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Original Research Article

TO STUDY THE MICROFLORA IN NICU ADMITTED PATIENTS AT A TERTIARY CARE HOSPITAL OF RURAL HARYANA: A CROSS SECTIONAL STUDY

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ABSTRACT

Background: In developing countries neonatal sepsis is responsible for 30-50% of neonatal deaths making it the leading cause of neonatal mortality (1,2). National Neonatal Perinatal Database from 2002-03 puts sepsis as the cause of 19% of neonatal deaths in India. Neonatal sepsis has an incidence of 30 per 1000 live births (3). This study sought to describe the pattern of microflora and antibiotic sensitivity patterns of neonates with neonatal sepsis. It also evaluates the association of various septic neonate related variables on different outcomes of sepsis like mortality, hypoglycaemia, bleeding tendencies etc. **Materials and Methods:** This study was carried out in department of Paediatrics at Bhagat Phool Singh Govt Medical College, Haryana (India). Neonatal records were reviewed and patient related variables of 135 neonates diagnosed with sepsis were recorded. The qualitative variables were expressed as percentages. We used binomial logistic regression models to predict various sepsis related outcomes (death, hypoglycaemia, feed intolerance, delay in feed initiation and bleeding tendencies) based on independent variables (gender, gestational age, birth weight, mode of delivery, growth in cultures, type of organisms, abnormality in haematological parameters and duration of antibiotics). Wald's $\chi 2$ was used to assess the strength of associations while Negelkerke R2 to explain the variance in outcomes in the logistic regression models. Models with $p<0.05$ were considered statistically significant. **Result:** Out of the 135 neonates 52% were males and 48% females. 60.7 % (n=82) of the participants were preterm with 48 (35.6%) having birth weight between 1-1.5 kgs.75 (55.6%) had growth on blood cultures. Klebsiella 20 (15%) was the most common organism isolated in blood cultures. Deaths, hypoglycaemia, feed intolerance, delayed introduction of progressive enteral feeds and bleeding were reported in 54.8%, 7.4%, 28.1%, 68.1% and 40.7% of neonates with sepsis respectively. Female gender (p=0.018) and duration of antibiotic use of (15- 21 days vs <7days; p=0.003) showed a significant decrease in likelihood of mortality while feed intolerance increased its probability (p=0.001). Neonates with thrombocytopenia at the time of diagnosis of sepsis was a predictor (p=0.01) of bleeding tendencies. Females had a lower probability (p=0.01) of delayed introduction of progressive enteral feeds. Term neonates with sepsis had a delayed introduction of progressive enteral feeds (p=0.023). Mode of delivery (LSCS) was also a strong predictor (p=0.03) of hypoglycaemia in participants. **Conclusion:** Neonatal sepsis is the leading cause of neonatal mortality. A rise in antimicrobial resistance might further add to this burden. Hence aspects like antimicrobial resistance patterns and predictors of sepsis related outcomes need to further studied with more large scale multicentric studies.

INTRODUCTION

In India, neonatal sepsis occurs in 30 per 1000 live births and accounts for 19% of all neonatal deaths (NNPD).[1-3] Despite having access to suitable medications, sepsis-related mortality has remained largely stable. Concerns about antimicrobial resistance in neonatal sepsis are increasing, supported by data from 64 studies across India. These studies report a case fatality rate of 34.4%, with Klebsiella sp. (53.6%) and Escherichia coli (42.2%) as the most frequently isolated organisms. The most common Gram-positive bacterium isolated was Staphylococcus aureus (43.2%). The studies also reveal a rising pattern of antimicrobial resistance, showing high resistance to the' access group'

antibiotics in common hospital pathogens. However, most pathogens remain susceptible to watch group" antibiotics like meropenem. Additionally, most Staphylococcus aureus isolates are still sensitive to vancomycin.[4] Neonatal sepsis has multiple short- and long-term consequences. Short-term outcomes include mortality, morbidity such as hypoglycaemia, feed intolerance, delays in starting enteral feeding, and issues related to haematological and other organ systems. Studies have also examined treatment failure, duration of antibiotic use, and changes in antibiotic therapy. However, few have explored the long-term neurodevelopmental effects of sepsis.[5] This study seeks to characterise the microbial profile and antimicrobial resistance patterns in neonates with sepsis. Additionally, it assesses neonatal variables and their impact on different sepsis outcomes, as well as how maternal factors influence the likelihood of neonatal sepsis.

