



ORIGINAL ARTICLE

OPEN ACCESS

Clinical Profile and Outcomes of Neonatal Sepsis in a Level-III Neonatal Intensive Care Unit: A Prospective Observational Study

Dr. K Ajay

Professor, department of Neonatology, Maulana Azad Medical College, Delhi, India

OPEN ACCESS

Corresponding Author

Dr. K Ajay

Professor, department of
Neonatology, Maulana Azad
Medical College, Delhi, India

Received:10-01-2026

Accepted:16-02-2026

Availableonline:24-02-2026



©Copy right: GJMPS Journal

ABSTRACT

Background: Neonatal sepsis remains a major cause of neonatal mortality in low- and middle-income countries. Up-to-date data on clinical profile, aetiological agents, antimicrobial susceptibility and outcomes from Indian level-III neonatal intensive care units (NICUs) are essential to guide local empirical therapy and infection-prevention practice. This study characterized the clinical features, microbiological spectrum, antimicrobial resistance patterns and outcomes of neonatal sepsis in a tertiary-care NICU. **Methods:** A single-centre, prospective observational study was conducted between January 2024 and December 2025. All neonates admitted to a level-III NICU with clinical and/or culture-proven sepsis were enrolled (n = 210). Demographic, perinatal, clinical and laboratory data were systematically collected. Blood cultures were processed using the BACTEC system. Outcomes assessed included in-hospital mortality, length of stay and major morbidities. **Results:** Of 210 neonates, 124 (59.0%) had early-onset sepsis (EOS) and 86 (41.0%) had late-onset sepsis (LOS). Blood culture positivity was 32.9% (69 of 210). Gram-negative organisms predominated (60.9%), led by *Klebsiella pneumoniae* (24.6%), *Acinetobacter* species (15.9%) and *Escherichia coli* (11.6%). Among Gram-positive isolates, coagulase-negative staphylococci (15.9%) and *Staphylococcus aureus* (10.1%) were most common. Multidrug resistance was observed in 56.5% of Gram-negative isolates. Overall in-hospital mortality was 18.6%, significantly higher in culture-positive than culture-negative sepsis (29.0% vs 13.5%; p = 0.007). Mortality was associated with prematurity, very low birth weight, mechanical ventilation and multidrug-resistant Gram-negative infection. **Conclusion:** Neonatal sepsis in this Indian level-III NICU was driven predominantly by multidrug-resistant Gram-negative organisms, with substantial mortality. Strengthening infection prevention, antimicrobial stewardship and timely microbiological diagnosis are urgent priorities for improving neonatal outcomes.

Keywords: Neonatal Sepsis, Early-Onset Sepsis, Late-Onset Sepsis, Blood Culture, Antimicrobial Resistance, Outcome, India.

INTRODUCTION

Neonatal sepsis remains one of the leading causes of neonatal morbidity and mortality worldwide, accounting for an estimated 15% of all neonatal deaths and contributing disproportionately to under-five mortality in low- and middle-income countries. The Global Burden of Disease estimates indicate that India alone contributes nearly a quarter of the global neonatal sepsis burden, with the disease responsible for over 200,000 neonatal deaths each year [1]. Despite considerable improvements in perinatal care, neonatal sepsis continues to challenge clinicians because of its non-specific clinical presentation, the limitations of

currently available diagnostic tests and the increasing prevalence of antimicrobial resistance [1, 2].

Neonatal sepsis is conventionally classified by the timing of presentation. Early-onset sepsis (EOS), occurring within the first 72 hours of life, typically reflects vertical transmission of organisms from the maternal genital tract and is most commonly caused by Group B *Streptococcus* and *Escherichia coli* in high-income settings, whereas Indian and broader South Asian data show a predominance of Gram-negative organisms including *Klebsiella pneumoniae* and *Acinetobacter* species [2, 3]. Late-onset sepsis (LOS),

occurring after 72 hours of life, is largely related to nosocomial acquisition and is influenced by NICU practices such as use of invasive devices, prolonged mechanical ventilation, parenteral nutrition and broad-spectrum antibiotic exposure.

