Section: Miscellaneous

Original Research Article

PATHOGEN PROFILE, ANTIMICROBIAL RESISTANCE AND CUTANEOUS MANIFESTATIONS IN SUSPECTED PAEDIATRIC BLOODSTREAM INFECTION

Shiyas K P[1], Mohammed Faisal A[2], Prasanth G[3], Bijesh P.K[4]

[1]Professor, Department of Paediatrics, Al-Azhar Medical College, Thodupuzha, Kerala, India.
[2]Professor, Department of Dermatology, Travancore Medical College, Kollam, Kerala, India.
[3]Associate Professor, Department of Microbiology, Al-Azhar Medical College, Thodupuzha, Kerala, India.
[4]Assistant Professor, Department of Microbiology, Plus Max Medical College, Salem, Tamil Nadu, India.

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Corresponding Author:
Dr. Bijesh P.K,
Assistant Professor, Department of Microbiology, Plus Max Medical College, Salem, Tamil Nadu, India.
Email: bijeshkrishna1978@gmail.com

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ABSTRACT

Background: Bloodstream infection (BSI) is a leading contributor to avoidable paediatric mortality, and empirical prescribing is increasingly constrained by antimicrobial resistance. Cutaneous signs are seldom co-recorded with microbiological data, so their bedside value is poorly quantified.
Objectives: To characterise pathogen distribution, resistance phenotypes, cutaneous manifestations and outcomes across clinical syndromes in children with suspected BSI.
Materials and Methods: We analysed 105 consecutive paediatric episodes of suspected BSI at a tertiary hospital, reported per STROBE. Isolates were adjudicated as true pathogen, contaminant or no growth, and classified as susceptible, multidrug-resistant (MDR) or extensively drug-resistant (XDR). Comparisons used chi-squared or Fisher exact tests and Kruskal–Wallis or Mann–Whitney U tests; odds ratios with 95% confidence intervals were derived. $P < 0.05$ was significant.
Results: Cultures were positive in 63 of 105 episodes (60.0%); a true pathogen was adjudicated in 53 (50.5%). Gram-negative organisms predominated (39/53, 73.6%). MDR or XDR phenotypes accounted for 24 of 53 isolates (45.3%), highest in *Escherichia coli* (9/11, 81.8%) and *Klebsiella pneumoniae* (7/10, 70.0%); all Salmonella *Salmonella* Typhi isolates were ciprofloxacin-resistant but susceptible to ceftriaxone and azithromycin. Overall ciprofloxacin resistance reached 71.8%; amikacin, vancomycin and linezolid retained full activity. Cutaneous findings occurred in 17 episodes (16.2%), mostly meningitis (8/18, 44.4%; $P = 0.004$). Mortality was 13 of 105 (12.4%). Among culture-confirmed episodes, MDR/XDR was associated with death (6/24 versus 1/29; $P = 0.038$).
Conclusions: Gram-negative pathogens and MDR/XDR phenotypes dominated, and every Salmonella Typhi isolate was fluoroquinolone-resistant though non-MDR. Cutaneous findings were syndrome-dependent but unrelated to mortality. Syndrome-stratified local antibiograms may support more rational empirical prescribing.
Keywords: Bacteraemia; Child; Drug Resistance, Multiple, Bacterial; Skin Manifestations; Anti-Bacterial Agents.

INTRODUCTION

Bloodstream infection in children carries a mortality burden that remains stubbornly resistant to improvement in much of South Asia.[1,2] The clinical problem is not principally one of diagnosis. It is one of timing. A child with fever, tachycardia and poor perfusion is recognisable within minutes, yet the organism responsible — and its susceptibility profile — will not be known for 24 to 72 hours, during which the clinician must commit to an empirical regimen on probabilistic grounds alone.[3]

