

## Bacteriological Profile and Antimicrobial Susceptibility Patterns of Blood Culture Isolates in Neonatal Septicemia at a Tertiary Care Hospital in Kurnool, Andhra Pradesh

**Akshatha peddinti<sup>1\*</sup> G. Mounika<sup>2</sup> Veeresh Kumar Kodi<sup>3</sup> Sarada.D<sup>4</sup> Sowmya Alahari<sup>5</sup>**

<sup>1</sup>Associate Professor, Department of Microbiology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India

<sup>2</sup>Assistant Professor, Department of Microbiology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India

<sup>3</sup>Assistant Professor, Department of Microbiology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India

<sup>4</sup>Professor, Department of Microbiology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India.

<sup>5</sup>Professor, Department of Pharmacology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India.

 OPEN ACCESS

### ABSTRACT

#### **Corresponding Author:**

**Akshatha Peddinti**

Associate Professor, Department of Microbiology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India

Mail-id:

[akshathapraveen01@gmail.com](mailto:akshathapraveen01@gmail.com)

Received: 20-12-2025

Accepted: 13-01-2026

Published: 31-02-2026

**Background:** Neonatal sepsis is a systemic infectious condition characterized by clinical signs and symptoms of infection with or without microbiological confirmation, occurring in the first month of life. It remains a major contributor to neonatal morbidity and mortality worldwide. Early recognition and timely initiation of appropriate antimicrobial therapy, along with adequate supportive care are crucial determinants of survival. **Methods:** This prospective study was conducted from January 2025 to December 2025 and included 198 neonates with clinical suspicion of sepsis. Blood samples were collected under strict aseptic conditions and processed using the BacT/ALERT automated blood culture system. Bacterial isolates were identified by standard microbiological methods and antimicrobial susceptibility testing was performed using the Kirby–Bauer disc diffusion method in accordance with CLSI guidelines. **Results:** Of the 198 blood samples processed, 54 (27.27%) were culture positive. Gram-negative bacteria were more frequently isolated (55.55%) than Gram-positive organisms (44.44%). *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* were the predominant Gram-negative isolates, each accounting for 43.33% of Gram-negative cases, followed by *Acinetobacter baumannii*, *Escherichia coli*, and *Citrobacter* species. Among Gram-positive organisms, Methicillin-resistant *Staphylococcus aureus* was the most common isolate (50%), followed by *Enterococcus* species, Coagulase-negative *Staphylococci* and Group B *Streptococcus*. A high level of resistance to commonly used Tier-1 and Tier-2 antibiotics was observed among both Gram-negative and Gram-positive isolates. **Conclusion:** The study demonstrates a predominance of resistance to commonly used drugs in neonatal sepsis. Regular monitoring of local bacteriological patterns, strict infection control practices, rational antibiotic use, and ongoing antimicrobial resistance surveillance are essential to improve neonatal outcomes and limit the spread of resistant organisms.

**Copyright © International Journal of Medical and Pharmaceutical Research**

**Keywords:** Neonatal sepsis; Blood culture; Antibiogram; Antimicrobial resistance.

### INTRODUCTION

Neonatal septicaemia refers to a serious bloodstream infection occurring during the first 28 days of life and continues to pose a major threat to neonatal survival worldwide. The burden of this condition is disproportionately higher in low- and middle-income countries, where it remains a leading cause of neonatal illness and death despite ongoing improvements in perinatal and neonatal care [1]. The rapid course of the disease and the absence of specific early clinical features contribute significantly to poor outcomes.

The estimated global occurrence of neonatal sepsis ranges between 1 and 5 cases per 1,000 live births, with developing regions reporting markedly higher incidence rates [2]. Multiple maternal and neonatal factors predispose newborns to

infection, including prematurity, low birth weight, prolonged rupture of membranes, maternal febrile illness or infections, invasive neonatal interventions, and extended hospital stays [3,4]. Early diagnosis is particularly challenging, as the initial manifestations of sepsis are subtle and frequently overlap with other neonatal disorders, often delaying appropriate treatment [5,6].

