International Journal of Contemporary Pediatrics
Upasani VN et al. Int J Contemp Pediatr. 2026 Mar;13(3):491-497
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20260413

Case Series

Bacteria identified in neonatal sepsis cases from a neonatal intensive care unit, Ahmedabad, India

Vivek N. Upasani[1], Ajay C. Memakiya[2], Alpesh R. Patel[1], Rajat R. Patel[1], Harshitkumar J. Savalia[1], Devendrasinh D. Jhala[3], Shiva S. Chettiar[1]*

[1]Department of Molecular Biology, GeneXplore, Diagnostics and Research Centre Pvt. Ltd., Ahmedabad, Gujarat, India
[2]Department of Microbiology, Gujarat Vidyapith, Ahmedabad, Gujarat, India
[3]Department of Zoology, School of Sciences, Gujarat University, Ahmedabad, Gujarat, India

Received: 22 December 2025
Revised: 17 January 2026
Accepted: 03 February 2026

***Correspondence:**
Dr. Shiva S. Chettiar,
E-mail: scheti@genexplore.co.in

ABSTRACT

Neonatal sepsis (NS) causes mortality in the neonatal intensive care unit (NICU) due to infections by group B *Streptococcus* (GBS), emerging pathogens and antimicrobial resistance (AMR) strains. This study was undertaken to isolate and identify GBS and other bacteria from cases of neonatal sepsis in a NICU, Ahmedabad, Gujarat. Skin swab samples were collected from neonatal sepsis cases and inoculated in LIM broth. Growth was observed in all the LIM broth tubes inoculated with the sample. Six (6) beta-hemolytic and four (4) non-hemolytic bacteria were isolated on Blood agar plate, and pure cultures were obtained on LB agar plates. These isolates were identified using the Vitek2 system, and 16S rRNA sequencing method. Two (2) isolates, namely NSNH5L and NSNH3S, were identified as *Klebsiella pneumoniae* (96%) and *Staphylococcus haemolyticus* (97%), respectively, by the VITEK2 rapid identification system. Antimicrobial susceptibility testing revealed that *K. pneumoniae* was resistant to aminoglycosides, beta-lactams, and polypeptide antibiotics (Card: AST-N405), except fosfomycin and trimethoprim/sulfamethoxazole; whereas *S. haemolyticus* was sensitive to most antibiotics, except benzylpenicillin, ciprofloxacin, erythromycin, levofloxacin, gentamicin, and oxacillin (Card: AST-P628). Two (2) isolates, namely, GXDRC_03 (NSH01) and GXDRC_04 (NSH05), were identified as *Kocuria* arsenatis (99.75%) and *Staphylococcus epidermidis* (99.59%) based on 16S rRNA sequence homology study. The 16S rRNA sequences have been deposited in GenBank accession nos. PX533098 and PX5331127, respectively. This is the first report on 16S rRNA phylogeny-based identification of bacterial isolates, and isolation of *K. arsenatis* from neonatal sepsis sample(s) in Gujarat, India.

Keywords: *Kocuria arsenatis*, Beta-hemolysis, Neonatal sepsis, NICU, India

INTRODUCTION

Sepsis is a life-threatening condition in which the body's high immune system response causes organ dysfunction.[1] It is a clinical syndrome characterized as infectious diseases affected by bacteria, fungi and viral infection with or without bacteremia developed in new-born babies within 30 days of life after birth.[2] Neonatal sepsis (NS) is a "clinical syndrome" caused due to the presence of bacteremia during the first 3 months of life or preterm birth. NS is a one of the most important causes of child mortality globally.[3,4] Most death in low-income and middle-income countries is due to poor hygiene during neonatal period, and infection by multidrug-resistance pathogens.[5] NS infection is rare about 1-50 cases/1000 live births.[6,7]

New born especially having very low birth weight (VLBW) <1500 g and preterm infants are admitted in neonatal intensive care unit (NICU). GBS remains the primary causes of neonatal sepsis since the 1970s. On the

basis of age and timing of infection neonatal sepsis can be divided into two subtypes – Early-onset neonatal sepsis (EONS) and late-onset neonatal sepsis (LONS). EONS is defined as signs and symptoms of sepsis usually appear within 72 h of life (3 days) after birth, but most neonates (61% to 95%) become ill within the first 24 hours.

