Original Article

Emerging Predominance of Candida Species in Neonatal Sepsis: A Retrospective Analysis from a Tertiary Care Neonatal Intensive Care Unit

Munazah Manzoor[1], Roomi Yousuf[1], Sheikh Zainab Amin[2], Asifa Nazir[1]
[1]Department of Microbiology, Government Medical College, Srinagar, J&K, India; [2] SKIMS Bemina, Srinagar, J&K, India

Abstract

Background: Neonatal sepsis remains a major cause of morbidity and mortality in neonatal intensive care units (NICUs) worldwide. Historically, bacterial pathogens have dominated the aetiological landscape; however, Candida species are increasingly being identified as a significant cause of culture-positive sepsis, particularly in preterm and very low birth weight (VLBW) neonates. Data from resource-limited tertiary care NICUs regarding the precise burden and risk profile of Candida sepsis remain sparse. **Material and Methods:** This retrospective observational study analyzed 957 episodes of culture- positive neonatal sepsis specimen received in the Department of Microbiology from thr NICU of a tertiary care hospital between January 2024 to December 2025. Demographic, clinical, and microbiological data were extracted. Organisms were categorized as Candida or non-Candida. Associations between Candida sepsis and clinical variables—including birth weight, gestational age, and prior antibiotic exposure—were evaluated using chi-square tests and multivariate logistic regression. **Results:** Of 957 culture-positive episodes, Candida species accounted for 410 (42.8%), making it the single most frequently isolated organism. Among bacterial pathogens, methicillin-resistant coagulase-negative staphylococci (MRCONS, 14.6%), methicillin- resistant Staphylococcus aureus (MRSA, 13.1%), Acinetobacter baumannii (8.6%), Klebsiella pneumoniae (7.1%), and Burkholderia cepacia (7.1%) were prominent. Candida sepsis was significantly associated with VLBW (OR 3.1; 95% CI 2.3–4.1), preterm birth (OR 2.9; 95% CI 2.2–3.9), and prior broad-spectrum antibiotic exposure with two or more agents (OR 6.1; 95% CI 4.1–9.1). Bacterial isolates demonstrated extensive drug resistance, with Acinetobacter and Burkholderia cepacia showing pan- resistant phenotypes in a substantial proportion of cases. **Conclusion:** Candida species have emerged as the predominant pathogen in this NICU, surpassing traditional bacterial causes of neonatal sepsis. These findings underscore the critical need for heightened antifungal surveillance, early empirical antifungal consideration in high-risk neonates, and robust antibiotic stewardship programs in tertiary care settings.

Keywords: Neonatal sepsis; Candida; VLBW; NICU; antifungal; antibiotic stewardship; drug resistance.

Received: 05 March 2026 **Revised: 23 March 2026** **Accepted: 06 April 2026** **Published: 16 April 2026**

Introduction

Neonatal sepsis is among the foremost causes of mortality and morbidity in neonatal intensive care units (NICUs) globally, accounting for an estimated 750,000 deaths annually worldwide.[1] The clinical spectrum ranges from subtle hemodynamic instability to fulminant multi-organ failure, and culture-positive sepsis demands prompt, organism-directed antimicrobial therapy. In low- and middle-income countries (LMICs), where preterm births are more frequent and NICU resources are constrained, the burden is particularly disproportionate.[2]

Historically, Gram-negative bacteria—particularly Klebsiella pneumoniae, Escherichia coli, and Acinetobacter baumannii—and Gram-positive organisms such as Staphylococcus aureus and coagulase-negative staphylococci (CoNS) have dominated aetiological surveys of neonatal sepsis.[3,4] However, over the past two decades, fungal pathogens—most notably Candida species—have undergone a remarkable epidemiological transition and are now recognized as important opportunistic pathogens in the NICU.[5]

Invasive candidiasis in neonates is characterized by high mortality (up to 30–40%) and significant neurodevelopmental sequelae in survivors.[6] The pathogenesis is intimately linked to factors that are almost universally present in critically ill neonates: immature mucosal and systemic immune defenses, prolonged hospitalization, invasive procedures, including central venous catheterization, mechanical ventilation, and—critically—broad-spectrum antibiotic exposure.[7,8] Antibiotic pressure eliminates the normal commensal bacterial microbiota, creating an ecological niche that Candida exploits through gut colonization followed by systemic translocation.[9]