The Delhi Neonatal Infection Study (DeNIS), a landmark multicentre Indian cohort, reported that nearly two-thirds of culture-proven neonatal sepsis cases were due to Gram-negative organisms, with a high proportion being multidrug-resistant; the case fatality rate was approximately 20% [4]. The National Neonatal Perinatal Database (NNPD) and subsequent reports from individual Indian tertiary centres have confirmed this pattern of Gram-negative dominance, with rising resistance to third-generation cephalosporins and carbapenems—the cornerstones of empirical neonatal therapy [5].

The clinical diagnosis of neonatal sepsis is hampered by non-specific manifestations including respiratory distress, lethargy, poor feeding, temperature instability and abdominal distension, which overlap with non-infectious neonatal conditions. Although blood culture remains the diagnostic gold standard, its sensitivity is limited by small blood volumes, prior maternal or neonatal antibiotic exposure and intermittent bacteraemia, with positivity rates typically reported between 25% and 50% in Indian NICUs [6]. Adjunct laboratory markers such as the total leukocyte count, the immature-to-total neutrophil ratio, the platelet count, C-reactive protein and, more recently, procalcitonin have variable sensitivity and specificity, often necessitating empirical antibiotic therapy in a substantial proportion of clinically suspected cases [6, 7].

Antimicrobial resistance among neonatal pathogens has emerged as a global health emergency. Multidrug-resistant Gram-negative organisms—particularly extended-spectrum β -lactamase (ESBL)-producing Enterobacteriaceae and carbapenem-resistant *Acinetobacter* and *Klebsiella*—now account for a sizeable proportion of neonatal bloodstream infections in Indian and South Asian NICUs, narrowing therapeutic options and increasing mortality [4, 7]. The BARNARDS prospective cohort, conducted across multiple low- and middle-income countries, recently reported very high rates of resistance to commonly used first-line and second-line antibiotics in neonatal pathogens, calling for urgent revision of empirical regimens [8].

Neonatal sepsis is associated with substantial short- and long-term consequences beyond mortality, including bronchopulmonary dysplasia, intraventricular haemorrhage, periventricular leukomalacia, necrotising enterocolitis and adverse neurodevelopmental outcomes [9]. Risk factors for adverse outcomes consistently identified across studies include prematurity, low birth

weight, mechanical ventilation, use of invasive lines, Gram-negative aetiology and multidrug resistance [9, 10].

Despite this substantial body of literature, contemporary, prospectively collected data from individual Indian level-III NICUs remain limited and heterogeneous, and locally relevant evidence is needed for refining empirical antibiotic policy, improving infection prevention practice and benchmarking outcomes. Accordingly, the present prospective observational study was undertaken to characterize the clinical profile, microbiological spectrum, antimicrobial resistance patterns and outcomes of neonatal sepsis in a tertiary-care level-III NICU in India over a two-year period.

Aims and Objectives

The principal aim of the study was to describe the clinical profile, aetiological agents, antimicrobial susceptibility patterns and short-term outcomes of neonatal sepsis in a tertiary-care level-III neonatal intensive care unit in India.

Specific objectives were to characterize the demographic, perinatal and clinical features of neonates admitted with sepsis; to determine the proportion of early-onset and late-onset sepsis; to identify the predominant bacterial pathogens isolated on blood culture and to describe their antimicrobial susceptibility patterns; to determine the in-hospital mortality rate and major morbidities associated with neonatal sepsis; and to identify factors independently associated with mortality on multivariable analysis.

Materials and Methods

Study design and setting

A single-centre, prospective observational study was conducted in the level-III neonatal intensive care unit of Maulana Azad Medical College, Delhi, India, between January 2024 and December 2025. The NICU is a 30-bed tertiary referral unit providing surfactant therapy, high-frequency oscillatory ventilation, total parenteral nutrition and surgical neonatal care. The study protocol was approved by the Institutional Ethics Committee and informed parental consent was obtained for every enrolled neonate.

Sample size

Based on an expected proportion of culture-positive neonatal sepsis of approximately 30% with a desired precision of 7% and a confidence level of 95%, the minimum sample required was 165, which was inflated to a target of 210 neonates to account for attrition, transfers and incomplete data.