Group B *streptococcus* (GBS) or *Streptococcus agalactiae*, and *Escherichia coli* cause EONS representing 38% and 23% cases, respectively. Other bacteria associated with EONS are *Streptococcus viridians*, *Enterococcus spp., Staphylococcus aureus, Pseudomonas aeruginosa* and other gram-negative bacilli.[4]

LONS is defined as GBS infection in infants 3 months of age or older. In most cases of LONS, GBS disease occur in premature infants and very low birth weights. LONS is caused by bacteria/pathogen growing in external environment of home or hospital. Common signs and symptoms of neonatal sepsis are nonspecific, both onset shows similar types of clinical manifestations. The early symptoms of sepsis can be lethargy feeling, poor feeding, or irritability. Other symptoms include fever or hypothermia, diarrhea, vomiting, poor cry, shock, bleeding, poor weight gain, blank look, seizures, excessive crying, etc. Neonatal bacterial infections of the new-born include septicemia, meningitis, pneumonia, and arthritis.[8,9]

Sepsis etiological framework is affected by many factors like quality of life, cultural values, antibiotic therapy, etc. In a study on 6215 infants admitted to National Institute of Child Health and Human Development (NICHD), Neonatal Research Network (NRN) centers, 70% of late-onset infections were caused by gram-positive organisms, with coagulase-negative staphylococci about 48% of the infections.[10] The most common pathogen causing NS are beta-hemolytic streptococcal species. The other gram-negative bacteria causing sepsis include *Escherichia coli, Citrobacter spp., Proteus spp., Klebsiella pneumoniae, Enterobacter spp., Haemophilus influenzae, Acinetobacter spp.*, and *Pseudomonas aeruginosa*.[11] *Listeria monocytogenes* causes invasive disease in the new-born, spontaneous abortions or stillbirth if acquired during pregnancy. *Candida albicans* infection has also been reported in such cases.[12]

The diagnosis of GBS and other infections is established by isolation of the organisms from culture of blood, CSF, CRP, body fluid, and surface of skin. Blood cultures continue to be the diagnostic gold standard as a diagnostic test used to detect the presence of pathogenic microorganisms in body fluids (blood, urine, cerebrospinal fluid) from neonatal sepsis. Identification of a specific pathogen gives several benefits, particularly the ideal antibiotic choice and treatment duration.

In case of GBS detection swab sample is collected and inoculated into the selective Todd-Hewitt broth (LIM broth) and incubated at 37°C in incubator for 18-24 hours. The enriched broth is streaked on a blood agar plate. GBS are gram-positive cocci, beta-hemolytic, and catalase negative.[13] Bacteria isolated from sepsis samples can be identified by Vitek2 system, GC-MS, FAME, 16S rRNA sequencing and amplification of target nucleic acid sequence by polymerase chain reaction (PCR). GBS can also be detected by PCR by amplification of cfb, scB and atr genes.[14]

The aim of our research work was to detect and identify GBS and other bacteria from neonatal sepsis samples collected from a local NICU in Ahmedabad, India.

CASE SERIES

A total of six (6) skin swab samples from neonates (7–45 days) with sepsis were collected from a NICU, Ahmedabad, Gujarat, India, as given in Table 1. Written informed consent of the parents was obtained in all cases.

Table 1: Details of swab samples from neonates.

Sample no.	Age (days)	Weight of child (kg)
1	8	0.70
2	7	2.1
3	7	1.8
4	7	3.3
5	45	1.6
6	37	1.1

The swab sticks from the sepsis area (skin) were inoculated into a selective enrichment culture medium, namely, LIM broth (Todd-Hewitt broth), primarily to isolate GBS.[15] All tubes were incubated at 37°C for 18-24 hours. After the enrichment culture step, growth from the broth were streaked onto Blood agar (BA) plates and incubated at 37°C for 24-48 hours. The plates were observed for colonies with a clear zone of hemolysis (beta-hemolysis). Bacterial colonies (beta-hemolytic and non-hemolytic) selected from BA plates were streaked onto Luria Bertani (LB) agar plates to obtain pure cultures. These pure cultures were further studied using the routine microbiological methods.