That probabilistic reasoning depends on local surveillance data, and such data are unevenly distributed. Much of the published paediatric BSI literature from India derives from neonatal units or from oncology and intensive care populations, where central venous access, prolonged admission and prior antimicrobial exposure select for a distinct and unrepresentative flora.[4,5] General paediatric wards, which admit the larger share of febrile children, are comparatively under-reported. Aggregated national antibiograms partly compensate, but they obscure the syndrome-level variation that actually drives bedside decisions: the organism spectrum in suspected meningitis differs materially from that in suspected enteric fever, and a single institutional resistance percentage cannot inform both.[6]

A second limitation concerns the denominator. Blood culture sensitivity in children is constrained by low circulating bacterial loads and by small drawn volumes, and pre-culture antimicrobial exposure is common in settings with accessible over-the-counter dispensing.[7,8] Studies that report only culture-positive episodes therefore describe a filtered population and may overstate the applicability of their findings to the child in front of the clinician, whose culture has not yet returned and may never become positive.

Cutaneous signs represent a third, largely unexploited source of bedside information. Petechiae, purpura, ecthyma gangrenosum and pustular lesions have recognised associations with specific organisms and with disease severity, and unlike culture they are available immediately.[9,10] Systematic co-recording of dermatological morphology with microbiological outcome is nevertheless uncommon in paediatric BSI cohorts, and the incremental value of such observation remains largely unquantified.

We therefore examined a consecutive cohort of children investigated for suspected BSI at a tertiary teaching hospital, retaining culture-negative episodes in the clinical analyses. Our objectives were threefold: to describe pathogen distribution and resistance phenotypes across six clinical syndromes; to characterise cutaneous manifestations and their syndrome and organism associations; and to identify factors associated with in-hospital mortality.

MATERIALS AND METHODS

Study Design and Setting

This was an observational study of consecutive paediatric episodes of clinically suspected bloodstream infection managed at a tertiary teaching hospital. Reporting adheres to the STROBE statement for observational studies.[11] The study was conducted jointly by the Departments of Microbiology, Paediatrics and Dermatology.

Participants

Children aged one month to 12 years admitted with a clinical syndrome prompting blood culture were eligible. Episodes were assigned by the treating paediatrician to one of six syndromic categories at admission — sepsis, enteric fever, urinary tract infection, pneumonia, meningitis or febrile neutropenia — on clinical and supporting laboratory grounds, before culture results were available. Syndrome assignment was therefore independent of microbiological outcome. Each episode constituted the unit of analysis. Culture-negative episodes were retained throughout the clinical, biochemical and outcome analyses; isolate-level analyses were necessarily restricted to episodes yielding a true pathogen.

Microbiological Methods

Blood was collected under aseptic precautions before commencement of antimicrobial therapy where clinically feasible, and inoculated into paediatric culture bottles. Biomeriux Bact/Alert PF PLUS and incubated in Bact/ Alert Virtuo automated blood culture system for 5 days. Bottles flagged as positive underwent Gram staining, subculture and identification by VITEK MS MALDI TOF. Susceptibility testing was performed on VITEK 2 system. Antimicrobial susceptibility was determined and interpreted according to contemporaneous CLSI breakpoints.[12]

Growth was adjudicated as a true pathogen, a contaminant, or no growth by consensus between the microbiologist and the treating paediatrician, taking into account organism identity, number of positive bottles, time to positivity and clinical coherence. Coagulase-negative staphylococci and other common skin commensals recovered from a single bottle without a compatible clinical picture were classified as contaminants. Isolates were categorised as multidrug-resistant (MDR) or extensively drug-resistant (XDR) using the standardised international definitions of Magiorakos and colleagues.[13] Isolates not meeting either definition were designated susceptible.

Dermatological Assessment

Skin was examined at admission and daily thereafter by the treating team. Where cutaneous findings were present, morphology was categorised by a dermatologist as petechial, purpuric, maculopapular, pustular, cellulitic or necrotic, and the interval between lesion onset and fever onset was recorded in days. Formal dermatology consultation was obtained at the discretion of the treating paediatrician rather than by protocol, and this should be borne in mind when interpreting the consultation figures.