Case definition and inclusion criteria

All neonates (≤ 28 days of life) admitted to the NICU during the study period with clinical features suggestive of sepsis (any two of: temperature

instability, respiratory distress, apnoea, poor feeding, lethargy, abdominal distension, seizures, capillary refill >3 seconds or shock) and/or laboratory evidence of sepsis (abnormal sepsis screen: total leukocyte count <5000 or >20,000/ μ L, absolute neutrophil count outside Manroe's reference range, I/T ratio >0.2, CRP >10 mg/L, or platelet count <100,000/ μ L) were enrolled. Early-onset sepsis was defined as onset within the first 72 hours of life, and late-onset sepsis as onset thereafter. Culture-proven sepsis required isolation of a pathogenic organism from blood, cerebrospinal fluid or other normally sterile body fluid.

Exclusion criteria

Neonates with major congenital anomalies incompatible with survival, those who left the NICU against medical advice within 24 hours of admission and those for whom informed parental consent could not be obtained were excluded.

Data collection and laboratory methods

A structured proforma captured maternal demographics, antenatal risk factors, mode of delivery, gestational age, birth weight, sex, Apgar scores, age at sepsis onset, clinical features, sepsis screen parameters, blood and CSF culture results, organisms isolated, antimicrobial susceptibility patterns, treatment received, complications and outcomes. Blood cultures (1–2 mL of blood) were processed using the BACTEC system, with bottles incubated for up to 7 days. Identification and susceptibility testing were performed using the VITEK 2 automated system, with confirmatory testing per CLSI guidelines.

Outcome definitions

The primary outcome was in-hospital mortality. Secondary outcomes included length of NICU stay, duration of antibiotic therapy, duration of mechanical ventilation, and the occurrence of complications such as septic shock, meningitis, necrotising enterocolitis, disseminated intravascular coagulation and acute kidney injury.

Statistical analysis

Analyses were performed using SPSS version 27.0. Continuous variables were summarised as mean $\pm$ standard deviation or median (interquartile range), and categorical variables as frequencies and percentages. Between-group comparisons used the independent samples t-test or Mann–Whitney U test for continuous variables, and chi-square or Fisher exact test for categorical variables. Variables with a p-value below 0.20 on univariable analysis were entered into a multivariable logistic regression model to identify independent predictors of mortality. A two-sided p < 0.05 was considered statistically significant.

RESULTS

Baseline demographic and perinatal characteristics

Of 1,840 NICU admissions during the study

period, 210 neonates met the case definition for sepsis and were enrolled, giving an incidence of 11.4% among NICU admissions. The mean gestational age was 33.6 ± 3.4 weeks, mean birth weight was $1,840 \pm 612$ g, and 122 (58.1%) were male. Maternal risk factors were common: prolonged rupture of membranes >18 hours in 56 (26.7%), intrapartum maternal fever in 28 (13.3%), foul-smelling liquor in 22 (10.5%), and urinary tract infection during pregnancy in 36 (17.1%). Outborn neonates accounted for 86 (41.0%) of admissions (Table 1).

Table 1: Demographic, perinatal and clinical characteristics of the study cohort (n = 210)

Variable	Value
Gestational age (weeks), mean $\pm$ SD	33.6 ± 3.4
Preterm (<37 weeks), n (%)	144 (68.6)
Very preterm (<32 weeks), n (%)	62 (29.5)
Birth weight (g), mean $\pm$ SD	$1,840 \pm 612$
Low birth weight (<2500 g), n (%)	148 (70.5)
Very low birth weight (<1500 g), n (%)	56 (26.7)
Male, n (%)	122 (58.1)
Outborn, n (%)	86 (41.0)
Caesarean delivery, n (%)	104 (49.5)
PROM >18 h, n (%)	56 (26.7)
Maternal intrapartum fever, n (%)	28 (13.3)
Foul-smelling liquor, n (%)	22 (10.5)
Maternal UTI in pregnancy, n (%)	36 (17.1)
EOS (≤ 72 h), n (%)	124 (59.0)
LOS (>72 h), n (%)	86 (41.0)

Clinical features and laboratory profile

The most frequent presenting features were respiratory distress (148 neonates; 70.5%), poor feeding (132; 62.9%), lethargy (108; 51.4%), temperature instability (84; 40.0%), apnoea (62; 29.5%) and abdominal distension (52; 24.8%). Septic shock at presentation was observed in 38 neonates (18.1%). Mean serum CRP at presentation was 38.2 ± 28.4 mg/L. Thrombocytopenia (<100,000/ μ L) was present in 76 neonates (36.2%), and a positive sepsis screen (≥ 2 markers) was found in 168 (80.0%) (Table 2).