Infectious diseases remain a common cause of morbidity and mortality during the neonatal period, with approximately one in ten infants experiencing an infection within the first month of life. The frequency of neonatal infections is strongly influenced by the availability and quality of healthcare services, being substantially higher in resource-limited settings. Newborn infections may result from exposure to a wide range of bacterial and non-bacterial pathogens acquired during the intrapartum or postnatal period. Microorganisms colonizing the maternal genitourinary and gastrointestinal tracts are important sources of neonatal infection.

The bacterial spectrum responsible for neonatal septicaemia in developing countries is diverse. Commonly isolated pathogens include *Klebsiella pneumoniae*, *Escherichia coli*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, coagulase-negative staphylococci, *Staphylococcus aureus*, and Group B *Streptococcus*. Other organisms such as *Citrobacter* species, *Enterococcus* species, *Neisseria gonorrhoeae*, *Listeria monocytogenes*, *Streptococcus pneumoniae*, and *Haemophilus influenzae* are encountered less frequently.

Based on the timing of clinical presentation, neonatal sepsis is categorized as early-onset or late-onset. Early-onset sepsis occurs within the first 7 days of life and is usually acquired through vertical transmission from the mother either before or during childbirth. In contrast, late-onset sepsis develops at or beyond 7 days of age and is generally associated with pathogens acquired from the hospital environment or the community. The onset of infection is influenced by both the timing of microbial exposure and the pathogenic potential of the organism. In some cases, very late-onset sepsis, presenting after one month of age, may occur, particularly among very low birth weight infants or neonates requiring prolonged intensive care support.

Epidemiological studies suggest a higher incidence of sepsis among term male neonates compared to females, although this gender difference is less evident in preterm and low birth weight infants. Several maternal and neonatal factors increase the risk of early-onset sepsis, including maternal Group B *Streptococcus* colonization, prolonged rupture of membranes, maternal urinary tract infection, prematurity, chorioamnionitis, and maternal fever exceeding 38°C. The risk is further amplified in low birth weight infants in the presence of congenital immune disorders, genetic defects affecting innate immunity, metabolic conditions such as galactosemia, absence of splenic function, and anatomical abnormalities that favor bacterial proliferation, such as obstructive uropathy. Progression from colonization to invasive disease is influenced by host immunity, pathogen virulence, bacterial load, genetic susceptibility, and the presence or absence of protective maternal antibodies. Additionally, invasive procedures including neonatal resuscitation, endotracheal intubation, and umbilical catheterization are associated with an increased likelihood of bloodstream infections [7–10].

The present study was undertaken to analyze the bacteriological profile and antimicrobial susceptibility patterns of blood culture isolates obtained from neonates diagnosed with septicaemia and admitted to Viswabharathi Medical College and General Hospital, a tertiary care centre located in Kurnool, Andhra Pradesh.

## METHODOLOGY

This Prospective study was carried out in the Department of Microbiology in collaboration with the Neonatal Intensive Care Unit (NICU), Department of Paediatrics, Viswabharathi Medical College and General Hospital, Kurnool. All neonates admitted to the NICU with clinical features suggestive of septicaemia during the study period were included. Relevant maternal and neonatal risk factors associated with septicaemia were recorded.

Strict aseptic precautions were followed during blood sample collection to minimize contamination. The venipuncture site was cleansed using a sterile cotton swab soaked in 70% alcohol, followed by application of 10% povidone-iodine, and allowed to air dry for 30–60 seconds. Blood sample was collected according to the body weight of the neonates and the collected venous blood was inoculated into properly labelled BACTEC Ped Plus aerobic culture bottles after disinfecting the bottle cap with alcohol. Wherever possible, blood samples were obtained prior to the initiation of antibiotic therapy, however, in cases where delay was unavoidable, samples were collected after starting antibiotics.