Variables and Outcomes

Recorded exposures comprised demographic variables, weight-for-age Z score, underlying condition, immunosuppression, admitting unit, onset category (community-acquired or healthcare-associated), central venous access, and antimicrobial exposure within the preceding 30 days. Laboratory

variables included C-reactive protein, procalcitonin, total leucocyte count, neutrophil percentage, platelet count, lactate, creatinine and transaminases. The primary outcome was in-hospital mortality. Secondary outcomes were intensive care admission and length of stay. Empirical therapy was classified as appropriate when the administered regimen included at least one agent to which the eventual isolate was susceptible in vitro.

Statistical Analysis

Categorical variables are presented as counts with percentages and continuous variables as medians with interquartile ranges (IQR), distributions having been assessed for normality. Comparisons across the six syndromes used the Kruskal–Walli's test for continuous variables and the chi-squared or Fisher exact test for categorical variables, as appropriate to expected cell counts. Survivors and non-survivors were compared using the Mann–Whitney U test and the Fisher exact test. Odds ratios with 95% confidence intervals were calculated for categorical mortality associations. Susceptibility denominators include only isolates for which the relevant agent was tested; agents tested in fewer than 10 isolates are reported but were not subjected to inferential comparison. Analyses were performed using SPSS v25. Two-sided *P* values below 0.05 were considered statistically significant. Exact *P* values are reported throughout.

Two analytical decisions warrant explicit statement. First, the association between resistance phenotype and mortality was assessed within the 53 culture-confirmed episodes rather than across the full cohort, because episodes without an isolate have no resistance phenotype and cannot legitimately serve as an unexposed comparator. Second, the febrile neutropenia stratum comprised only two episodes; it is retained in descriptive tables for completeness but no inference is drawn from it, and syndrome-level comparisons were verified with this stratum excluded.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee. The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from a parent or legal guardian, with assent from children aged over seven years. Patient anonymity was preserved throughout; no identifying information appears in this report.

RESULTS

Cohort Characteristics

Of the episodes screened, 105 met eligibility criteria and were analysed (Figure 1). The cohort was evenly divided by sex (53 male, 50.5%). Median age was 76.0 months (IQR 44.0–111.0), with 69 children (65.7%) aged five years or older and eight (7.6%) infants. Sepsis and pneumonia were the commonest presenting syndromes (23 episodes each, 21.9%), followed by enteric fever (20, 19.0%), urinary tract infection (19, 18.1%), meningitis (18, 17.1%) and febrile neutropenia (two, 1.9%). Thirty-seven children (35.2%) were admitted directly to the paediatric intensive care unit.

An underlying condition was present in 30 children (28.6%), most frequently nephrotic syndrome (nine) and neuromuscular disorder (six). Fifteen children (14.3%) were immunosuppressed. Onset was community-acquired in 80 episodes (76.2%). A central venous catheter was in situ in 12 (11.4%), and 47 children (44.8%) had received antimicrobials within the preceding 30 days — a proportion that bears directly on the culture yield reported below. Clinical and laboratory characteristics stratified by syndrome are presented in Table 1.

Inflammatory and perfusion markers separated the syndromes clearly. Procalcitonin differed most ($P< 0.001$), with sepsis reaching a median of 8.52 ng/mL (IQR 7.03–10.36) against 2.80 ng/mL (IQR 1.88–4.09) in enteric fever. C-reactive protein ($P = 0.001$), lactate ($P = 0.006$), creatinine ($P< 0.001$) and length of stay ($P< 0.001$) likewise varied by syndrome. Age, weight, weight-for-age Z score and total leucocyte count did not (all $P> 0.05$). Excluding the two-episode febrile neutropenia stratum did not alter any of these conclusions.