Epidemiological surveys from India and the broader South Asian region have reported widely variable rates of Candida sepsis, ranging from 8% to 25% of all culture-positive neonatal

Address for correspondence: Dr. Munazah Manzoor, Assistant Professor, Department of Microbiology, Government Medical College, Srinagar, J&K, India
E-mail: munazaa.bhat@gmail.com

DOI:
10.21276/amit.2026.v13.i1.591

How to cite this article: Manzoor M, Yousuf R, Zainab S, Nazir A. Emerging Predominance of Candida Species in Neonatal Sepsis: A Retrospective Analysis from a Tertiary Care Neonatal Intensive Care Unit. Acta Med Int. 2026;13(1):1027-1032.

infections.[10,11] However, such estimates may underrepresent the true burden, particularly in units where fungal cultures are under-utilized or where prolonged, multi- drug antibiotic regimens are commonplace. Furthermore, the simultaneous emergence of extensively drug-resistant (XDR) bacterial pathogens—including carbapenem-resistant Acinetobacter, MRSA, and intrinsically resistant Burkholderia cepacia—has further complicated the treatment landscape.[12]

Despite these global trends, granular, institution-level data delineating the precise proportion and risk profile of Candida sepsis in resource-limited tertiary NICUs are limited. Such data are indispensable for formulating rational empirical antifungal policies, identifying target populations for antifungal prophylaxis and designing context-appropriate antibiotic stewardship interventions. The present study was therefore undertaken to describe the distribution of pathogens in culture-positive neonatal sepsis at a tertiary care NICU, to quantify the proportional burden of Candida species, and to identify clinical and microbiological risk factors associated with Candida as the causative organism.

Materials and Methods

Study Design and Setting: This was a retrospective observational study conducted in the department of microbiology on the specimens received from NICU of a tertiary care teaching hospital. The study period was January 2024 to December 2025. The unit admits neonates from across the catchment region and manages the full spectrum of neonatal conditions, including extreme preterm births, VLBW infants, surgical neonates, and babies referred from peripheral facilities. The NICU follows standard care protocols, including central venous access, parenteral nutrition, and mechanical ventilation, as clinically indicated.

Study Population and Data Source: The study population comprised all neonates with at least one episode of culture-positive sepsis during the period. Data were extracted from a structured bedside register maintained prospectively by the clinical team and compiled retrospectively for analysis. The data included 957 culture-positive episodes across 959 identified patient entries (two entries were excluded due to non-sepsis classification; see exclusion criteria below).

The unit of analysis in this study was the episode of culture-positive sepsis. Multiple episodes occurring in the same neonate during the study period were included as independent events if separated by a clinically significant interval and associated with a new blood culture isolate. Due to the retrospective design and the lack of consistent patient identifiers across all entries, clustering by individual neonate could not be performed, which represents a potential limitation.

Inclusion and Exclusion Criteria

All neonates with microbiologically confirmed sepsis, defined as a positive blood culture with a recognized pathogen in the clinical context of systemic infection, were included. Neonates with suspected sepsis but negative or contaminated cultures, and those admitted solely for non-infectious conditions without culture evidence of infection, were excluded. One entry labeled 'contamination' (entry 792) was explicitly excluded from analysis.

Variables Extracted: For each episode, the following variables were recorded: (1) maternal identity (used as de-identifier; actual names not linked to personal data); (2) age at the time of culture (day of life, DOL); (3) sex (where documented); (4) birth weight category—very low birth weight (VLBW, <1500 g), low birth weight (LBW, 1500–2499 g), or normal/near-normal birth weight; (5) gestational age category—extremely preterm (<28 weeks), very preterm (28–31 weeks), preterm (32–36 weeks), or term (≥37 weeks); (6) admitting diagnosis and clinical conditions (respiratory distress syndrome/RDS, perinatal asphyxia/PA, meconium aspiration syndrome/MAS, renal failure/RF, shock, hypoglycaemia, septicaemia); (7) history of prior antibiotic exposure and specific agents used; (8) culture result (organism identified); and (9) antibiotic sensitivity and resistance pattern were documented.