MATERIALS AND METHODS

Study design: This retrospective cross-sectional study collected and analysed medical records of all neonates diagnosed with neonatal sepsis admitted to BPSGMCW NICU ensuring complete anonymity.

Operational definitions

1. Culture-positive sepsis refers to neonates suspected of sepsis based on clinical features or maternal or perinatal risk factors, with treatment involving appropriate type and duration of antibiotic therapy and a recognised pathogen isolated from blood. Cases of sepsis with a positive culture for coagulase-negative staphylococci were labelled only if the clinical course suggested sepsis and appropriate antibiotic therapy was given.[6]
2. Culture-negative sepsis was identified when the clinical presentation indicated sepsis or the sepsis screen was positive, but no pathogen was isolated, or a blood culture was not performed.[6]
3. Neonatal hypoglycemia is defined as blood glucose levels below 45 mg/dl.[7]
4. Feeding intolerance was characterised by at least one of the following: increased abdominal girth from baseline, vomiting, pre-feed residuals exceeding 30% of the feeding volume, or bloody stools.[8]
5. Delayed introduction of progressive enteral feeds refers to starting enteral feeding more than 4 days after birth.[9]

Variables: Data were gathered and documented for the specified neonatal and maternal variables.

1. Neonatal variables

A. Independent variables include gender, mode of delivery, gestational age, birth weight, place of birth (inborn/outborn), duration of antibiotic therapy, Haemoglobin levels, total leukocyte count, and platelet count at the time of sepsis diagnosis.

B. Dependent variables include mortality, bleeding, feed intolerance, delayed introduction of progressive enteral feeds, and hypoglycemia.

2. Mother-related variables

A. Dependent variable: culture-positive neonatal sepsis.

B. Independent variables include: presence of meconium-stained liquor, gestational age, induction of labour, mode of delivery, prolonged rupture of membranes (PROM) exceeding 18 hours, and the need for resuscitation during delivery.

Data collection, processing and analysis: Medical records of neonates diagnosed with sepsis were reviewed. Sepsis diagnosis followed departmental protocols adapted from standard guidelines.[4] Relevant variable data were entered into an Excel sheet and coded for analysis. We checked the data for completeness, correcting or removing missing entries and duplicates. We then transferred the cleaned data to SPSS version 20 for statistical analysis. We presented qualitative data for maternal and neonatal variables as proportions (percentages). We assessed the impact of each independent variable as a risk factor on neonatal outcomes using binary logistic regression. Similarly, we analysed the predictive effect of maternal variables on culture-positive neonatal sepsis using comparable models. We evaluated each model fit using the Hosmer-Lemeshow test. Results were considered statistically significant if the p value was less than 0.05.

RESULTS

Participant flow: During the study, our centre recorded 4,215 live births. Of these, 8.9 % (378 neonates) were admitted to the NICU. Among the NICU admissions, 47.6 % (180 neonates) were diagnosed with sepsis. Complete data were available for 135 of these 180 neonates, categorised as culture-positive (75 cases) and culture-negative sepsis (60 cases), based on blood culture pathogen isolation. The neonatal variables for these 135 neonates are summarised in [Table 1].

Descriptive statistics:

Table 1: Distribution of neonatal variables

Neonatal Variables	Category	n (%)
Sex	male	70(51.9%)
	female	65(48.1%)
Mode of delivery	Normal vaginal delivery	110(81.5%)
	LSCS	25(18.5%)
Gestation	Preterm	82(60.7%)
	Term	53(39.3%)
Birth weight	>1000gms	9(6.7%)
	1000 to 1500 gms	48(35.6%)

