmental cleaning and disinfection were enforced, and hand hygiene practices were reinforced through staff training and regular monitoring to ensure compliance. Disposable equipment and sterile materials were prioritized, and the use of central venous catheters (CVCs) was restricted to cases with absolute indications. Additionally, environmental sampling and microbiological surveillance were intensified to detect potential reservoirs or contamination points early. These actions, combined with enhanced awareness among healthcare personnel, successfully halted the outbreak and prevented further cases.

DISCUSSION

Candida lusitaniae is a dimorphic organism capable of producing yeast cells in ovoid, elliptical, or elongated forms, measuring 2–6 × 2–10 µm. Unlike other members of the Candida genus, such as *Candida albicans*, this species exhibits a unique characteristic: it lacks the ability to form true hyphae and instead produces only pseudohyphae. Furthermore, *C. lusitaniae* has been recognized as a pathogen in various infections, particularly in immunocompromised individuals,

Table 2. Report about *Candida lusitaniae* neonatal infection

No	Reference	Age, Gender	Isolate	Disease or Condition	Vascular Access	Antibiotic	Antifungal	Outcome
1	Sanchez and Cooper (1987)[12]	7-day, Male	Blood, Urine, CSF	Prematurity, Low-birth weight	CVC	Ampicillin, Gentamicin	Amphotericin B, Flucytosine	Discharged
2	Yinnon, et al. (1992)[13]	14-day, Male	Blood, Urine, CVC tip	Prematurity, Low-birth weight, PDA, Intracerebral haemorrhage	CVC	Ampicillin, Amikacin	Amphotericin B, Ketoconazole, Flucytosine	Discharged
3	Oleinik, et al. (1993)[14]	8-day, Male	Blood	Prematurity, Low-birth weight	Peripheral Catheter	Ampicillin, Gentamicin	Amphotericin B	Discharged
4	Favel, et al. (2003)[15]	12-day, Male	Blood, Urine, Nephrostomy Catheter	Prematurity, Low-birth weight	CVC	Cefotaxime, Amikacin, Metronidazole, Amikacin, Vancomycin, Ceftazidime	Fluconazole, Amphotericin B	Death
5	Gautam, et al. (2014)[16]	7-day, Male and Female	Blood	Prematurity, Low-birth weight, Twins	PICC	N/A	Fluconazole	Discharged

including those with comorbidities, undergoing long-term antibiotic therapy, diagnosed with malignancies, or receiving chemotherapy or bone marrow transplantation.[6]

This outbreak of *Candida lusitaniae* bloodstream infections in our neonatal intensive care unit (NICU) highlights the complexities associated with managing rare fungal pathogens in high-risk neonates. While *Candida albicans* and *Candida parapsilosis* are more commonly associated with neonatal fungal sepsis, *C. lusitaniae* is an uncommon etiological agent that presents unique diagnostic and therapeutic challenges, particularly in the context of a healthcare-associated outbreak. Data regarding *C. lusitaniae* infection in the neonatal population is limited. We identified five studies on *C. lusitaniae* in neonatal infections and summarized in **Table 2**.

Several risk factors are associated with *C. lusitaniae* infection in neonates. In our cases, the identified risk factors included premature delivery, low birth weight, prolonged antibiotic therapy, and the use of central venous access. Other risk factors reported in the literature include immunocompromised states, malignancies, chemotherapy, use of catheters, and extended antibiotic treatments. Sousa et al. further highlighted low birth weight, the use of broad-spectrum and multiple antibiotics, mechanical ventilation, parenteral nutrition, and central catheters as key risk factors for candidemia in neonates.[10]

Most neonates with candidemia exhibit non-specific clinical manifestations. The most common clinical features include abdominal distention, fever, shock, and edema, along with elevated procalcitonin levels, anemia, thrombocytopenia, and liver dysfunction.[11] Additional symptoms such as feeding intolerance, thermal instability, bradycardia, respiratory distress, apnea, lethargy, and anuria have also been observed in neonates with *C. lusitaniae* infection.[12–16] Furthermore, neonates with low birth weight are more likely to present with severe manifestations due to the immaturity of their immune systems, which complicates the clearance of the fungal infection.[17] In this report, the clinical presentation of affected neonates included sepsis-like symptoms, such as respiratory distress, abdominal distension, and hematological abnormalities, which are consistent with manifestations of invasive Candida infections in neonates. The clustering of cases within a defined time frame raised the suspicion of a nosocomial outbreak, underscoring the critical need for vigilance, prompt investigation, and a multidisciplinary response in such settings.

The management of neonatal candidemia primarily involves three classes of antifungal agents: polyenes (Amphotericin B Deoxycholate [DAMB], Liposomal Amphotericin B [LAMB]), triazoles (Fluconazole, Itraconazole, Voriconazole), and echinocandins (Micafungin, Caspofungin). Although *C. lusitaniae* is generally sensitive to amphotericin B, triazoles, and echinocandins in vitro, it differs from most clinically significant Candida species in its ability to rapidly develop in vivo resistance to amphotericin B after exposure to this agent. This resistance is primarily due to mutations in the ergosterol biosynthesis pathway, which may affect gene expression in this haploid yeast.[18] This resistance pattern occurs more in haploid species, such as *C. lusitaniae, C. auris,* and *C. glabrata,* compared to diploid species such as *C. albicans*.[19]