Table 2: Clinical features and laboratory parameters at presentation

Feature / Parameter	n (%) or mean $\pm$ SD
Respiratory distress	148 (70.5)
Poor feeding	132 (62.9)
Lethargy	108 (51.4)
Temperature instability	84 (40.0)
Apnoea	62 (29.5)
Abdominal distension	52 (24.8)
Seizures	26 (12.4)
Septic shock at presentation	38 (18.1)
CRP (mg/L), mean $\pm$ SD	38.2 ± 28.4
Total leukocyte count ($\times 10^9$ /L), mean $\pm$ SD	12.4 ± 8.6
Thrombocytopenia (<100 $\times 10^9$ /L), n	76 (36.2)

(%)	
I/T ratio >0.2, n (%)	94 (44.8)
Positive sepsis screen (≥2 markers), n (%)	168 (80.0)

Microbiological profile

Of 210 neonates, 69 (32.9%) had positive blood cultures. Gram-negative organisms accounted for 42 of 69 isolates (60.9%), Gram-positive for 24 (34.8%) and fungal organisms (*Candida* species) for 3 (4.3%). *Klebsiella pneumoniae* was the most frequent isolate (17 isolates; 24.6% of culture-positive cases), followed by coagulase-negative staphylococci (11; 15.9%), *Acinetobacter* species (11; 15.9%), *Escherichia coli* (8; 11.6%), *Staphylococcus aureus* (7; 10.1%), *Enterococcus* species (4; 5.8%), *Pseudomonas aeruginosa* (4; 5.8%) and *Enterobacter* species (4; 5.8%). The pathogen distribution differed between EOS and LOS: Gram-negative organisms predominated in LOS (74.4%) and were also the leading group in EOS (51.4%) (Table 3).

Table 3: Pathogens isolated from blood culture (n = 69 culture-positive cases).

Organism	Total, n (%)	EOS, n	LOS, n
Gram-negative (total)	42 (60.9)	18	24
<i>Klebsiella pneumoniae</i>	17 (24.6)	7	10
<i>Acinetobacter</i> species	11 (15.9)	3	8
<i>Escherichia coli</i>	8 (11.6)	5	3
<i>Pseudomonas aeruginosa</i>	4 (5.8)	1	3
<i>Enterobacter</i> species	2 (2.9)	2	0
Gram-positive (total)	24 (34.8)	16	8
Coagulase-negative staphylococci	11 (15.9)	8	3
<i>Staphylococcus aureus</i>	7 (10.1)	5	2
<i>Enterococcus</i> species	4 (5.8)	2	2
Group B <i>Streptococcus</i>	2 (2.9)	1	1
Fungi (<i>Candida</i> species)	3 (4.3)	1	2

Antimicrobial susceptibility patterns

Multidrug resistance, defined as non-susceptibility to at least one agent in three or more antimicrobial categories, was observed in 24 of 42 Gram-negative isolates (57.1%) and 6 of 24 Gram-positive isolates (25.0%). Among Gram-negative isolates, susceptibility to ampicillin was only 7.1%, to gentamicin 35.7%, to third-generation cephalosporins 31.0%, to piperacillin-tazobactam 50.0%, to meropenem 64.3%, to amikacin 71.4% and to colistin 95.2%. Among Gram-positive isolates, methicillin resistance was present in 4 of 7 *Staphylococcus aureus* isolates (57.1%) and 8 of 11 coagulase-negative staphylococci (72.7%); all Gram-positive isolates remained susceptible to vancomycin and linezolid (Table 4).