Identification and AMR characteristics of two isolates were carried out using the VITEK2 rapid identification system version 9.02 at Supratech Micropath Laboratory, Ahmedabad. Two beta-hemolytic isolates were identified by 16S rRNA gene sequencing and phylogenetic analysis. The quantity and quality of the gDNA were determined by the Epoch microplate spectrometric method and agarose gel electrophoresis, respectively.

The polymerase chain reaction (PCR) products were purified using ExoSAP-ITTM PCR. The products were analysed by cycle sequencing using 27F and 1392 primers on the ABI3500 Genetic Analyzer (Sanger Sequencer). The 16S rRNA sequence contigs were analysed for similar sequences using the BLASTN program. MEGA11

software was used to construct the phylogenetic tree by the Neighbour-joining method.[16]

Growth was observed in all six LIM broth tubes inoculated with the sample after 24 hours of incubation. Turbidity was absent in the control uninoculated tube. GBS was not detected in any of the samples. However, ten isolates were obtained, out of which six (6) were beta-hemolytic and four (4) non-hemolytic. Most of the isolates were catalase-negative, except NSH4S and NSNH1L.

Two (2) isolates, NSH5L and NSH3S, were identified as *Klebsiella pneumoniae* (96%) and *Staphylococcus haemolyticus* (97%), respectively. *K. pneumoniae* was resistant to aminoglycoside, beta-lactam, and polypeptide antibiotics (Card: AST-N405), except fosfomycin and trimethoprim/sulfamethoxazole. *S. haemolyticus* was sensitive to most of the antibiotics, except benzylpenicillin, ciprofloxacin, erythromycin, levofloxacin, gentamicin, and oxacillin (Card: AST-P628) (Tables 2-5).

Two (2) isolates, namely NSH01 and NSH5A were identified as *Kocuria arsenatis* (99.75%) and *Staphylococcus epidermidis* (99.59%), respectively (Figures 1 and 2. The 16S rRNA sequences have been submitted in GenBank with accession nos. PX533098 and PX533112, respectively.

Table 2: Biochemical tests* result of *Klebsiella pneumoniae*.

Characteristics	Biochemical tests							
2	APPA	-	**21**	BXYL	+	**42**	SUCT	+
3	ADO	+	**22**	BAlap	-	**43**	NAGA	-
4	PyrA	+	**23**	ProA	+	**44**	AGAL	+
5	IARL	-	**26**	LIP	-	**45**	PHOS	+
7	dCEL	+	**27**	PLE	+	**46**	GlyA	+
9	BGAL	+	**29**	TyrA	+	**47**	ODC	-
10	H2S	-	**31**	URE	-	**48**	LDC	+
11	BNAG	+	**32**	dSOR	+	**53**	IHISA	-
12	AGLTp	-	**33**	SAC	+	**56**	CMT	-
13	dGLU	+	**34**	dTAG	-	**57**	BGUR	-
14	GGT	+	**35**	dTRE	+	**58**	O129R	+
15	OFF	+	**36**	CIT	+	**59**	GGAA	-
17	BGLU	+	**37**	MNT	+	**61**	IMLTa	-
18	dMAL	+	**39**	5KG	-	**62**	ELLM	-
19	dMAN	+	**40**	ILATk	+	**64**	ILATa	-
20	dMNE	+	**41**	AGLU	-			

*Full forms for the abbreviations as per the Vitek2 GN Card manual

Figure 1: Phylogenetic tree of *Kocuria arsenatis*_GXDRC_03 (GenBank accession: PX533098).

Table 3: Antimicrobial sensitivity of *Klebsiella pneumoniae*.

Antimicrobial	MIC* (µg/ml)	Interpretation	Antimicrobial	MIC* (µg/ml)	Interpretation
Amoxicillin/ Clavulanic Acid	≥32	R	Meropenem	≥16	R
Piperacillin/	≥128	R	Amikacin	32	R

Continued.