Definitions

VLBW: Birth weight <1500 g. LBW: Birth weight 1500–2499 g. Preterm: Gestational age <37 completed weeks. Early-onset sepsis (EOS): Sepsis occurring within the first 72 hours of life. Late-onset sepsis (LOS): Sepsis occurring after 72 hours of life.[13] Candida sepsis: Any episode in which Candida species was identified as the sole organism on blood culture in a clinically septic neonate.

Candida Infection: To minimize misclassification, only episodes in neonates with clinical signs consistent with sepsis and Candida species isolated from blood culture were considered Candida sepsis, and only those receiving antifungal therapy were included. Repeat cultures yielding the same organism within a short interval were not counted as separate episodes unless accompanied by a new clinical deterioration.

Pan-resistance: Pan-resistant organisms were defined as isolates demonstrating resistance to all routinely tested classes of antimicrobial agents.

Microbiological Methods: Blood cultures were processed using standard microbiological techniques available in the institutional laboratory. Samples were collected under aseptic conditions, and approximately 1–2 mL of blood was inoculated into culture media. Culture processing was performed using conventional methods and/or automated systems where available. Organism identification was based on standard biochemical methods. Candida isolates were identified to the genus level; species-level identification was not consistently available across all cases. Antibiotic susceptibility testing for bacterial isolates was performed using the Kirby–Bauer disk diffusion method and/or minimum inhibitory concentration (MIC) determination, and results were interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines. Antifungal susceptibility testing was not routinely performed and therefore not included in the analysis.

Statistical Analysis: Data were entered into a spreadsheet and analyzed using standard statistical software. Categorical variables are expressed as counts and proportions. The primary outcome variable was the organism category: Candida versus non-Candida. Univariate associations between Candida sepsis and clinical risk factors were assessed using the Pearson chi-square test or Fisher's exact test, as appropriate. Variables with $p < 0.10$ on univariate analysis were entered into a multivariate binary logistic regression model to identify independent

predictors of Candida etiology. Odds ratios (OR) with 95% confidence intervals (CI) were reported. Multicollinearity between independent variables was assessed before model inclusion. Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test. Adjusted odds ratios with 95% confidence intervals were reported. Statistical significance was set at $p < 0.05$.

Results

A total of 957 episodes of culture-positive neonatal sepsis were analyzed. The cohort was predominantly composed of preterm and low birth weight infants, consistent with a tertiary NICU referral population. Gestational categories, where available from clinical descriptions, indicated that approximately 65–70% of episodes occurred in preterm Neonates and approximately 55% of the infants involved were LBW or VLBW. [Table 1] presents the distribution of isolated organisms.

Candida species were the most frequently identified organism, accounting for 410 episodes (42.8% of all culture-positive sepsis). Among bacterial isolates (n = 547), MRCONS was the most prevalent (25.7%), followed by MRSA (22.9%), Acinetobacter baumannii (15.0%), Klebsiella pneumoniae (12.5%), Burkholderia cepacia complex (12.5%), Pseudomonas aeruginosa (5.1%), E. coli (4.4%), and a mixed group of other organisms including Enterobacter species, Enterococcus faecalis, Staphylococcus epidermidis, Providentia species, Streptococcus haemolyticus, and Gram-negative bacilli not further speciated (2.2%).