The inoculated culture bottles were incubated in the BacT/ALERT 3D automated blood culture system at 37°C for a maximum duration of five days, in accordance with the manufacturer's instructions. The BacT/ALERT system detects microbial growth by monitoring carbon dioxide-induced pH changes using a colorimetric sensor located at the base of each bottle. Carbon dioxide produced during microbial metabolism diffuses through a permeable membrane into the sensor matrix, resulting in a color change that is automatically detected by the instrument. The bottles flagged with colour change were noted, sub cultured onto blood agar and MacConkey agar plates and incubated at 37°C for 18–24 hours. The colony morphology was assessed and Gram staining was performed. The final identification of bacterial isolates were achieved using standard biochemical tests including catalase, oxidase, coagulase, motility, indole, methyl red, Voges–Proskauer, citrate utilization, urease, and triple sugar iron tests. Bottles that showed no growth signal after five days were reported as culture negative.

Antimicrobial susceptibility testing was performed for all the confirmed isolates using the Kirby–Bauer disc diffusion method on Mueller–Hinton agar, following the Clinical and Laboratory Standards Institute (CLSI) 2025 guidelines. The bacterial inoculum was standardized to a 0.5 McFarland turbidity standard (approximately  $1.5 \times 10^8$  CFU/mL). The inoculum was evenly spread over the agar surface in three directions to ensure uniform distribution. Antibiotic discs were placed within 15 minutes of inoculation, maintaining an inter-disc distance of 24 mm. The plates were incubated in an

inverted position at 35°C for 16–18 hours in ambient air. Zones of inhibition were measured in millimetres, and isolates were categorized as susceptible, intermediate, or resistant based on CLSI breakpoint criteria.

## RESULTS

During the study period from January 2025 to December 2025, a total of 198 neonates with clinical suspicion of septicemia were admitted and investigated. Blood samples were collected under strict aseptic precautions and processed for culture. Out of the 198 samples analyzed, 54 (27.27%) were positive for bacterial growth, while 144 (72.73%) samples showed no growth (Table 1).

**Table 1: Sample distribution**

Category	Number	Percentage (%)
Culture positive	54	27.27
Culture negative	144	72.73
<b>Total</b>	<b>198</b>	<b>100</b>

Among the culture-positive cases, 29 (53.70%) isolates were obtained from male neonates and 23 (42.59%) from female neonates, with a male-to-female ratio of 1.2:1 (Table 2).

**Table 2: Gender-wise distribution of culture positive cases**

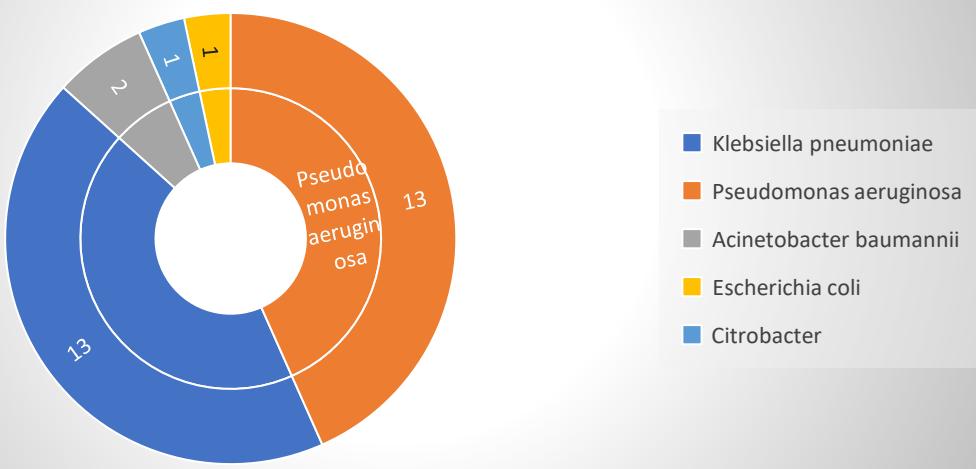
Gender	Number	Percentage (%)
Male	29	53.70
Female	23	42.59
<b>Total</b>	<b>54</b>	<b>100</b>