Antimicrobial	MIC* (µg/ml)	Interpretation	Antimicrobial	MIC* (µg/ml)	Interpretation
Tazobactam					
Cefuroxime	≥64	R	Gentamicin	8	R
Cefuroxime Axetil	≥64	R	Ciprofloxacin	≥4	R
Ceftriaxone	≥64	R	Tigecycline	4	I
Cefoperazone/ Sulbactam	≥64	R	Fosfomycin	≥16	S
Cefepime	≥32	R	Colistin	2	I
Ertapenem	≥8	R	Trimethoprim/ sulfamethoxazole	≥20	S
Imipenem	8	R			

*Minimum inhibitory concentration

Table 4: Biochemical tests* result *Staphylococcus haemolyticus*.

Characteristics	Biochemical test							
2	AMY	-	**25**	AGAL	-	**47**	NOVO	-
4	PIPLC	-	**26**	PyrA	+	**50**	NC6.5	+
5	dXYL	-	**27**	BGUR	-	**52**	dMAN	+
8	ADH1	+	**28**	AlaA	-	**53**	dMNE	-
9	BGAL	-	**29**	TyrA	-	**54**	MBdG	+
11	AGLU	-	**30**	dSOR	-	**56**	PUL	-
13	APPA	-	**31**	URE	-	**57**	dRAF	-
14	CDEX	-	**32**	POLYB		**58**	0129R	-
15	AspA	-	**37**	dGAL	+	**59**	SAL	-
16	BGAR	-	**38**	dRIB	+	**60**	SAC	+
17	AMAN	-	**39**	ILATK	+	**62**	dTRE	-
19	PHOS	-	**42**	LAC	+	**63**	ADH2s	+
20	LeuA	-	**44**	NAG	+	**64**	OPTO	+
23	ProA	-	**45**	dMAL	+			
24	BGURr	-	**46**	BACI	+			

*Full forms for the abbreviations as per the Vitek2 GN Card manual (https://www.biomerieux.com)

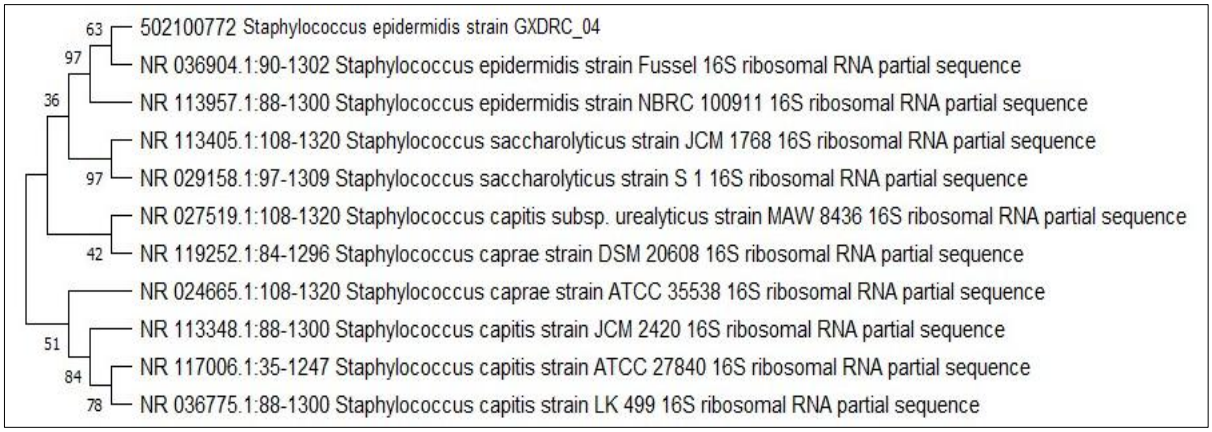

Figure 2: Phylogenetic tree of *Staphylococcus epidermidis*_GXDRC_04 (GenBank accession: PX533112).

Table 5: Antimicrobial sensitivity of *Staphylococcus haemolyticus*.

Antimicrobial	MIC* value (µg/ml)	Interpretation	Antimicrobial	MIC* value (µg/ml)	Interpretation
Cefoxitin screen	POS	+	Linezolid	4	S
Benzylpenicillin	≥0.5	R	Daptomycin	1	S
Oxacillin	≥4	R	Teicoplanin	2	S
Gentamicin	≥16	R	Vancomycin	1	S

Continued.