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	1501 to 2500 gms	54(40%)
	>2500 gms	24(17.8%)
Organisms isolated on culture	Fungal	10(7.4%)
	Gram negative bacteria	52(38.5%)
	Gram positive bacteria	13(9.6%)
	none	60(44.4%)
Sepsis	Culture positive sepsis	75(55.6%)
	Culture negative sepsis	60(44.4%)
Place of birth	Inborn	77(57%)
	Outborn	58(43%)
Mortality	No	61(45.2%)
	Yes	74(54.8%)
Delayed introduction of progressive enteral feeds	No	43(31.9%)
	Yes	92(68.1%)
Feed intolerance	No	39(28.9%)
	Yes	38(28.1%)
	Feed not initiated.	58(43%)
Duration of antibiotic	0 to 7 days	44(32.6%)
	8 to 14 days	46(34.1%)
	15 to 21 days	33(24.4%)
	More than 21 days	12(8.9%)
Hemoglobin at the time of diagnosis of sepsis	≤10 gm/dl	10(7.4%)
	>10 gm/dl	125(92.6%)
Leucocyte count at time of diagnosis of sepsis	5000 to 15000 cells/cmm	80(59.3%)
	>15000cells/cmm or <5000cells/cmm	55(40.7%)
Platelets at the time of diagnosis of sepsis	≥1.5 lac /cmm	22(16.3%)
	<1.5 lac/cmm	113(83.7%)
Bleeding	Absent	80(59.3%)
	Present	55(40.7%)
Blood component administration	No	80(59.3%)
	Yes	55(40.7%)
Hypoglycaemia	No	125(92.6%)
	Yes	10(7.4%)

Out of 75 neonates diagnosed with culture-positive sepsis, antenatal records were available for 69, as shown in [Table 2].

Table 2: Distribution of maternal variables.

Maternal Variables	Category	n (%)
Prolonged rupture of membrane >18 hrs	No	17(24.6%)
	Yes	52(75.4%)
Presence of meconium-stained liquor	No	50(72.5%)
	Yes	19(27.5%)
Induction of labour	No	24(34.8%)
	Yes	45(65.2%)
Breech presentation	No	61(88.4%)
	Yes	8(11.6%)
Need for resuscitation during delivery.	No	51(73.9%)
	Yes	18(26.1%)
Presence of foul-smelling liquor.	No	57(82.6%)
	Yes	12(17.4%)
Neonate diagnosed with sepsis after birth	No	16(23.2%)
	Yes	53(76.8%)

[Table 3 and Table 4] illustrate the types of isolates along with their antimicrobial sensitivity and resistance patterns.

Table 3: Gram-negative isolates

Sensitivity pattern of isolates of Gram-negative organisms.				
Antimicrobials	**Klebsiella (n=20)**	**Citrobacter (n=16)**	**Acinetobacter(n=10)**	**Enterobacter(n=6)**
Colistin	ND	94% (15)	50% (5)	83% (5)
Meropenem	90% (18)	75% (12)	40% (4)	50% (3)
Piperacillin -tazobactam	50% (10)	12% (2)	60% (6)	33% (2)
cefotaxime	25% (5)	ND	40% (4)	ND
Gentamicin	50% (10)	44% (7)	30% (3)	25% (2)
Amikacin	50% (10)	25% (4)	20% (2)	50% (3)
Ciprofloxacin	25% (5)	50% (8)	10% (1)	25% (2)
Tigecycline	ND	25% (4)	10% (1)	ND
Polymyxin	ND	ND	20% (2)	ND
Resistance pattern of isolates of Gram-negative organisms.				
Doxycycline	67% (8)	50% (8)	ND	66% (4)

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Cefotaxime	50% (6)	38% (6)	ND	ND
ceftazidime	17% (2)	12% (2)	ND	50% (3)
meropenam	5% (1)	19% (3)	30% (3)	33% (2)
ciprofloxacin	25% (3)	19% (3)	30% (3)	ND
gentamicin	17% (2)	38% (6)	20% (2)	ND

Table 4: Gram-positive isolates

Sensitivity pattern of Gram-positive organisms		
Antimicrobials	**Staphylococcus aureus (n=10)**	**coagulase negative Staphylococcus aureus (n=3)**
Linezolid	100%(10)	100%(3)
Vancomycin	50%(5)	100%(3)
Cefotaxime	40%(4)	100%(3)
Gentamicin	40%(4)	ND
Ofloxacin	30%(3)	ND
Doxycycline	20%(2)	ND
Ampicillin	ND	100%(3)
Resistance pattern of Gram-positive organisms.		
Ciprofloxacin	60%(6)	100%(3)
Doxycycline	40%(4)	ND
Cefotaxime	20%(2)	ND
Vancomycin	20%(2)	ND
Meropenem	ND	33%(1)

We identified Candida albicans isolates in 10 blood cultures from patients with sepsis. All strains showed sensitivity to fluconazole and amphotericin B.