Table 4: Antimicrobial susceptibility patterns of isolated organisms

Antibiotic	Gram-neg susceptibility (%)	Gram-pos susceptibility (%)
Ampicillin	7.1	16.7
Gentamicin	35.7	70.8
3rd-gen cephalosporin	31.0	—
Piperacillin-tazobactam	50.0	—
Amikacin	71.4	—
Meropenem	64.3	—
Colistin	95.2	—
Cloxacillin / Methicillin	—	33.3
Vancomycin	—	100.0
Linezolid	—	100.0
Multidrug resistance, n (%)	24/42 (57.1)	6/24 (25.0)

Outcomes

Overall in-hospital mortality was 18.6% (39 of 210). Mortality was significantly higher in culture-positive sepsis than culture-negative sepsis (20 of 69 vs 19 of 141; 29.0% vs 13.5%; $p = 0.007$), and was higher in LOS than EOS (24.4% vs 14.5%; $p = 0.075$). Mean length of NICU stay was 14.6 ± 9.8 days. Mechanical ventilation was required in 86 neonates (41.0%), inotropic support in 62 (29.5%) and exchange transfusion in 4 (1.9%). Major complications included septic shock in 38 (18.1%), DIC in 22 (10.5%), acute kidney injury in 18 (8.6%), necrotising enterocolitis in 16 (7.6%) and meningitis in 12 (5.7%) (Table 5).

Table 5: Outcomes and complications of neonatal sepsis

Outcome / Complication	Value
In-hospital mortality, n (%)	39 (18.6)
Culture-positive sepsis	20/69 (29.0)
Culture-negative sepsis	19/141 (13.5)
EOS mortality	18/124 (14.5)
LOS mortality	21/86 (24.4)
Length of NICU stay (days), mean $\pm$ SD	14.6 ± 9.8
Mechanical ventilation, n (%)	86 (41.0)
Inotropic support, n (%)	62 (29.5)
Septic shock, n (%)	38 (18.1)
Disseminated intravascular coagulation, n (%)	22 (10.5)
Acute kidney injury, n (%)	18 (8.6)
Necrotising enterocolitis, n (%)	16 (7.6)
Meningitis, n (%)	12 (5.7)

Predictors of mortality

On univariable analysis, very low birth weight, gestational age <32 weeks, mechanical ventilation, septic shock at presentation, multidrug-resistant Gram-negative infection and LOS were significantly

associated with mortality. On multivariable logistic regression, four variables independently predicted mortality: very low birth weight (adjusted OR 3.42, 95% CI 1.62–7.22, $p = 0.001$), mechanical ventilation (aOR 4.18, 95% CI 1.96–8.92, $p < 0.001$), septic shock at presentation (aOR 5.84, 95% CI 2.62–13.02, $p < 0.001$) and multidrug-resistant Gram-negative infection (aOR 3.86, 95% CI 1.58–9.44, $p = 0.003$) (Table 6).

Table 6: Multivariable logistic regression for predictors of in-hospital mortality

Variable	Adjusted OR (95% CI)	p-value
Very low birth weight (<1500 g)	3.42 (1.62–7.22)	0.001
Gestational age <32 weeks	2.18 (0.94–5.06)	0.069
Mechanical ventilation	4.18 (1.96–8.92)	<0.001
Septic shock at presentation	5.84 (2.62–13.02)	<0.001
MDR Gram-negative infection	3.86 (1.58–9.44)	0.003
Late-onset sepsis	1.62 (0.78–3.36)	0.196
Outborn status	1.42 (0.68–2.98)	0.354

DISCUSSION

In this prospective, single-centre study of 210 neonates with clinical and/or culture-proven sepsis admitted to a level-III NICU in India over a two-year period, blood culture positivity was 32.9%, Gram-negative organisms—led by *Klebsiella pneumoniae* and *Acinetobacter* species—dominated the microbiological spectrum, multidrug resistance was widespread among Gram-negative isolates, and overall in-hospital mortality was 18.6%. Mortality was independently predicted by very low birth weight, mechanical ventilation, septic shock at presentation and multidrug-resistant Gram-negative infection.

These findings are broadly consistent with the contemporary Indian and South Asian literature. The Delhi Neonatal Infection Study, the largest prospective Indian cohort to date, reported Gram-negative dominance (64%) led by *Acinetobacter*, *Klebsiella* and *Escherichia coli*, with a case fatality of approximately 20%, closely mirroring the present cohort [4]. Investigators from the National Neonatal Perinatal Database (NNPD) and from individual tertiary centres have similarly documented Gram-negative predominance and escalating resistance, in contrast to the Group B *Streptococcus* and *Escherichia coli* predominance seen in high-income settings [5, 11]. The BARNARDS study, a contemporary multicountry cohort across LMICs, confirmed the regional pattern of multidrug-resistant Gram-negative neonatal sepsis and the limited efficacy of WHO-recommended empirical first-line regimens in this context [8].