Antimicrobial	MIC* value (µg/ml)	Interpretation	Antimicrobial	MIC* value (µg/ml)	Interpretation
Ciprofloxacin	≥8	R	Tetracycline	≥1	S
Levofloxacin	≥8	R	Tigecycline	≥12	S
Erythromycin	≥8	R	Nitrofurantoin		
Inducible clindamycin resistance	NEG**	-	Rifampicin	≥0.03	S
Clindamycin	0.25	S	Trimethoprim/ sulfamethoxazole	≥10	S

*Minimum inhibitory concentration; ** NEG: Negative for inducible clindamycin resistance. This indicates the organism is susceptible to clindamycin, and resistance is not triggered by erythromycin.

DISCUSSION

Clinical manifestations of neonatal sepsis can range from subclinical infection to severe systemic disease that may include respiratory symptoms (e.g., apnea, tachypnea), cardiovascular signs (e.g., bradycardia, hypotension), gastrointestinal issues (e.g., vomiting, abdominal distention), skin changes (e.g., petechiae, purpura), and central nervous system symptoms (e.g., lethargy, irritability). Other signs included are nutritional intolerance and temperature regulation. The symptoms are often non-specific, making diagnosis challenging.[13] According to the present study, GBS was not detected in any of the cases. However, *Klebsiella pneumoniae*, *Staphylococcus haemolyticus, Kocuria arsenatis* GXDRC_03, and *Staphylococcus epidermidis* GXDRC_04 were isolated and identified from the neonatal sepsis samples.

K. pneumoniae is a gram-negative, non-motile bacillus that is typically encapsulated and belongs to the family *Enterobacteriaceae*. This organism is widely distributed in the environment and can be found in soil, water, plants, and sewage. It is also a part of the microbiome in the nasopharynx and gastrointestinal (GI) tract of healthy individuals. As an opportunistic pathogen, it can cause both hospital-acquired and community-acquired infections. When neonates inhale or swallow infected amniotic fluid, it can lead to the colonization of pathogenic *K. pneumoniae* in their gut, potentially resulting in intrapartum sepsis. Neonates admitted for longer stays in the NICU, or those receiving prolonged feeding through an enteral tube, are particularly prone to such colonization. Furthermore, molecular typing has revealed that about 50% of *K. pneumoniae* strains isolated from blood are identical to those found in the gut, and a possible link between gut colonization and neonatal sepsis.[17] From the 4578 samples, 378 blood cultures tested positive for *K. pneumoniae, S. epidermidis, S. haemolyticus*, and other bacterial species. The study on *K. pneumoniae* showed the highest susceptibility to amikacin, imipenem, and ciprofloxacin and resistance to oxacillin, tetracycline, and ciprofloxacin.[18] *K. pneumoniae* infections in 166 premature infants and 68 term infants showed that it had a high prevalence and poor treatment outcomes among premature infants, along with increased antibiotic resistance.[19] Although the World Health Organisation (WHO) still recommends ampicillin and gentamicin for treating neonatal sepsis, *K. pneumoniae* is quickly becoming resistant to these antibiotics, particularly in vulnerable populations and is undoubtedly identified as an organism that easily develops antibiotic resistance, harbours multiple plasmids, survives in the environment and within the human gut, and poses a significant threat in NICUs in low and middle-income countries (LMICs). The results of this study showed that *K. pneumoniae* was susceptible to fosfomycin and trimethoprim, and resistant to amoxicillin, piperacillin, cefuroxime, imipenem, meropenem, amikacin, and gentamicin.

Kocuria is a gram-positive coccus (pairs, short chains, tetrads, cubical packets of eight, and irregular clusters). It belongs to the phylum *Actinobacteria*, class *Actinobacteria*, order *Actinomycetales,* sub-order *Micrococcinae* and family *Micrococcaceae*. It has been reported to be present in the normal flora of the human skin and oral cavity, and is considered a laboratory contaminant. It is ignored when isolated from the clinical specimens, undermining its pathogenic potential. *Kocuria* (named as *Micrococcus kristinae*) was first reported in a case of urinary tract infection in 1974. Since then, reports of infection with *Kocuria* species have increased in the late twentieth century, and the trend continues, signifying its pathogenic potential. Infections associated with isolation of *Kocuria spp*. include urinary tract infections, cholecystitis, catheter-associated bacteremia, dacryocystitis, canaliculitis, keratitis, native valve endocarditis, peritonitis, descending necrotizing mediastinitis, brain abscess, and meningitis.[20] According to the results of the present study, phylogenetic analysis based on 16S rRNA gene sequences revealed that the isolate showed the highest sequence similarity of 99.75% to *Kocuria arsenatis* (strain CM1E1). Although K. *arsenatis* usually does not produce beta-hemolysis. To our knowledge, this is the first report of a beta-hemolytic *K. arsenatis* strain from a neonatal sepsis sample.