Inferential statistics:

We developed logistic regression models, as shown in [Table 5], to evaluate how independent variables influence the dependent variable.

A) Effect of neonatal variables on:

1. A statistically significant logistic regression model ($\chi2 = 37.13$, $p = 0.005$) examined the link between neonatal variables and mortality. It explained 52.2% of the variance (Nagelkerke R^2) and correctly classified 74.7% of cases. The analysis showed that female gender and antibiotic treatment lasting over 7 days significantly reduced mortality risk, whereas feed intolerance increased it. Other factors- such as gestational age, birth weight, cultured organisms, place of birth, hypoglycaemia, delivery mode, bleeding, thrombocytopenia, and anaemia at diagnosis- had no impact on mortality.

2. The likelihood of bleeding in culture-positive septic neonates was higher among those with Gram-positive cultures and thrombocytopenia at diagnosis. The regression model was statistically significant ($\chi2(17) = 36.97$, $p = 0.003$), explained 52.8% of the variance (Nagelkerke R2) in bleeding occurrence, and accurately classified 76% of cases. No significant association was found between bleeding risk and gender, gestational age, hypoglycaemia, delivery mode, delayed enteral feeding, anaemia, or leucocyte count at sepsis diagnosis.

3. The delayed start of progressive enteral feeds was analysed using a regression model that was statistically significant, $\chi2 = 54.73$, $p < 0.001$. This model explained 71.9 % of the variance and correctly classified 88% of cases. It showed that males, preterm infants, and out-born septic neonates are at a higher risk of delay. Antibiotic duration, birth weight, feed intolerance, organism type, hypoglycaemia, and other haematological parameters did not significantly affect the risk of delayed feeds.

Table 5: logistic regression analysis for different outcomes of neonatal sepsis

Logistic regression analysis on the association of culture-positive septic neonate-related variables with mortality								**95% C.I.for EXP(B)**	
S.No	**Variables**		**β**	**SE β**	**Wald's $\chi2$**	**p**	**eβ (odds ratio)**	**Lower**	**Upper**
1	Gender	female (vs male)	-1.744	0.735	5.637	0.018	0.175	0.041	0.738
2	Duration of antibiotic use.					0.028			
		8- 14 days (vs <7 days.)	-3.379	1.289	6.875	0.009	0.034	0.003	0.426
		15 - 21 days. (vs <7 days.)	-3.96	1.317	9.042	0.003	0.019	0.001	0.252
		>21 days. (vs <7 days.)	-3.321	1.47	5.103	0.024	0.036	0.002	0.644
3	Feed intolerance	Feed intolerance (vs no feed intolerance)	2.708	0.832	10.603	0.001	15	2.939	76.561
Logistic regression analysis of the association between culture-positive septic neonate-related variables and bleeding.									
1	Platelet count at the time of diagnosis of sepsis.	Thrombocytopenia at diagnosis of sepsis (vs normal platelet count at diagnosis of sepsis)	4.01	1.583	6.417	0.011	5.156	2.478	127.805
2	Type of organism.					0.027			
		Fungal (vs Gram-negative bacteria)	-1.645	1.093	2.267	0.13	0.193	0.23	1.63

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		Gram-positive bacteria (vs Gram-negative bacteria)	1.363	0.288	3.675	0.04	3.908	1.059	14.426
Logistic regression analysis of the association between culture-positive septic neonate-related variables and delayed introduction of progressive enteral feeds.									
1	Gender	Female (vs male)	-3.711	1.501	6.108	0.013	0.024	0.001	0.464
2	Gestation	Term (vs preterm)	-3.82	1.676	5.196	0.023	0.022	0.001	0.585
3	Location of birth	outborn (vs inborn)	2.218	1.005	4.868	0.027	9.193	1.281	65.961
Logistic regression analysis of the association of culture-positive septic neonate-related variables with feed intolerance.									
1	Gender	female (vs male)	-2.094	0.872	5.763	0.016	0.123	0.022	0.681
2	Platelet count at the time of diagnosis of sepsis.	Thrombocytopenia at diagnosis of sepsis (vs normal platelet count at diagnosis of sepsis)	2.204	1.01	4.761	0.029	9.057	1.251	65.543
Logistic regression analysis on the association of culture-positive septic neonate-related variables with hypoglycaemia.									
1	Mode of delivery	LSCS (vs normal delivery)	2.286	1.055	4.694	0.03	9.836	1.244	77.795
Logistic regression analysis on the association of maternal variables with culture-positive sepsis in neonates.									
s.no	**Variables**		**β**	**SE β**	**Wald's $\chi 2$**	**p**	**eβ (odds ratio)**	**Lower**	**Upper**
1	Prolonged rupture of membrane ≥ 18 hrs	Prolonged rupture of membrane ≥ 18 hrs (vs no Prolonged rupture of membrane)	2.48	0.76	10.45	0.001	11.95	2.65	52.78