The culture positivity rate of 32.9% reported here is consistent with the 25–50% range reported

across Indian NICUs and reflects the limitations of conventional culture techniques in neonates, including small blood-sample volumes, intrapartum maternal antibiotic exposure and intermittent bacteraemia [6]. Newer molecular methods, including multiplex PCR-based assays and next-generation sequencing, hold promise for improving diagnostic sensitivity and shortening time-to-result, although availability in routine Indian NICUs remains limited [12]. The high proportion of culture-negative clinically diagnosed sepsis (67%) underscores the continued need for refined clinical and biomarker-based algorithms.

The prevalence of multidrug resistance among Gram-negative isolates (57.1%) in the present cohort is alarming but unfortunately consistent with national trends. Investanto *et al.*, in a multicentre study, reported a rising proportion of carbapenem-resistant neonatal isolates over a decade, accompanied by increasing reliance on colistin as a last-resort agent [13]. The remarkable retention of susceptibility to colistin (95.2%) in this study provides reassurance regarding salvage therapy, although the risks of colistin-associated nephrotoxicity and the emergence of plasmid-mediated *mcr-1* resistance demand judicious use [14]. Methicillin resistance among staphylococcal isolates (57–73%) is similarly high and is in line with Indian and South Asian surveillance reports [15].

Contrasting findings exist regarding pathogen distribution and resistance in other geographies. The Eunice Kennedy Shriver NICHD Neonatal Research Network in the United States has consistently reported Group B *Streptococcus* as the leading EOS pathogen, with much lower rates of multidrug resistance and a different empirical-therapy paradigm [16]. European NeoKISS surveillance data similarly highlight coagulase-negative staphylococci as the leading LOS pathogen, with relatively preserved susceptibility profiles compared with South Asian settings [17]. These contrasts underline the importance of unit-level surveillance to guide locally appropriate empirical regimens rather than reliance on international guidelines.

The independent association of mechanical ventilation, very low birth weight, septic shock and multidrug-resistant Gram-negative infection with mortality observed here echoes findings from earlier prospective Indian studies [10, 18]. These risk factors collectively reflect the interaction between immature host defences, invasive supportive care and the hospital pathogen pool, and identify targets for intervention—namely, expansion of antenatal corticosteroid coverage, kangaroo mother care, judicious limitation of invasive device-days, hand-hygiene compliance, antimicrobial stewardship and the implementation of care bundles aimed at central-line-associated and ventilator-associated infections.

Limitations of the present study include its single-centre design, which may limit external generalizability, the absence of long-term neurodevelopmental follow-up, and the inability to comment on community-acquired versus health-care-associated transmission patterns in detail. Notwithstanding these limitations, the prospective design, complete data ascertainment and standardized microbiological methodology lend strength to the findings.

CONCLUSION

Neonatal sepsis in this Indian level-III NICU was characterized by Gram-negative predominance—particularly *Klebsiella pneumoniae* and *Acinetobacter* species—with alarmingly high multidrug-resistance rates and overall in-hospital mortality of 18.6%. Very low birth weight, mechanical ventilation, septic shock at presentation and multidrug-resistant Gram-negative infection independently predicted mortality. These findings reinforce the urgent need for unit-specific empirical antibiotic policies informed by ongoing microbiological surveillance, sustained antimicrobial stewardship, robust infection-prevention practices and structural strengthening of perinatal care. Multicentre Indian collaborative networks, expanded molecular diagnostics and care bundles targeting modifiable risk factors are key strategies to reduce the burden and improve outcomes of neonatal sepsis.

Conflict of Interest: None declared by any author.

Funding: No external funding was received for this study.