Coagulase-negative staphylococci (CoNS) are major skin commensals in humans, and almost 84% of healthy neonates' skin was colonized in a day.[21] LONS (approximately 50% cases) is commonly caused by CoNS (*S. epidermidis* and *S. haemolyticus*) in neonates with very low birth weight.[22] Recent evidence also links these bacteria to the pathogenesis of the immunologic skin

reaction erythema toxicum, frequently observed in healthy neonates.[23] The similarity of the 16S rRNA sequence is very high, 90% to 99% in *Staphylococcus* species.[24] Blood culture involving 1221 samples obtained from 1330 neonates with sepsis; 111 (9.1%) showed bacterial growth. Fifty-one (51) *staphylococcal* isolates were obtained, out of which 39 (76.5%) were identified as CoNS species and 12 (23.5%) as S. aureus. CoNS species were identified as *S. epidermidis, S. hominis, S. haemolyticus*, and *S. warneri* based on the SesC gene PCR and 16S rRNA sequencing. The frequency of NS caused by CoNS isolates was 35.1% (n=39/111). These CoNS isolates were resistant to methicillin and showed 100.0% antibiotic resistance to cefoxitin, ampicillin, erythromycin, and linezolid.[25] In our study, an isolate was identified as *S. epidermidis* based on 16S rRNA phylogenetic analysis. Recent studies on NS in Gujarat have shown the presence of *K. pneumoniae, Acinetobacter baumannii, E. coli, S. aureus, P. aeruginosa, Burkholderia spp., Streptococcus spp*., and other CoNS species in neonatal sepsis.[26-30] A retrospective study on LONS in neonates at a tertiary care center in central India showed that *Klebsiella* and *Acinetobacter spp*. were the most commonly isolated organisms.[3] *S. aureus, CoNS, E. coli*, and *K. pneumoniae* were isolated during a study on suspected cases of neonatal sepsis in a tertiary care hospital in western Uttar Pradesh, India.[31] A study was undertaken on a cohort of 100 neonates (50 pre-term and 50 term births) at a NICU in Northern India. Out of the 365 isolates: Gram-positive (n=241), *Coagulase Negative Staphylococcus* spp. (CoNS) (n=195) was the most common, followed by *Enterococcus spp*. (n=29) and *S. aureus* (n=17). Among the rest, 124 were *E. coli* (n=51), *K. pneumoniae* (n=43), and *Acinetobacter* species (n=25). *P. vulgaris, P. aeruginosa*, and *C. freundii* were also isolated.[32]

Thus, the present study provides new information on the identification of bacterial species causing neonatal sepsis. It also throws light on the other pathogens/bacteria that are not envisaged to be present in neonatal sepsis samples, and can grow in the selective LIM broth. This is the first report on the isolation of *K. arsenatis* from neonatal sepsis in Gujarat, India.

This study is based on enrichment of bacteria from neonatal sepsis swab samples in LIM broth, a selective medium for GBS. The limitation is that other isolates/bacteria present in the sample that are inhibited by the antibiotics present in the LIM broth cannot be isolated.

CONCLUSION

This study was undertaken to detect the presence of GBS and other bacteria causing neonatal sepsis in a NICU, Ahmedabad. GBS was not detected in the six (6) samples tested. However, *K. pneumoniae, S. haemolyticus, K. arsenatis,* and *S. epidermidis* were isolated from the samples and presumed to be the most likely causative agents in neonatal sepsis. The antimicrobial resistance pattern of *K. pneumoniae* and *S. haemolyticus* in this study could be useful for the control and treatment of sepsis in neonates. Thus, the study supports other studies that reported bacteria other than GBS causing bacterial infections in neonates. Moreover, our findings revealed that several bacterial species (opportunistic and emerging pathogens) can grow in LIM broth, a selective medium for the detection of GBS. GenBank accession nos. PX533098 and PX5331127, have been assigned to *Kocuria arsenatis* GXDRC_03 and *Staphylococcus epidermidis* GXDRC_04, respectively.