4. The logistic regression model analyzing patient variables' impact on hypoglycemia was statistically significant, $\chi 2(4) = 9.73$, $p < 0.05$. It accounted for 33.1% (Nagelkerke R2) of the variance in hypoglycemia occurrence and accurately classified 90.6% of cases. Septic neonates born via LSCS had 9.8 times higher odds of experiencing hypoglycemia compared to those born through normal delivery.

5. A regression model examining feed intolerance in septic neonates was statistically significant, with $\chi 2 = 17.01$, $p < 0.05$. It accounted for 36.6% of the variance (Nagelkerke R2) and correctly classified 69.8% of cases. The analysis showed that male neonates and those with thrombocytopenia at sepsis diagnosis had a higher risk of feed intolerance. Conversely, factors like birth weight, gestational age, place of birth, and haematological parameters did not significantly affect the likelihood of feed intolerance in these neonates.

B) Effect of maternal variables on probability of neonates having culture-positive sepsis-

The effects of maternal factors such as prolonged rupture of membrane for ≥18 hours, meconium-stained liquor, induction of labour, resuscitation at birth, mode of delivery, and gestational age less than 37 weeks on the likelihood of neonates being diagnosed with culture-positive sepsis were analysed using a logistic regression model [Table 5]. The model was statistically significant ($\chi 2(6) = 16.27$, $p = 0.012$) and explained 31.8% of the variance. It showed that neonates born to mothers with PROM ≥18 hours had an 11.95 times higher chance of being diagnosed with culture-positive sepsis. Other maternal factors did not significantly influence this risk.

DISCUSSION

The present study found a predominance (69.3%) of gram-negative organisms in blood cultures, with Klebsiella species (n=20, 27%), Citrobacter (n=16, 21.3%), and Acinetobacter (n=10, 13.5%) being the most common. The most frequent Gram-positive organism was Staphylococcus aureus (n=10, 13.5%), followed by coagulase-negative Staphylococcus (n=3, 4%). Candida albicans was also isolated in 10 cases (13.5%). These results align with a large-scale meta-analysis of 24273 isolates from hospitals in Southeast Asia, where gram-negative organisms accounted for 63% of neonatal sepsis pathogens. The top isolates in that study were Klebsiella species (23%), Escherichia coli (14%), and Acinetobacter species (8%), with Staphylococcus aureus (20%) and CONS (9%) being the most common gram-positive isolates.[4]

In 2019, a comprehensive meta-analysis examined antimicrobial resistance patterns in neonatal sepsis across Southeast Asia. It found that over half of the gram-negative bacteria were susceptible to antibiotics listed as "watch group" by WHO, including carbapenems, piperacillin-tazobactam, and fluoroquinolones. Less than a third of these organisms showed resistance to these drugs. Additionally, more than two-thirds of the isolates from Southeast Asia were sensitive to "reserve group' antibiotics like colistin. For Gram-positive bacteria such as Staphylococcus aureus, resistance to 'watch group' antibiotics like vancomycin was 19.1%, while resistance to 'reserve group' drugs like linezolid was only 2.8%.[4] Our observations align with these findings, with Gram-negative bacteria showing 50-90% sensitivity to 'watch group' antibiotics (see TABLE 3). Vancomycin resistance in S. aureus was approximately 20%, with nearly all isolates remaining sensitive to linezolid.