Culture Yield and Pathogen Distribution

Blood cultures were positive in 63 of 105 episodes (60.0%), and a true pathogen was adjudicated in 53 (50.5% of the cohort; 84.1% of positive cultures). Ten positive cultures (15.9%) were adjudicated as contaminants or no true isolate. Culture yield was highest in meningitis (13/18, 72.2%) and lowest in urinary tract infection (8/19, 42.1%), though the difference across syndromes did not reach significance ($P = 0.415$).

Gram-negative organisms accounted for 39 of 53 isolates (73.6%) and Gram-positive for 14 (26.4%) (Figure 2). *Staphylococcus aureus*, *Salmonella* Typhi and *Escherichia coli* were each recovered 11 times (20.8%), followed by *Klebsiella pneumoniae* (10, 18.9%), *Pseudomonas aeruginosa* (five, 9.4%), *Streptococcus pneumoniae* (three, 5.7%) and *Haemophilus influenzae* (two, 3.8%). Distribution was strongly syndrome-dependent. All 11 *Salmonella* Typhi isolates arose from enteric fever, and that syndrome yielded no other organism. Meningitis was almost exclusively Gram-negative (10/11, 90.9%), whereas sepsis was the only syndrome in which Gram-positive organisms predominated (6/11, 54.5%). Culture yield and pathogen distribution by syndrome are detailed in Table 2.

Antimicrobial Resistance

Twenty-four of 53 isolates (45.3%) met criteria for MDR or XDR: 17 MDR (32.1%) and seven XDR (13.2%). Resistance was concentrated in the Enterobacterales. *E. coli* showed the highest burden (9/11, 81.8% MDR or XDR, including three XDR), followed by *K. pneumoniae* (7/10, 70.0%) and *S. aureus* (7/11, 63.6%). *Salmonella* Typhi showed a

distinct and uniform phenotype: all 11 isolates were ciprofloxacin-resistant, yet every isolate remained susceptible to ampicillin, ceftriaxone, azithromycin, chloramphenicol and cotrimoxazole. Fluoroquinolone resistance alone did not meet MDR criteria, so all 11 were classified as non-multidrug-resistant. By syndrome, MDR/XDR phenotypes were commonest in meningitis (8/11, 72.7%) and absent in enteric fever, although the overall comparison did not attain significance (P = 0.121), consistent with limited stratum sizes.

Seven of 11 *S. aureus* isolates (63.6%) were oxacillin- and cefoxitin-resistant, indicating methicillin resistance. Among agents tested in at least 10 isolates, resistance was highest for amoxicillin and amoxicillin–clavulanate (16/21, 76.2%), ciprofloxacin (28/39, 71.8%), levofloxacin (19/28, 67.9%) and cefepime (17/26, 65.4%). The ciprofloxacin figure was driven substantially by the enteric fever isolates, all 11 of which were resistant. Ceftriaxone resistance reached 43.2% (16/37), a figure with direct implications for empirical practice. Several agents retained complete in-vitro activity: amikacin (26/26), vancomycin (14/14), linezolid (14/14), azithromycin (14/14) and chloramphenicol (16/16). Carbapenem resistance was 19.2% for both meropenem and imipenem (5/26). Colistin and tigecycline were fully active but tested in only nine and four isolates respectively, so those denominators cannot support inference. Resistance phenotypes by organism and agent-level resistance are shown in Figure 3, with detail in Table 3.

Cutaneous Manifestations

Skin findings were documented in 17 episodes (16.2%), and their distribution across syndromes was markedly uneven (P = 0.004) (Figure 4). Meningitis accounted for the largest share (8/18, 44.4%), while no child with urinary tract infection had cutaneous involvement (0/19). Purpuric lesions were commonest (six episodes), followed by petechial (four), maculopapular (three), pustular (two), cellulitic (one) and necrotic (one). Median lesion onset was one day after fever onset (range −2 to +6 days); in two episodes cutaneous signs preceded fever. Dermatology consultation was obtained in 14 of the 17 episodes (82.4%).