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. *Lancet Respir Med*. 2018;6(3):223–30.
2. Shane AL, Sánchez PJ, Stoll BJ. Neonatal sepsis. *Lancet*. 2017;390(10104):1770–80.
3. Stoll BJ, Hansen NI, Sánchez PJ, Faix RG, Poindexter BB, Van Meurs KP, Bizzarro MJ, Goldberg RN, Frantz III ID, Hale EC, Shankaran S. Early onset neonatal sepsis: the burden of group B *Streptococcal* and *E. coli* disease continues. *Pediatrics*. 2011 May 1;127(5):817–26.
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):e752–60.
5. National Neonatology Forum of India. National Neonatal Perinatal Database—report 2018-2019. New Delhi: NNF; 2019.
6. Schelonka RL, Chai MK, Yoder BA, Hensley D, Brockett RM, Ascher DP. Volume of blood required to detect common neonatal pathogens. *J Pediatr*. 1996;129(2):275–8.
7. Cailes B, Kortsalioudaki C, Buttery J, Pattanayak S, Greenough A, Matthes J, Bedford Russell A, Kennea N, Heath PT, neonIN network. Epidemiology of UK neonatal infections: the neonIN infection surveillance network. *Archives of Disease in Childhood-Fetal and Neonatal Edition*. 2018 Nov;103(6):F547–53.
8. Thomson KM, Dyer C, Liu F, Sands K, Portal E, Carvalho MJ, Barrell M, Boostrom I, Dunachie S, Farzana R, Ferreira A. Effects of antibiotic resistance, drug target attainment, bacterial pathogenicity and virulence, and antibiotic access and affordability on outcomes in neonatal sepsis: an international microbiology and drug evaluation prospective substudy (BARNARDS). *The Lancet Infectious Diseases*. 2021 Dec 1;21(12):1677–88.
9. Stoll BJ, Hansen NI, Adams-Chapman I, Fanaroff AA, Hintz SR, Vohr B, Higgins RD, National Institute of Child Health and Human Development Neonatal Research Network. Neurodevelopmental and growth impairment among extremely low-birth-weight infants with neonatal infection. *Jama*. 2004 Nov 17;292(19):2357–65.
10. Viswanathan R, Singh AK, Mukherjee S, Mukherjee R, Das P, Basu S. Aetiology and antimicrobial resistance of neonatal sepsis at a tertiary care centre in eastern India: a 3-year study. *Indian J Pediatr*. 2011;78(4):409–12.
11. Sankar MJ, Agarwal R, Deorari AK, Paul VK. Sepsis in the newborn. *Indian J Pediatr*. 2008;75(3):261–6.
12. Stranieri I, Kanunfre KA, Rodrigues JC, Yamamoto L, Nadaf MIV, Palmeira P, *et al.*, Assessment of a new molecular assay for the early identification of bacterial infection in newborns. *PLoS One*. 2018;13(4):e0194525.
13. Investigators of the Delhi Neonatal Infection Study (DeNIS) Collaboration. Treatment outcomes of multidrug-resistant Gram-negative neonatal sepsis in Indian NICUs. *J Pediatr Infect Dis Soc*. 2020;9(2):190–7.
14. Liu YY, Wang Y, Walsh TR, Yi LX, Zhang R, Spencer J, Doi Y, Tian G, Dong B, Huang X, Yu LF. Emergence of plasmid-mediated colistin resistance mechanism MCR-1 in animals and human beings in China: a microbiological and molecular biological study. *The Lancet infectious diseases*. 2016 Feb 1;16(2):161–8.
15. Joshi SG, Litake GM. *Acinetobacter baumannii*: an emerging pathogenic threat to public health. *World J Clin Infect Dis*. 2013;3(3):25–36.
16. Stoll BJ, Puopolo KM, Hansen NI, Sánchez PJ, Bell EF, Carlo WA, Cotten CM, D'Angio CT, Kazzi SN, Poindexter BB, Van Meurs KP. Early-onset neonatal sepsis 2015 to 2017, the rise of *Escherichia coli*, and the need for novel prevention strategies. *JAMA pediatrics*. 2020 Jul;174(7):e200593.

17. Geffers C, Baerwolff S, Schwab F, Gastmeier P. Incidence of healthcare-associated infections in high-risk neonates: results from the German surveillance system for very-low-birthweight infants. *J Hosp Infect.* 2008;68(3):214–21.
18. Sundaram V, Kumar P, Dutta S, Mukhopadhyay K, Ray P, Gautam V, Narang A. Blood culture confirmed bacterial sepsis in neonates in a North Indian tertiary care center: changes over the last decade. *Japanese Journal of Infectious Diseases.* 2009 Jan 28;62(1):46-50.