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The mortality rate for all neonates diagnosed with sepsis in this study was 54% (74 out of 135). The case fatality rate for culture-proven sepsis was 45% (34 out of 75), slightly higher than the median rate of 34.4% in India (n=14; 95% CI = 33-35.7%).[4] Mortality rates across South Asia vary widely, ranging from 16% to 72%,[10] likely due to differences in socioeconomic status, racial factors, and geography.

The current study also evaluated predictors of mortality and various morbidity outcomes in neonatal sepsis. We found that male gender, shorter duration of antibiotic therapy, and feed intolerance were significant predictors of mortality [Table 5]. Meshram et al. also reported a similar male preponderance for mortality ($p<0.05$). To our knowledge, the latter two factors have not been previously studied. Although previous research identified birth weight, gestational age, place of birth, type of organism on culture, bleeding, hypoglycaemia, and thrombocytopenia as predictors of mortality in culture-proven sepsis, our regression analysis did not find these factors to be significant.[10-13]

Neonates with sepsis frequently experience low platelet counts. The risk of bleeding in these infants is multifactorial and cannot be solely attributed to thrombocytopenia.[14] However, low platelet count is a significant risk factor, with an odds ratio of 2.504 ($p=0.017$) for bleeding in neonates.[15] In this study, thrombocytopenia was confirmed as a risk factor for bleeding in septic neonates (OR=5.15; $p=0.01$). Additionally, the bleeding risk was notably higher in neonates with gram-positive sepsis compared to those with gram-negative sepsis (OR=3.9; $p=0.04$). Refer to [Table 5] for more details. Other studies have reported similar findings.

In our study, thrombocytopenia was linked to feed intolerance in septic neonates ($p=0.029$, [Table 5]). Feed intolerance occurred in 60% (25/42) of neonates with low platelet counts, compared to only 18% (2/11) in those with normal counts. Alhamad et al also noted a similar link between sepsis-related feed intolerance and low platelet levels ($p<0.001$). This may be because feed intolerance is commonly seen in neonatal sepsis,[16] which is also associated with hematological changes like thrombocytopenia.[17-19]

Preterm and low birth weight neonates with sepsis often experience longer durations of parenteral nutrition, delayed start of enteral feeds, and take more time to reach full enteral feeding. This is likely due to concerns about feed intolerance and the higher risk of necrotising enterocolitis.[20] In this study, prematurity ($p=0.023$), male gender ($p=0.013$), and being outborn ($p=0.027$) were identified as risk factors for delayed initiation of enteral feeds in septic neonates.

There are limited studies linking caesarean section with neonatal hypoglycemia. In 2019, Turner et al. identified LSCS as a risk factor for neonatal hypoglycemia ($p<0.005$).[21] In this study, septic neonates born via LSCS had a 9.8 times higher risk ($p=0.03$) of hypoglycemia compared to those delivered vaginally. One possible explanation is reflex hypoglycemia, which can occur in hyperglycemic conditions such as gestational diabetes mellitus. During LSCS, mothers' hyperglycemic state may result from preoperative administration of dextrose-containing fluids.[22]

We also examined maternal factors as potential risk factors for culture-positive neonatal sepsis. We identified premature rupture of membranes (PROM) lasting more than 18 hours as a significant risk factor. The risk of developing culture-positive neonatal sepsis is 11.95 times higher ($p=0.03$). Although factors such as PROM, meconium-stained liquor, resuscitation at birth, and gestational age ≤ 37 weeks have been identified as risk factors in previous studies,[7,11,23,24] this study did not find these factors to increase the risk of sepsis.

This study details the organism profile and antimicrobial sensitivity patterns at a rural tertiary care hospital in India. It also examines how neonatal variables impact multiple neonatal sepsis outcomes, such as mortality, delayed introduction of progressive enteral feeds, feed intolerance, bleeding, and hypoglycemia. Maternal factors as risks for neonatal sepsis were assessed as well. The broad scope of analysis in a single study strengthens it. However, the study's single-centre design limits the generalizability of its findings.

CONCLUSION

Neonatal sepsis is the leading cause of neonatal mortality. A rise in antimicrobial resistance might further add to this burden.This study underscores the need for local antibiogram guided empirical therapy, vigilant monitoring of high risk neonates and targeted maternal neonatal interventions to reduce mortality and morbidity due to neonatal sepsis .Hence aspects like antimicrobial resistance patterns and predictors of sepsis related outcomes need to be further studied with more large scale multicentric studies.

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