Of the 54 bacterial isolates, Gram-negative organisms predominated, accounting for 30 isolates (55.55%), whereas Gram-positive organisms constituted 24 isolates (44.44%). The distribution of Gram-negative isolates is shown in Table 3 & Fig.1. *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* were the most frequently isolated Gram-negative organisms, each accounting for 13 isolates (43.33%). This was followed by *Acinetobacter baumannii* in 2 cases (6.66%), and *Escherichia coli* and *Citrobacter* species in 1 case each (3.33%).

**Table 3 : Distribution of Gram negative isolates (n=30)**

Sl no.	Isolate	No. of isolates	Percentage (%)
1.	<i>Klebsiella pneumoniae</i>	13	43.33
2.	<i>Pseudomonas aeruginosa</i>	13	43.33
3.	<i>Acinetobacter baumannii</i>	2	6.66
4.	<i>Escherichia coli</i>	1	3.33
5.	<i>Citrobacter</i>	1	3.33

**Fig 1 : Distribution of Gram negative isolates (n=30)**

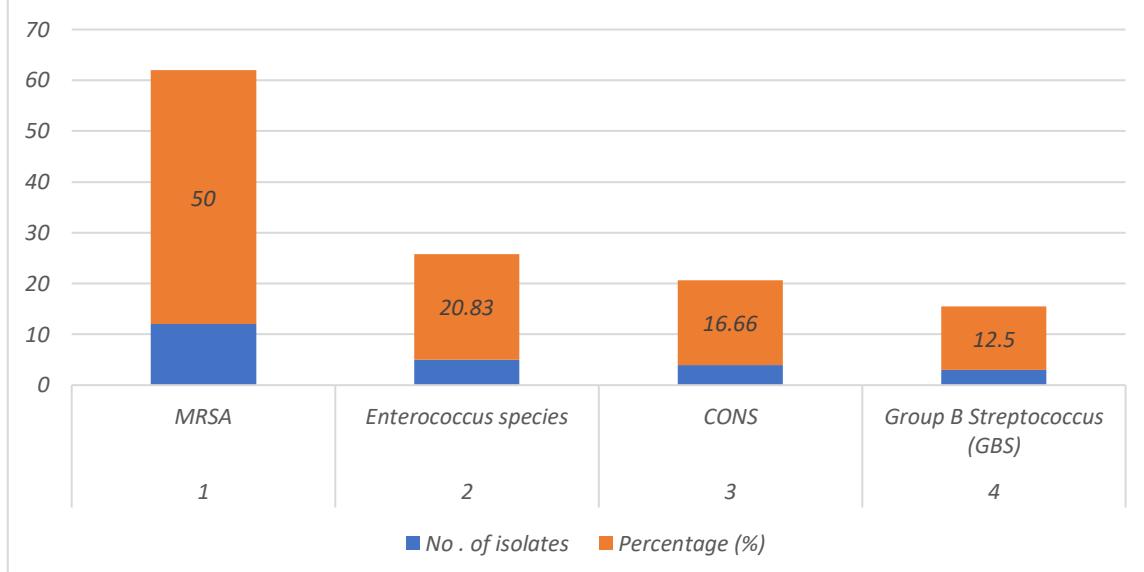


The distribution of Gram-positive isolates is depicted in Table 4 & Fig.2. Methicillin-resistant *Staphylococcus aureus* (MRSA) was the predominant Gram-positive pathogen, accounting for 12 isolates (50%). This was followed by *Enterococcus* species with 5 isolates (20.83%), coagulase-negative staphylococci (CONS) with 4 isolates (16.66%), and Group B *Streptococcus* with 3 isolates (12.50%).