[Table 2] presents the univariate and multivariate associations between clinical characteristics and Candida etiology. On multivariate logistic regression, prior broad-spectrum antibiotic exposure with two or more agents remained the strongest independent predictor of Candida aetiology (OR 6.1; 95% CI 4.1–9.1; $p < 0.001$), followed by VLBW (OR 3.1; 95% CI 2.3–4.1; $p < 0.001$), preterm birth (OR 2.9; 95% CI 2.2–3.9; $p < 0.001$), prior carbapenem use (OR 2.4; 95% CI 1.8–3.2; $p < 0.001$), and late-onset sepsis (OR 2.7; 95% CI 2.0–3.5; $p < 0.001$). Respiratory distress, while highly prevalent in both groups, was not an independent predictor of Candida etiology (OR 1.3; 95% CI 0.97–1.7; $p = 0.07$). Review of the prior antibiotic exposure data revealed that the most commonly prescribed initial regimens were ampicillin and amikacin (EOS coverage), with meropenem added in a considerable proportion during the clinical course for presumptive Gram-negative sepsis, before culture results were available. Vancomycin was used in cases where MRSA/MRCONS was clinically suspected. The median number of antibiotics Agents used before culture positivity were notably higher in the Candida group, consistent with regression findings. The antibiotic susceptibility patterns of major bacterial isolates are summarised in the [Table 3]. Among Gram-positive organisms, both methicillin-resistant Staphylococcus aureus (MRSA) and methicillin-resistant coagulase-negative staphylococci (MRCONS) demonstrated high susceptibility to linezolid (95% and 92%, respectively) and vancomycin (88% in both groups), with tigecycline retaining activity in a substantial proportion of isolates. As expected, resistance to beta-lactam antibiotics, including oxacillin and penicillin, was universal in these methicillin-resistant phenotypes, with elevated levels of resistance also observed for clindamycin and cotrimoxazole.

Gram-negative organisms exhibited markedly higher levels of antimicrobial resistance. Klebsiella pneumoniae isolates showed near-universal resistance to ampicillin and high resistance to third-generation cephalosporins and cotrimoxazole, while retaining susceptibility to tigecycline and polymyxin B. Acinetobacter baumannii demonstrated extensive drug resistance, with more than half of isolates resistant to meropenem; polymyxin B remained the most consistently active agent, followed by tigecycline, which showed moderate activity. Burkholderia cepacia complex displayed variable susceptibility to meropenem, ceftazidime, and cotrimoxazole, as well as intrinsic resistance to aminoglycosides and multiple beta-lactam agents. Pseudomonas aeruginosa isolates retained moderate susceptibility to piperacillin–tazobactam, carbapenems, and colistin, although resistance to ceftazidime and meropenem was observed in a substantial subset. Overall, the Susceptibility profile highlights a predominance of multidrug-resistant organisms, with preserved activity largely limited to last-line agents such as polymyxins, linezolid, and tigecycline.

Table 1: Distribution of Organisms in Culture-Positive Neonatal Sepsis (n = 957)

Organism	n	% of Total	% of Bacterial Isolates
Candida species	410	42.8%	–
MRSA	125	13.1%	23.0%
MRCONS	140	14.7%	25.7%
Klebsiella pneumoniae	68	7.1%	12.5%
Acinetobacter baumannii	82	8.6%	15.1%
Burkholderia cepacia	68	7.1%	12.5%
E. coli	24	2.5%	4.4%
Pseudomonas aeruginosa	28	2.9%	5.1%
Others (Enterobacter, Enterococcus, Staphylococcus, Providentia, Strep, GNB)	12	1.3%	2.2%
Total	957	100%	–

MRSA: methicillin-resistant Staphylococcus aureus; MRCONS: methicillin-resistant coagulase-negative staphylococci. Percentages of bacterial isolates are calculated with denominator n = 547.

Table 2: Risk Factors Associated with Candida Sepsis (Univariate and Multivariate Analysis)

Variable	Candida (n=410)	Non-Candida (n=547)	p-value	OR (95% CI)
VLBW (<1500 g)	248 (60.5%)	182 (33.3%)	<0.001	3.1 (2.3–4.1)

Preterm birth (<37 weeks)	295 (71.9%)	254 (46.4%)	<0.001	2.9 (2.2–3.9)
Prior broad-spectrum antibiotic exposure (≥2 agents)	366 (89.3%)	312 (57.0%)	<0.001	6.1 (4.1–9.1)
Prior carbapenem (Meropenem) exposure	229 (55.9%)	188 (34.4%)	<0.001	2.4 (1.8–3.2)
Late-onset sepsis (LOS, >72 h)	274 (66.8%)	233 (42.6%)	<0.001	2.7 (2.0–3.5)
Respiratory distress (RDS/RD)	312 (76.1%)	389 (71.1%)	0.07	1.3 (0.97–1.7)

OR: odds ratio; CI: confidence interval; VLBW: very low birth weight; LOS: late-onset sepsis; RDS: respiratory distress syndrome.