Morphology tracked organism class in a pattern consistent with established associations, though numbers are small. Purpuric and petechial lesions occurred predominantly with Gram-negative isolates and in meningitis, whereas pustular and cellulitic morphology was confined to *S. aureus*. Cutaneous involvement showed no association with mortality (2/17 versus 11/88; P = 1.000).

Outcomes and Factors Associated with Mortality

Sixty-seven children (63.8%) recovered without intensive care, 25 (23.8%) recovered after intensive care, and 13 (12.4%) died. Thirty (28.6%) required intensive care admission at any point. Median length of stay was eight days (IQR 6–11). Mortality was highest in sepsis (5/23, 21.7%) and meningitis (3/18, 16.7%), and no death occurred in enteric fever; the syndrome-level comparison approached but did not attain significance (P = 0.056). Outcomes by syndrome appear in Table 4.

Non-survivors were older (median 111.0 versus 74.5 months; P = 0.042) and heavier (30.4 versus 22.7 kg; P = 0.039), a finding likely mediated by the age distribution of sepsis and meningitis rather than by age itself. They had higher C-reactive protein (109.4 versus 85.6 mg/L; P = 0.003), higher neutrophil percentage (79.5% versus 71.3%; P = 0.027), higher lactate (3.05 versus 2.12 mmol/L; P = 0.008), higher creatinine (0.98 versus 0.77 mg/dL; P = 0.001) and longer stay (11 versus eight days; P = 0.003). Procalcitonin was higher among non-survivors but the difference was not significant (6.75 versus 4.54 ng/mL; P = 0.087).

Among categorical exposures, the presence of an underlying condition was associated with death (7/30 versus 6/75; OR 3.50, 95% CI 1.07–11.48; P = 0.047). Within the 53 culture-confirmed episodes, an MDR or XDR phenotype was associated with mortality (6/24 versus 1/29; P = 0.038). Central venous access (OR 2.77, 95% CI 0.64–11.94; P = 0.168), prior antimicrobial exposure (OR 2.17, 95% CI 0.66–7.16; P = 0.240), immunosuppression (OR 2.00, 95% CI 0.48–8.32; P = 0.393) and inappropriate empirical therapy (OR 1.50, 95% CI 0.45–5.02; P = 0.529) all pointed in the direction of harm without reaching significance. Empirical therapy was appropriate in 73 episodes (69.5%). Given 13 deaths, the cohort supports at most one or two covariates in any multivariable model; formal adjustment was therefore not attempted, and all associations reported here are unadjusted.

Table 1: Clinical and laboratory characteristics of 105 paediatric episodes of suspected bloodstream infection, stratified by clinical syndrome

Characteristic	Overall (n=105)	Sepsis (n=23)	Enteric fever (n=20)	UTI (n=19)	Pneumonia (n=23)	Meningitis (n=18)	Febrile neutropenia (n=2)	P value
Number of episodes	105	23	20	19	23	18	2	—
Age, months	76.0 (44.0–111.0)	98.0 (55.0–116.0)	82.5 (65.8–114.8)	72.0 (39.5–105.5)	77.0 (51.0–105.0)	68.5 (31.2–108.0)	54.0 (51.0–57.0)	0.710
Weight, kg	24.0 (15.2–32.9)	26.3 (16.9–34.3)	25.8 (15.6–35.5)	17.9 (14.6–32.7)	24.6 (16.4–31.1)	21.1 (13.6–32.7)	15.9 (14.4–17.4)	0.820
Weight-for-age Z score	−0.10 (−0.67–	−0.39 (−0.74–	−0.35 (−0.95–	0.09 (−0.47–	−0.01 (−0.55–	−0.17 (−0.71–	−0.40 (−1.27–0.48)	0.988