**Table 4: Distribution of Gram positive isolates ( n =24)**

Sl no.	Organism	No . of isolates	Percentage (%)
1.	MRSA	12	50
2.	Enterococcus species	5	20.83
3.	CONS	4	16.66
4.	Group B <i>Streptococcus</i>	3	12.50

**Fig 2.: Distribution of Gram positive isolates ( n =24)**



**Table 5: Antibiogram pattern of Gram negative isolates**

Isolate	N =	A M P	CO T	GE N	AK	TO B	CI P	LE	C T R	C A Z	CP M	A M C	A /S	PI T	IP M	M R P	M I	AZ
<i>Klebsiella pneumonia</i>	1 3	N A	9 69. 23 %	3 23. 07 %	2 15. 38 %	3 23. 07 %	4 30. 76 %	4 30. 76 %	1 7.6 9 %	1 7.6 9 %	NA	N A	N A	4 30. 76 %	1 7.6 9% %	2 15. 38 %	N A	NA
<i>Pseudomonas aeruginosa</i>	1 3	N A	NA	NA	NA	7 53. 84 %	10 76. 92 %	10 76. 92 %	N A	0 % %	4 30. 76 %	N A	N A	11 84. 61 %	5 38. 46 %	10 76. 92 %	N A	2 15. 38 %
<i>Acinetobacter baumannii</i>	2	N A	2 100 %	2 100 %	2 100 %	1 50 %	1 50 %	1 50 %	N A	0 0 %	0 0% %	N A	1 50 0 %	1 50 %	2 100 %	2 100 0 %	2 10 %	NA
<i>Escherichia coli</i>	1	0 0 %	0 0% %	1 100 %	0 0% %	0 0% %	1 100 %	1 100 %	0 0 %	0 0 %	NA	0 0 %	0 0 %	1 100 %	1 100 %	N A	NA	

<b>Citrobacter species</b>	1	N A	1 100 %	1 100 %	0 0%	1 100 %	1 100 %	1 100 %	0 0%	0 0%	NA	0 0%	0 0%	1 100 %	0 0%	1 100 %	N A	NA
----------------------------	---	-----	---------	---------	------	---------	---------	---------	------	------	----	------	------	---------	------	---------	-----	----

Antimicrobial susceptibility patterns of Gram-negative isolates are summarized in Table 5. Klebsiella pneumoniae and Pseudomonas aeruginosa were the major isolates among Gram negative bacteria followed by Acinetobacter baumannii, Escherichia coli and citrobacter species. Among thirteen isolates of Klebsiella pneumoniae, nine isolates were sensitive to cotrimoxazole (69.23%), four isolates were sensitive piperacillin – tazobactum (30.76%), ciprofloxacin (30.76%), Levofloxacin (30.76%) , two isolates were meropenem (15.38%) and Amikacin (15.38%) and one isolate was sensitive to cefotaxime (7.69%) and imipenem (7.69%). Three isolates of Klebsiella pneumoniae were multidrug resistant, exhibited resistance to more than three antimicrobial classes tested.

Among thirteen isolates of Pseudomonas aeruginosa, eleven isolates were sensitive to piperacillin tazobactum (84.61%), ten isolates were sensitive to ciprofloxacin (76.92%), Levofloxacin (76.92%) and meropenem (76.92%), seven isolates were sensitive to tobramycin (53.84%), five isolates were sensitive to imipenem (38.46%), four isolates were sensitive to cefipime (30.06%) ,and two isolates were sensitive to Aztreonam (15.38%).

Two isolated Acinetobacter baumannii species showed 100% sensitivity to Gentamicin, Amikacin, Imipenem and 50% sensitivity to Ciprofloxacin, Levofloxacin, Tobramycin, Ampicillin- sulbactum and piperacillin –sulbactum.

Among 12 isolated MRSA,11 isolates were sensitive to Linezolid (91.16%), six isolates were sensitive to were sensitive to Gentamicin (50%), Tetracycline (50%), Doxycycline (50%), three isolates were sensitive to ciprofloxacin (25 %), Levofloxacin (25 %), Cotrimoxazole (25 %),two isolates were sensitive to Erythromycin (16.66%) and Clindamycin (16.66%).