ACKNOWLEDGEMENTS

Authors would like to thank Niraj Sheth for the academic support to AM and new-borns at NICU for the samples. The authors also thank the colleagues who facilitated this work.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Fernando SM, Rochwerg B, Seely AJE. Consensus definitions for sepsis and septic. Can Med Assoc J. 2018;190(36):1058-59.
2. Jin Z, Wang Z, Li J, Yi L, Liu N, Luo L. Clinical laboratory features of microbes that cause neonatal sepsis: An 8-year retrospective study. Infect Drug Resist. 2022;15:2983-93.
3. Pataskar A, Chandel A, Jain M. Gram-negative Late-onset neonatal sepsis in a tertiary care center from Central India: A retrospective analysis. Clin Med Insights: Pediatr. 2023;17.
4. De Rose DU, Ronchetti MP, Martini L, Rechichi J, Iannetta M, Dotta A, et al. Diagnosis and management of neonatal bacterial sepsis: Current challenges and future perspectives. Trop Med Infect Dis. 2024;28(9):199.
5. Laxminarayan R, Duse A, Wattal C, Zaidi AK, Wertheim HF, Sumpradit N, et al. Antibiotic resistance-the need for global solutions. Lancet Infect Dis. 2013;13(12):1057-98.
6. Gabriel J, Escobar M. The Neonatal "Sepsis Work-up": Personal reflections on the development of an evidence-based approach toward newborn infections in a managed care organization. Pediatr. 1999;103:360-73.
7. Yadav AK, Nataraj S, Sharma R, Vishwanath P, Doddaiah N, Murthy S, Prashant A. Assessing the diagnostic potential of 16S rRNA gene for neonatal sepsis: A tertiary care hospital study in South India. Med J Armed Forces India. 2024;80(6):695-701.
8. Stefanovic IM. Neonatal sepsis. Biochem Med (Zagreb). 2011;21(3):276-81.
9. Healy CM, Baker CJ. Streptococcus Group B Vaccines. Plotkin's Vaccines. 2017;1046-151.
10. Stoll BJ, Hansen N, Fanaroff AA, Wright LL, Carlo WA, Ehrenkranz RA, et al. Late-onset sepsis in very