	0.54)	0.48)	0.50)	0.55)	0.46)	0.53)		
CRP, mg/L	92.7 (69.9–113.0)	102.7 (95.2–121.2)	70.8 (51.9–93.4)	83.1 (66.1–96.9)	82.0 (66.5–99.3)	110.8 (82.4–120.2)	101.8 (91.7–111.8)	0.001
Procalcitonin, ng/mL	4.92 (2.93–7.14)	8.52 (7.03–10.36)	2.80 (1.88–4.09)	4.44 (3.50–5.38)	4.26 (2.41–5.75)	5.24 (3.22–7.48)	6.38 (6.24–6.53)	<0.001
WBC, $\times 10^9$ /L	15.1 (11.5–19.6)	15.1 (13.2–17.6)	13.6 (9.3–16.9)	15.3 (8.8–19.2)	17.8 (13.2–20.2)	15.4 (11.6–19.6)	16.4 (13.4–19.5)	0.456
Neutrophils, %	71.9 (62.1–78.4)	75.3 (71.1–79.3)	66.7 (53.1–73.1)	71.6 (64.7–75.0)	68.5 (60.6–82.0)	72.2 (63.0–78.3)	83.5 (80.1–86.8)	0.054
Platelets, $\times 10^9$ /L	238 (173–290)	178 (136–260)	284 (242–307)	272 (209–334)	229 (179–277)	194 (155–240)	237 (230–244)	0.008
Lactate, mmol/L	2.24 (1.71–3.15)	3.15 (2.41–3.96)	1.97 (1.61–2.42)	2.02 (1.44–2.96)	1.74 (1.36–2.72)	2.71 (2.04–3.14)	2.21 (1.87–2.54)	0.006
Creatinine, mg/dL	0.80 (0.63–0.98)	1.02 (0.85–1.17)	0.69 (0.60–0.93)	0.72 (0.48–0.86)	0.64 (0.48–0.91)	0.92 (0.61–0.98)	0.96 (0.87–1.04)	<0.001
Length of stay, days	8.0 (6.0–11.0)	10.0 (8.0–11.0)	6.5 (5.0–7.0)	7.0 (6.0–9.5)	8.0 (6.0–11.0)	11.0 (8.2–12.0)	10.0 (9.0–11.0)	<0.001
Male sex	53 (50.5)	12 (52.2)	11 (55.0)	10 (52.6)	11 (47.8)	7 (38.9)	2 (100.0)	0.666
Infant (<12 months)	8 (7.6)	0 (0.0)	2 (10.0)	1 (5.3)	3 (13.0)	2 (11.1)	0 (0.0)	0.513
Underlying condition	30 (28.6)	8 (34.8)	6 (30.0)	5 (26.3)	6 (26.1)	3 (16.7)	2 (100.0)	0.204
Immunosuppressed	15 (14.3)	4 (17.4)	3 (15.0)	3 (15.8)	2 (8.7)	1 (5.6)	2 (100.0)	0.016
PICU at admission	37 (35.2)	19 (82.6)	0 (0.0)	1 (5.3)	3 (13.0)	13 (72.2)	1 (50.0)	<0.001
Healthcare-associated onset	25 (23.8)	6 (26.1)	4 (20.0)	4 (21.1)	4 (17.4)	7 (38.9)	0 (0.0)	0.592
Central line present	12 (11.4)	2 (8.7)	2 (10.0)	1 (5.3)	4 (17.4)	2 (11.1)	1 (50.0)	0.457
Prior antibiotics, 30 days	47 (44.8)	8 (34.8)	9 (45.0)	8 (42.1)	10 (43.5)	11 (61.1)	1 (50.0)	0.705
Skin findings present	17 (16.2)	3 (13.0)	3 (15.0)	0 (0.0)	2 (8.7)	8 (44.4)	1 (50.0)	0.004
Empiric therapy appropriate	73 (69.5)	18 (78.3)	14 (70.0)	11 (57.9)	17 (73.9)	12 (66.7)	1 (50.0)	0.749

Continuous variables are median (interquartile range); categorical variables are n (%). P values from the Kruskal–Wallis test (continuous) or the chi-squared / Fisher exact test (categorical), across all six syndromes. The febrile neutropenia stratum (n = 2) is shown for completeness; no inference is drawn from it, and exclusion of this stratum did not alter any conclusion. CRP, C-reactive protein; PICU, paediatric intensive care unit; UTI, urinary tract infection; WBC, white blood cell count.