Five Enterococcus species were isolated. Four isolates were sensitive to linezolid (80%) ,two isolates were sensitive to Ampicillin (40%) and High-level Gentamicin (40%).

Among twenty-four Gram positive cocci isolates, CONS was isolated in four samples and all isolated CONS were methicillin resistant, three isolates were sensitive to Linezolid (75%),two isolates were sensitive to Doxycycline (50%) , one isolate was sensitive to Gentamicin (25%) Tetracycline (25%) and Doxycycline (25%).

Three isolated streptococcus pneumoniae were 100% sensitive to Imipenem, Linezolid, 66.66% sensitive to Erythromycin, Clindamycin, Tetracycline, Doxycycline, Cefotaxime, Cefipime and 33.33% sensitive to Ampicillin, Ciprofloxacin, Levofloxacin.

Isolate	N =	AMP	E	CD	COT	CIP	LE	TE	DO	GEN	HLG	CTX	IMP	LZ
<i>Staph aureus</i>	1 2	NA	2 16.66 %	2 16.66 %	3 25 %	3 25 %	3 25 %	6 50%	6 50%	6 50 %	NA	NA	NA	11 91.66 %
<i>Enterococcus spp</i>	5	2 40%	NA	NA	NA	NA	NA	NA	NA	2 40 %	NA	NA	5 100 %	
<b>CONS</b>	4	0 0%	0 0%	0 0%	0 0%	0 0%	0 0%	1 25%	1 25%	1 25 %	NA	NA	NA	3 75%
<i>Strep pneumoniae</i>	3	1 33.33 %	2 66.66 %	2 66.66 %	0 0%	1 33.33 %	1 33.33 %	2 66.66 %	2 66.66 %	NA	NA	2 66.66 %	3 100 %	3 100 %

**Table 6: Antibiogram pattern of Gram-positive isolates**

AMP- Ampicillin, E-Erythromycin, CD-Clindamycin, COT-Cotrimoxazole, TE-Tetracycline, DO-Doxycycline, G- Gentamicin, HLG-High level Gentamicin, CTX-Cefotaxime, LZ-Linezolid, IPM-Imipenem, AMC-Amoxycav, CIP- Ciprofloxacin, LE-Levofloxacin

## DISCUSSION

Neonatal bacterial sepsis remains a significant contributor to illness and death during the newborn period. Hospital-based studies from South Asia report a culture-confirmed neonatal sepsis incidence of nearly 15.8 per 1,000 live births, with mortality approaching one-third of affected neonates [11]. Although advances in neonatal intensive care have led to improved outcomes among term infants, preterm and very-low-birth-weight neonates continue to face a substantial risk of both early- and late-onset sepsis, particularly infections acquired within healthcare settings. Survivors of neonatal sepsis may experience long-term complications, including neurodevelopmental impairment secondary to central nervous system involvement, as well as systemic complications such as septic shock, pulmonary hypertension, and chronic lung disease [12].

In the present study, blood cultures were positive in 54 out of 198 clinically suspected cases, yielding a positivity rate of 27.27%. This finding aligns with reports by Shittu et al. (22.22%), exceeds the rates observed by Neerul Pandita et al. (10.2%), and is lower than those documented by Bipin Gupta et al. (35.4%). Such variability across studies may be influenced by several factors, including prior empirical antibiotic therapy, undetected viral or fungal infections, differences in patient demographics, and variability in laboratory practices and infection control measures among institutions [13–15]. Gram-negative bacteria constituted the majority of isolates (55.55%) in this study, exceeding Gram-positive organisms (44.44%), a trend consistent with findings from studies conducted in India and Nigeria by Neerul Pandita et al., Mane et al., Mustafa et al., and Irrege et al. [16–18]. *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* were the most frequently isolated pathogens, collectively accounting for 43.33% of cases, followed by *Acinetobacter baumannii* (6.66%), corroborating earlier reports [14]. Infections caused by Gram-negative organisms are often associated with more severe clinical illness and demonstrate higher resistance to empirically used first- and second-line antibiotics recommended by the World Health Organization [19].