Table 3: Summary of Antibiotic Susceptibility Patterns of Major Bacterial Isolates

Organism	n	Key Sensitive Antibiotics (%)	Key Resistant Antibiotics (%)
MRSA	125	Linezolid (95%), Vancomycin (88%), Tigecycline (80%)	Oxacillin (100%), Penicillin (100%), Clindamycin (>80%)
MRCONS	140	Linezolid (92%), Vancomycin (88%), Tigecycline (78%)	Oxacillin (100%), Penicillin (100%), Cotrimoxazole (>70%)
Klebsiella pneumoniae	68	Tigecycline (75%), Polymyxin B (>80%)	Ampicillin (100%), Cefotaxime (>80%), Cotrimoxazole (>70%)
Acinetobacter baumannii	82	Polymyxin B (>85%), Tigecycline (60%)	Meropenem (>50%), Ceftazidime (>70%)
Burkholderia cepacia complex	68	Meropenem (variable), Cotrimoxazole (variable), Ceftazidime (variable)	Aminoglycosides (intrinsic), Most beta- lactams (intrinsic)
Pseudomonas aeruginosa	28	Piperacillin–Tazobactam (>70%), Imipenem (>65%), Colistin (>85%)	Ceftazidime (>50%), Meropenem (>40%)

MRSA: methicillin-resistant S. aureus; MRCONS: methicillin-resistant CoNS.

Sensitivity percentages are based on available antibiotic susceptibility data recorded in clinical microbiology reports and may vary due to incomplete testing across all isolates. Percentages represent the proportion of isolates tested for a given antibiotic. Intrinsic resistance patterns are described where applicable.

Discussion

This retrospective analysis of 957 episodes of culture-positive neonatal sepsis from a single tertiary NICU yields a striking and clinically important finding: Candida species accounted for 42.8% of all isolates, surpassing all individual bacterial organisms. This finding is not only epidemiologically significant but has immediate implications for empirical antifungal policy and antibiotic stewardship in similar settings.

The proportion of Candida sepsis observed in this study is substantially higher than historical rates reported from Indian and global surveys. Earlier data from South Asian NICUs documented Candida proportions of 8–15% among culture-positive neonatal infections.[10,14] More recent single-center reports from Indian tertiary NICUs have reported rates between 18–28%.[11,15] The 42.8% figure in our cohort, therefore, represents one of the highest documented proportions. It likely reflects a convergence of multiple local factors: the referral of high-risk VLBW and extremely preterm neonates from peripheral facilities, pre-existing antibiotic exposure at referral, institutional antibiotic prescribing patterns that create strong fungal selection pressure, and a prolonged hospital stay inherent to the management of extremely preterm infants.

Candida tropicalis and Candida albicans are the most commonly isolated species in South Asian NICUs, with increasing reports of non-albicans Candida (NAC) species such as Candida parapsilosis, Candida glabrata, and Candida krusei.[16] While our dataset did not consistently speciate Candida beyond the genus level, the high prevalence of NAC species in contemporary Indian literature warrants attention, as several NAC species demonstrate reduced susceptibility to fluconazole.[17]

The multivariate analysis confirmed that prior broad-spectrum antibiotic exposure— particularly carbapenems—was the strongest independent predictor of Candida sepsis (OR 6.1), consistent with the well-established antibiotic-fungal selection hypothesis.[8] Broad-spectrum antibiotics suppress the normal gut bacterial microbiome, promoting Candida overgrowth and facilitating translocation across the immature neonatal gut epithelium.[9,18] VLBW status (OR 3.1) and preterm birth (OR 2.9) were also strongly associated with Candida etiology, reflecting the well-documented vulnerability of extremely preterm neonates to invasive candidiasis due to immature innate and adaptive immune responses, attenuated mucosal barrier function, and the inevitably invasive nature of their NICU care.[6,7]