low birth weight neonates: the experience of the NICHD Neonatal Research Network. Pediatr. 2002;110(2,Pt1):285-91.
11. Fenta GM, Woldemariam HK, Metaferia Y, Seid A, Gebretsadik D. Admission outcome and antimicrobial resistance pattern of bacterial isolates among neonates with suspected sepsis in neonatal intensive care unit at Dessie Comprehensive Specialized Hospital, Dessie, Northeastern Ethiopia. Interdiscip Perspect Infect Dis. 2022(1):1318295.
12. Shah BA, Padbury JF. An old problem with new insights. Virulence. 2014;5(1):170-8.
13. Odabasi IO, Bulbul A. Neonatal sepsis. Sisli Etfal Hastanesi Tip Bulteni. 2020;54(2):142-58.
14. Mousavi SM, Hosseini SM, Mashouf RY, Arabestani MR. Identification of Group B streptococci using 16S rRNA, cfb, scpB, and atr genes in pregnant women by PCR. Acta Med Iran. 2016;54(12):765-70.
15. Lim DV, Morales WJ, Walsh AF. Lim Group B Strep Broth and coagglutination for rapid identification of Group B Streptococci in preterm pregnant women. J Clin Microbiol. 1987;25(2):452-3.
16. Tamura K, Stecher G, Kumar S. MEGA11: Molecular evolutionary genetics analysis version 11. Mol Biol Evol. 2021;38(7):3022-7.
17. Mukherjee S, Mitra S, Dutta S, Basu S. Neonatal Sepsis: The impact of carbapenem-resistant and hypervirulent Klebsiella pneumoniae. Front Med. 2021;8:634349.
18. Hosseini MB, Oskouei SA, Heidari F, Sadat SA. Salimi, Z, Sharif SAA. Determination of the frequency of microbial agents and drug susceptibility pattern of the neonatal sepsis in the neonatal intensive care unit at Alzahra hospital. Iran J Neonatol. 2019;10(4):4-11.
19. Wei X, Liang J, Zhang H, Yan C, Lu X, Chen Y, et al. Clinical features and risk factors of Klebsiella pneumoniae infection in premature infants: a retrospective cohort study. BMC Infect Dis. 2024;24(1):1311.
20. Kandi V, Palange P, Vaish R, Bhatti AB, Kale V. Emerging bacterial infection: Identification and clinical significance of Kocuria species, cultural characteristics of Kocuria. Cureus. 2016;8(8).
21. Marchini G, Nelson A, Edner J, Lonne-Rahm S, Stavréus-Evers A, Hultenby K. Erythema toxicum neonatorum is an innate immune response to commensal microbes penetrating the skin of the newborn infant. Pediatr Res. 2005;58(3):613-6.
22. Strunk T, Richmond P, Simmer K, Currie A, Levy O, Burgner D. Neonatal immune responses to coagulase-negative staphylococci. Curr Opin Infect Dis. 2007;20(4):370-5.
23. Nelson A, Hultenby K, Hell E, Riedel HM, Brismar H, Flock JI, et al. Staphylococcus epidermidis isolated from newborn infants express pilus-like structures and are inhibited by the cathelicidin-derived antimicrobial peptide LL37. Pediatr Res. 2009;66(2):174-8.
24. Enany S, Zakeer S, El A, Hosny DMS. Phylogenetic relationships among Staphylococcus epidermidis based on 16S rRNA gene sequence. Egypt J Med Microbiol. 2019;2(4);157-63.
25. Malakian A, Dehghan S, Montazeri EA, Aramesh MR. 16S rRNA – based molecular identification of coagulase-negative Staphylococcus species in neonates with sepsis and their antibiotic resistance patterns in Ahvaz, Iran. BMC Microbiol. 2025;25(1):114.
26. Ghedia BJ, Shah MR. Microbiological Profile of neonatal septicemia by automated blood culture system (BacT-ALERT) at a tertiary care hospital in Jamnagar, Gujarat, India. J Pharmaceut Res Int. 2021;33(59B):1722.
27. Gamit SC, Patel RD. Bacteriological profile and antibiotic susceptibility pattern of neonatal septicemia in a tertiary hospital in Vadodara, Gujarat. J Pure Appl Microbiol. 2022;16(1):638-42.
28. Chauhan H, Khokhar N, Patel P, Shrimali G, Patel K, Makwana N. Study of risk factors associated with neonatal septicemia and its bacteriological profile at one of the tertiary care hospitals of Gujarat, India. Healthline. 2023;14(2):117-22.
29. Gandhi DD, Vasava UG, Sonaiya DK. A prospective study on predictors of mortality in neonatal septicemia at a special newborn care unit (SNCU) of a tertiary care center of South Gujarat. J Popul Ther Clin Pharmacol. 2025;32(05):870-8.
30. Shah NS, Srivastava R, Mehta T, Shah A, Chandwani C. Study of etiological profile, risk factors, and immediate outcome of neonatal sepsis. Int J Contemp Pediatr. 2025;12(5):755-62.
31. Sethi K, Verma RK, Yadav RK, Singh DP, Singh S. A study on bacteriological profile in suspected cases of neonatal sepsis and its correlation with various biomarkers in the rural population of a university hospital. J Family Med Prim Care. 2023;12(10):2313-7.
32. Pandey R, Gupta V, Jain S, Gulati N. Skin colonization by pathogenic bacteria as a risk factor for neonatal sepsis. Indian J Med Microbiol. 2025;53:100782.

Cite this article as: Upasani VN, Memakiya AC, Patel AR, Patel RR, Savalia HJ, Jhala DD, et al. Bacteria identified in neonatal sepsis cases from a neonatal intensive care unit, Ahmedabad, India. Int J Contemp Pediatr 2026;13:491-7.