The pathogenicity of *Klebsiella pneumoniae* is largely attributed to its polysaccharide capsule, which confers resistance to phagocytosis and complement-mediated lysis. Additionally, its ability to adhere to host tissues through fimbrial and non-fimbrial adhesins enhances colonization and is frequently linked to multidrug resistance [20,21]. *Pseudomonas aeruginosa* is characterized by multiple virulence determinants, including endotoxins, exotoxin A, adhesins, and invasins, all of which contribute to its invasive potential and high fatality rates in neonatal infections [22]. *Acinetobacter baumannii* poses a serious therapeutic challenge due to its intrinsic resistance mechanisms and capacity to acquire resistance genes via mobile genetic elements [23].

Among Gram-positive organisms, methicillin-resistant *Staphylococcus aureus* (MRSA) accounted for half of the isolates, a proportion comparable to that reported by C. P. Nnamani et al. [24]. Resistance to  $\beta$ -lactam antibiotics in MRSA is mediated by the *mecA* gene, which encodes an altered penicillin-binding protein (PBP2a) with reduced affinity for these agents, resulting in broad  $\beta$ -lactam resistance [25]. The rapid spread of MRSA within neonatal units is often facilitated by cross-transmission through healthcare personnel, highlighting the critical role of adherence to infection prevention protocols [26].

*Enterococcus* species represented 20.83% of Gram-positive isolates, a finding similar to that of Elizabeth Antony et al. [27]. These organisms possess several virulence attributes, including capsule formation and biofilm production, enabling persistence on indwelling medical devices. Their intrinsic resistance to cephalosporins necessitates combination therapy, commonly involving aminoglycosides in conjunction with ampicillin or vancomycin, for effective treatment [28,29]. Coagulase-negative staphylococci accounted for 16.66% of Gram-positive isolates, a lower proportion than reported by Bipin Gupta et al. This reduced frequency may reflect stringent aseptic techniques during blood sample collection and improved catheter care practices in the study setting [15]. In neonatal intensive care units, CONS are frequent skin commensals that cause bloodstream infections by adhering to intravascular devices, followed by biofilm formation, which protects them from host immune responses [30,31].

Group B *Streptococcus* constituted 12.53% of isolates in the present study. The use of intrapartum antibiotic prophylaxis with penicillin or ampicillin has been shown to significantly reduce maternal-neonatal transmission and the incidence of early-onset GBS disease [32].

## CONCLUSION

Findings from the present study indicate that neonatal septicaemia in the study setting is predominantly caused by *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Enterococcus* species, coagulase-negative staphylococci, and *Acinetobacter baumannii*. A substantial proportion of these pathogens exhibited reduced susceptibility to routinely used CLSI Tier-1 and Tier-2 antimicrobial agents, thereby narrowing the scope of effective empirical therapy. The emergence of such resistance patterns can be attributed to a combination of inherent bacterial resistance mechanisms, injudicious antibiotic prescribing, and suboptimal infection prevention practices. Enhancing infection control protocols, strengthening antimicrobial stewardship initiatives, conducting continuous monitoring of local resistance trends, and implementing community-based educational programs to discourage irrational antibiotic use are essential strategies to curb antimicrobial resistance and reduce healthcare-associated infections.