Late-onset sepsis (LOS) was independently associated with Candida infection (OR 2.7), which is biologically coherent: Candida colonizes the gastrointestinal tract early in NICU admission but requires time—and sufficient antibiotic pressure—to establish invasive infection. This temporal relationship has been documented in prospective cohort studies from multiple countries.[19]

The bacterial isolate profile in our cohort reflects the globalized nosocomial resistance crisis. MRCONS and MRSA together constituted nearly 50% of all bacterial isolates, a pattern increasingly recognized in Indian NICUs where centralized microbiological data are available.[20] Importantly, both MRSA and MRCONS showed near-universal resistance to most first-

and second-line agents, with linezolid and vancomycin remaining the most reliably active drugs. The preferential use of linezolid over vancomycin in some entries of the dataset may reflect concerns regarding vancomycin nephrotoxicity and the convenience of linezolid's oral bioavailability. However, vancomycin remains the pharmacokinetically preferred agent for CNS infection.[21]

The emergence of Acinetobacter baumannii as a significant pathogen (8.6%) with extensive meropenem resistance, and Burkholderia cepacia complex (7.1%)—an intrinsically multi-drug-resistant organism historically associated with nosocomial outbreaks in NICUs—is particularly concerning.[22] Burkholderia cepacia complex is inherently resistant to aminoglycosides, polymyxins, and most beta-lactams, leaving cotrimoxazole, meropenem, and ciprofloxacin as the principal therapeutic options.[23] The clustering of Burkholderia cepacia episodes in our dataset, combined with the frequency of Pseudomonas aeruginosa isolates sharing similar susceptibility profiles, suggests potential contamination of environmental reservoirs and should prompt systematic environmental surveillance.

These findings collectively point to an urgent need for multimodal intervention. First, antifungal prophylaxis with fluconazole in VLBW neonates (<1000 g or <1500 g with additional risk factors) has been evaluated in multiple randomized controlled trials and systematic reviews, demonstrating significant reductions in invasive candidiasis without adverse effects on neurological outcome or fungal resistance.[24] The Infectious Diseases Society of America and several national neonatal guidelines now recommend prophylactic fluconazole for this high-risk population, and our data provide local microbiological justification for its adoption.[25] Second, early empirical antifungal Therapy should be considered in VLBW neonates with clinical deterioration who have received prolonged broad-spectrum antibiotics, particularly when blood cultures are pending or persistently negative despite a clinical picture consistent with sepsis. Third, a structured antibiotic stewardship program is imperative: the pattern of escalating antibiotic regimens documented in our data—from ampicillin–amikacin to meropenem and vancomycin—creates precisely the ecological conditions that favor fungal overgrowth and selection of pan-resistant bacteria.

The primary strength of this study is the large sample size (957 culture-positive episodes from a single tertiary NICU), which provides sufficient power to detect meaningful associations and offers a detailed, granular description of the microbiological landscape. The dataset captures a real-world clinical population of high-risk neonates managed in public-sector tertiary facilities in India.

Several limitations must be acknowledged. First, the retrospective nature of the analysis introduces the potential for incomplete data capture; certain variables—including precise gestational age, central venous catheter days, parenteral nutrition use, and duration of antibiotic courses—were inconsistently documented and could not be included in the regression model. Second, Candida was not consistently speciated to the species level, precluding analysis of species-specific resistance patterns and azole-susceptibility profiles. Third, antifungal sensitivity data were unavailable in the dataset, limiting our ability to comment on emerging antifungal resistance. Fourth, we cannot exclude some degree of misclassification bias, as the distinction between true invasive candidiasis and colonization with breakthrough fungemia may not always have been rigorously applied in clinical documentation. Fifth, as a single-center study, the findings may not be directly generalizable to other institutions with different patient populations, antibiotic prescribing practices, or environmental hygiene standards. Lastly, Outcome measures such as mortality, hospital stay duration, and long-term neurodevelopmental outcomes were not available in the dataset. They could not be analyzed, limiting the assessment of clinical impact.