There are several theories regarding the cause of resistance in *C. lusitaniae,* most of them is caused by mutation in specific genes. Resistance in fluconazole group is believed to be related with mutation causing an overexpression in MFS7 gene, known also as MDR1, a gene that well known causing resistance in *Candida* species.[6] Mutation in FKS gene also plays important role in developing resistance against echinocandins group; by increasing the minimum inhibitory concentration (MIC) to some echinocandins.[20] Resistance to antifungal agents, including Amphotericin B, Itraconazole, Caspofungin, and Fluconazole, has been

reported in several studies, reflecting the challenges in achieving effective treatment in neonates.[21,22] Among the polyenes, LAMB is often preferred over DAMB due to its lower risk of adverse effects, as it is minimally eliminated via the kidneys. However, DAMB remains the preferred option for infections involving the renal or central nervous system (CNS) due to its superior tissue penetration.[23] Echinocandins show comparable efficacy to polyenes and are associated with a lower risk of treatment discontinuation caused by adverse effects.[24,25]

Triazoles, particularly Fluconazole, are widely used as prophylaxis in neonatal candidemia. Studies indicate that Fluconazole significantly reduces Candida colonization and mortality in preterm infants, particularly those weighing ≤ 1,000 grams.[23,26] These findings underscore the importance of selecting antifungal regimens based on clinical presentation, patient characteristics, and pathogen susceptibility to achieve optimal treatment outcomes.

In our study, *Candida lusitaniae* was identified as the causative agent in all cases. This species, first recognized in 1959 as part of the normal gastrointestinal flora of warm-blooded animals and later reported in 1979 as an opportunistic pathogen in humans, has been associated with resistance to Amphotericin B in some cases.[7,11] Notably, all isolates from our cases demonstrated susceptibility to Voriconazole, Amphotericin B, and Flucytosine. The successful treatment of these neonates with Voriconazole for 14–21 days led to complete clinical and microbiological resolution. This is particularly significant as it highlights the efficacy of Voriconazole, which has shown good tissue penetration and a favourable pharmacokinetic profile, making it a viable option for treating *C. lusitaniae* infections in neonates.

The variability in antifungal susceptibility patterns among *C. lusitaniae* isolates further underscores the necessity for species-level identification and routine antifungal susceptibility testing in clinical laboratories to guide effective treatment decisions. Our findings suggest that Voriconazole may be a reliable treatment option for *C. lusitaniae*-induced neonatal candidemia, particularly in cases where resistance to other antifungal agents is a concern.

The source of the outbreak was identified as contamination of the sink within the NICU, likely due to nosocomial transmission from this environmental reservoir, contaminated medical equipment, or healthcare personnel. Additional risk factors such as prolonged hospitalization, invasive medical procedures, and the use of broad-spectrum antibiotics may have increased the neonates' susceptibility to fungal infections. This finding aligns with existing literature, which emphasizes the role of environmental factors and the importance of maintaining rigorous infection control practices in preventing outbreaks in hospital settings.

To prevent future incidents, recommended strategies include strict hand hygiene protocols, proper cleaning and sterilization of medical equipment, and minimizing the use of invasive devices. Proactive microbiological surveillance, including routine environmental sampling and colonization screening, is also crucial. The identification of *C. lusitaniae* in the sink highlights the importance of maintaining handwashing facilities, which, while essential for preventing cross-contamination, can inadvertently become reservoirs for pathogens if not properly cleaned. These corrective actions, along with reinforced staff hygiene practices, were essential in halting the outbreak and protecting vulnerable neonates. Despite the successful resolution of the outbreak, the occurrence of mild diffuse encephalopathy in one patient underscores the potential for long-term sequelae in neonates recovering from invasive fungal infections. This finding highlights the importance of long-term neurodevelopmental follow-up in affected patients to assess and address any residual complications.

This single-center case series have several limitations. It is descriptive and underpowered; denominators and pre/post surveillance windows were limited, constraining incidence comparisons and assessment of sustained impact. Case ascertainment may be incomplete (small neonatal blood volumes, variable number of culture sets); colonization screening (patients/HCWs) and environmental sampling were limited, so colonization pressure and alternative reservoirs were not fully characterized. We lacked molecular typing to prove clonality with the sink isolate and had incomplete antifungal susceptibility panels/quality control; therapeutic drug monitoring was unavailable. Management heterogeneity (agent choice, line removal timing) and retrospective data abstraction introduce confounding and missingness; follow-up was largely in-hospital, precluding long-term outcomes. Accordingly, temporality does not establish causality, and generalizability to other NICUs is limited.

CONCLUSION

This report documents a nosocomial outbreak of Candida lusitaniae bloodstream infections in five high-risk neonates, linked to a contaminated environmental reservoir. Despite significant comorbidities such as prematurity and asphyxia, early diagnosis and targeted Voriconazole therapy resulted in favorable outcomes for all patients. The successful containment of the outbreak through strict infection control measures and environmental decontamination highlights the critical importance of continuous surveillance and hygiene compliance in preventing rare fungal pathogens in neonatal intensive care settings.

DISCLOSURE

Ethical Clearance

This study was approved by the Health Research Ethics Committee of RSUD Bali Mandara, Bali Province, with Ethical Clearance No. 045/EA/KEPK.RSBM. DISKES/2023. Written informed consent was obtained from the parents or legal guardians of all patients included in this study for the publication of this case series

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Conflict of Interest

Authors declare no conflict of interest.

Author contribution

All authors were involved in the clinical management of the cases and the collection of data. All authors participated equally in the literature review, writing of the original draft, and final editing of the manuscript. All authors agree to be accountable for all aspects of the work.

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