## REFERENCES

- Shane AL, Sánchez PJ, Stoll BJ. Neonatal sepsis. *Lancet*. 2017;390(10104):1770–1780.
- Simonsen KA, Anderson-Berry AL, Delair SF, Davies HD. Early-onset neonatal sepsis. *Clin Microbiol Rev*. 2014;27(1):21–47.
- Dong Y, Speer CP. Late-onset neonatal sepsis. *Arch Dis Child Fetal Neonatal Ed*. 2015;100:F257–F263.
- Zaidi AKM, Huskins WC, Thaver D, et al. Hospital-acquired neonatal infections. *Lancet*. 2005;365:1175–1188.
- Liu L, Oza S, Hogan D, et al. Global causes of child mortality. *Lancet*. 2021;398(10302):870–905.
- Vernano S, Sharland M, Kazembe P, et al. Neonatal sepsis: An international perspective. *Arch Dis Child Fetal Neonatal Ed*. 2005;90:F220–F224.
- Forbes BA, Sahm DF, Weissfeld AS, editors. *Bailey & Scott's diagnostic microbiology*. 15th ed. St. Louis (MO): Elsevier; 2019.
- Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. *Nelson textbook of pediatrics*. 22nd ed. Philadelphia (PA): Elsevier; 2023.

9. Sandora TJ, Harper MB, editors. *Oxford handbook of infectious diseases and microbiology*. Oxford (UK): Oxford University Press; 2009.
10. Eichenwald EC, Hansen AR, Martin CR, Stark AR, editors. *Cloherty and Stark's manual of neonatal care*. South Asian ed. New Delhi: Wolters Kluwer; 2017.
11. Shane AL, Sánchez PJ, Stoll BJ. Neonatal sepsis. *Lancet*. 2017;390:1770–1780.
12. Edmond K, Zaidi A. New approaches to preventing neonatal sepsis. *PLoS Med*. 2010;7:e1000213.
13. Shittu OB, et al. Pattern of neonatal sepsis. *Afr J Clin Exp Microbiol*. 2018.
14. Pandita N, et al. Bacteriological profile of neonatal sepsis. *J Clin Diagn Res*. 2015;9:DC01–DC04.
15. Gupta B, et al. Etiology of neonatal septicemia. *Int J Contemp Pediatr*. 2016.
16. Mane AK, et al. Neonatal septicemia profile. *Int J Med Res Rev*. 2017.
17. Mustafa M, et al. Neonatal sepsis bacteriology. *J Evid Based Med Healthc*. 2016.
18. Irrege L, et al. Neonatal sepsis in Nigeria. *BMC Pediatr*. 2020;20:409.
19. WHO. *Managing possible serious bacterial infection in young infants*. WHO; 2015.
20. Podschun R, Ullmann U. Klebsiella spp. virulence. *Clin Microbiol Rev*. 1998;11:589–603.
21. Paczosa MK, Mecsas J. Klebsiella pneumoniae pathogenesis. *Microbiol Mol Biol Rev*. 2016;80:629–661.
22. Moradali MF, et al. Pseudomonas aeruginosa virulence. *Front Microbiol*. 2017;8:691.
23. Peleg AY, et al. Acinetobacter baumannii resistance. *Clin Microbiol Rev*. 2008;21:538–582.
24. Nnamani CP, et al. MRSA in neonatal sepsis. *Niger J Clin Pract*. 2019.
25. Chambers HF. Methicillin resistance mechanisms. *Clin Microbiol Rev*. 1997;10:781–791.
26. Boyce JM, Pittet D. Hand hygiene guideline. *MMWR*. 2002;51:1–45.
27. Antony E, et al. Enterococcal infections in neonates. *Indian J Med Microbiol*. 2016.
28. Arias CA, Murray BE. Enterococcal resistance. *N Engl J Med*. 2012;366:1919–1930.
29. Hollenbeck BL, Rice LB. Enterococcal intrinsic resistance. *Virulence*. 2012;3:421–433.
30. Otto M. Staphylococcal biofilms. *Curr Top Microbiol Immunol*. 2008;322:207–228.
31. Becker K, et al. CONS infections. *Clin Microbiol Rev*. 2014;27:870–926.
32. Verani JR, et al. Prevention of perinatal GBS disease. *MMWR*. 2010;59:1–36.