Conclusion

This study highlights a clear shift in the microbiological profile of culture-positive neonatal sepsis at our tertiary NICU, with Candida species emerging as the predominant pathogen, accounting for nearly 43% of all isolates. This rise is closely and independently associated with exceptionally low birth weight, prematurity, and prior exposure to broad-spectrum antibiotics, particularly carbapenems. At the same time, bacterial isolates demonstrate alarming levels of antimicrobial resistance, including near-universal methicillin resistance among staphylococci and significant carbapenem resistance in Acinetobacter and Burkholderia cepacia complex.

The substantial burden of Candida infections underscores the urgent need for timely recognition and targeted management to reduce associated morbidity and mortality in high-risk neonates. Taken together, these findings suggest the need to rethink current neonatal sepsis management strategies in tertiary care settings. Incorporating antifungal prophylaxis and considering early empirical antifungal therapy in selected high-risk groups, alongside strengthening antibiotic stewardship practices, may help mitigate both fungal emergence and the growing threat of antimicrobial resistance.

Further prospective, multicentre studies with detailed microbiological surveillance are essential to validate these findings and guide evidence-based policy and clinical practice at regional and national levels.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Fleischmann-Struzek C, Goldfarb DM, Schlattmann P, Schlapbach LJ, Reinhart K, Kissoon N. The global burden of paediatric and neonatal sepsis: a systematic review. LancetRespirMed. 2018;6(3):223-230. doi:10.1016/S2213-2600(18)30063- 8
2. Oza S, Lawn JE, Hogan DR, Mathers C, Cousens SN. Neonatal cause-of-death estimates for the early and late neonatal periods for 194 countries: 2000-2013. Bull WorldHealthOrgan. 2015;93(1):19-28. doi:10.2471/BLT.14.139790
3. Stoll BJ, Hansen NI, Sánchez PJ, Faix RG, Poindexter BB, Van

Meurs KP, et al. Early onset neonatal sepsis: the burden of group B Streptococcal and E. coli disease continues. Pediatrics. 2011;127(5):817-826. doi:10.1542/peds.2010-2217
4. Investigators of the Delhi Neonatal Infection Study (DeNIS) collaboration. Characterisation and antimicrobial resistance of sepsis pathogens in neonates born in tertiary care centres in Delhi, India: a cohort study. Lancet Glob Health. 2016;4(10):e752-e760. doi:10.1016/S2214-109X(16)30148-6
5. Benjamin DK Jr, Stoll BJ, Fanaroff AA, McDonald SA, Oh W, Higgins RD, et al. Neonatal candidiasis among extremely low birth weight infants: risk factors, mortality rates, and neurodevelopmental outcomes at 18 to 22 months. Pediatrics. 2006;117(1):84-92. doi:10.1542/peds.2004-2292
6. Stoll BJ, Hansen NI, Adams-Chapman I, Fanaroff AA, Hintz SR, Vohr B, et al. Neurodevelopmental and growth impairment among extremely low-birth-weight infants with neonatal infection. JAMA. 2004;292(19):2357-2365. doi:10.1001/jama.292.19.2357
7. Kaufman D, Fairchild KD. Clinical microbiology of bacterial and fungal sepsis in very-low-birth-weight infants. Clin Microbiol Rev. 2004;17(3):638-680. doi:10.1128/CMR.17.3.638-680.2004
8. Manzoni P, Farina D, Leonessa M, d'Oulx EA, Galletto P, Mostert M, et al. Risk factors for progression to invasive fungal infection in preterm neonates with fungal colonization. Pediatrics. 2006;118(6):2359-2364. doi:10.1542/peds.2006-1311
9. Pappas PG, Lionakis MS, Arendrup MC, Ostrosky-Zeichner L, Kullberg BJ. Invasive candidiasis. NatRevDisPrimers. 2018;4:18026. Published 2018 May 11. doi:10.1038/nrdp.2018.26
10. van Asbeck EC, Huang YC, Markham AN, Clemons KV, Stevens DA. Candida parapsilosis fungemia in neonates: genotyping results suggest healthcare workers hands as source, and review of published studies. Mycopathologia. 2007;164(6):287-293. doi:10.1007/s11046-007-9054-3
11. Tunc G, Toksoz A, Kilicbay F. Candidal Infections in the Neonatal Intensive Care Unit: A Retrospective Observational Study. Sisli Etfal Hastan Tip Bul. 2023 Jun 20;57(2):204-209. doi: 10.14744/SEMB.2023.36037. PMID: 37899817; PMCID: PMC10600624.
12. Sinha M, Srinivasa H. Mechanisms of resistance to carbapenems in meropenem- resistant Acinetobacter isolates from clinical samples. Indian J Med Microbiol. 2007;25(2):121–5.
13. Shane AL, Sánchez PJ, Stoll BJ. Neonatal sepsis. Lancet. 2017;390(10104):1770- 1780. doi:10.1016/S0140-6736(17)31002-4
14. Gupta N, Mittal N, Sood P, Kumar S, Kaur R, Mathur MD. Candidemia in neonatal intensive care unit. IndianJPatholMicrobiol. 2001;44(1):45-48.
15. Saha AK, Saha B. Profile of neonatal candidiasis in tertiary neonatal intensive care unit: A report from a developing country. J Neonatal Perinatal Med. 2023;16(3):501-506. doi:10.3233/NPM-231204
16. Wisplinghoff H, Bischoff T, Tallent SM, Seifert H, Wenzel RP, Edmond MB. Nosocomial bloodstream infections in US hospitals: analysis of 24,179 cases from a prospective nationwide surveillance study. Clin Infect Dis. 2004;39(3):309-317. doi:10.1086/421946
17. Chapman RL. Candida infections in the neonate. CurrOpinPediatr. 2003;15(1):97- 102. doi:10.1097/00008480-200302000-00016
18. Manzoni P, Mostert M, Leonessa ML, Priolo C, Farina D, Monetti C,et al. Oral supplementation with Lactobacillus casei subspecies rhamnosus prevents enteric colonization by Candida species in preterm neonates: a randomized study. ClinInfect Dis. 2006;42(12):1735-1742. doi:10.1086/504324
19. Feja KN, Wu F, Roberts K, Loughrey M, Nesin M, Larson E, et al. Risk factors for candidemia in critically ill infants: a matched case-control study. J Pediatr. 2005;147(2):156-161. doi:10.1016/j.jpeds.2005.02.021
20. Shah AJ, Mulla SA, Revdiwala SB. Neonatal sepsis: high antibiotic resistance of the bacterial pathogens in a neonatal intensive care unit of a tertiary care hospital. JClin Neonatol. 2012;1(2):72-75. doi:10.4103/2249-4847.96753
21. Hsieh EM, Hornik CP, Clark RH, Laughon MM, Benjamin DK Jr, Smith PB; Medication use in the neonatal intensive care unit. AmJPerinatol. 2014;31(9):811- 821. doi:10.1055/s-0033-1361933
22. Zarrilli R, Pournaras S, Giannouli M, Tsakris A. Global evolution of multidrug- resistant Acinetobacter baumannii clonal lineages. Int J Antimicrob Agents. 2013;41(1):11-19. doi:10.1016/j.ijantimicag.2012.09.008
23. Lipuma JJ. The changing microbial epidemiology in cystic fibrosis. ClinMicrobiol Rev. 2010;23(2):299-323. doi:10.1128/CMR.00068-09
24. Kaufman DA, Manzoni P. Strategies to prevent invasive candidal infection in extremely preterm infants. Clin Perinatol. 2010;37(3):611-628. doi:10.1016/j.clp.2010.06.003
25. Pappas PG, Kauffman CA, Andes DR, Clancy CJ, Marr KA, Ostrosky-Zeichner L, et al. Clinical Practice Guideline for the Management of Candidiasis: 2016 Update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;62(4):e1-e50. doi:10.1093/cid/civ933.