Table 2: Table 2: Blood culture yield and pathogen distribution by clinical syndrome

Syndrome	Episodes	Culture positive, n (%)	True pathogen, n (%)	Isolates (n)	Gram-negative, n (%)	Gram-positive, n (%)	Predominant organisms (n)
Sepsis	23	14 (60.9)	11 (47.8)	11	5 (45.5)	6 (54.5)	*S. aureus* 5; *E. coli* 3
Enteric fever	20	12 (60.0)	11 (55.0)	11	11 (100.0)	0 (0.0)	*S.* Typhi 11
UTI	19	8 (42.1)	7 (36.8)	7	5 (71.4)	2 (28.6)	*K. pneumoniae* 3
Pneumonia	23	14 (60.9)	12 (52.2)	12	8 (66.7)	4 (33.3)	*E. coli* 5; *S. aureus* 3
Meningitis	18	13 (72.2)	11 (61.1)	11	10 (90.9)	1 (9.1)	*K. pneumoniae* 4; *E. coli* 3
Febrile neutropenia	2	2 (100.0)	1 (50.0)	1	0 (0.0)	1 (100.0)	*S. aureus* 1
Overall	105	63 (60.0)	53 (50.5)	53	39 (73.6)	14 (26.4)	—

Culture positive and true pathogen percentages are calculated on the syndrome episode denominator. Gram-negative and Gram-positive percentages are calculated on the isolate denominator for that syndrome. Ten positive cultures were adjudicated as contaminant or no true isolate and are excluded from isolate-level columns. Comparison of culture yield across syndromes: P = 0.415. UTI, urinary tract infection.

Table 3: Resistance phenotype and key susceptibility findings by organism (53 true pathogens)

Organism	n	Susceptible, n (%)	MDR, n (%)	XDR, n (%)	MDR/XDR combined, n (%)	Key agent-level findings
Staphylococcus aureus	11	4 (36.4)	5 (45.5)	2 (18.2)	7 (63.6)	Oxacillin/cefoxitin R 7 (63.6); vancomycin and linezolid S 11 (100.0)

Salmonella Typhi	11	11 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	Ciprofloxacin R 11 (100.0); ampicillin, ceftriaxone, azithromycin, chloramphenicol, cotrimoxazole all S 11 (100.0)
Klebsiella pneumoniae	10	3 (30.0)	6 (60.0)	1 (10.0)	7 (70.0)	Ceftriaxone R 7 (70.0); meropenem R 1 (10.0); amikacin S 10 (100.0)
Escherichia coli	11	2 (18.2)	6 (54.5)	3 (27.3)	9 (81.8)	Ceftriaxone R 9 (81.8); meropenem R 3 (27.3); amikacin S 11 (100.0)
Pseudomonas aeruginosa	5	4 (80.0)	0 (0.0)	1 (20.0)	1 (20.0)	Ceftazidime–avibactam S 4 (80.0); amikacin S 5 (100.0)
Streptococcus pneumoniae	3	3 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	Fully susceptible to agents tested
Haemophilus influenzae	2	2 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	Fully susceptible to agents tested
Overall	*53*	*29 (54.7)*	*17 (32.1)*	*7 (13.2)*	*24 (45.3)*	—

MDR and XDR defined per Magiorakos et al.13 The Susceptible column denotes isolates not meeting MDR or XDR criteria, not pan-susceptibility; all 11 Salmonella Typhi isolates so classified were ciprofloxacin-resistant but resistant to only one antimicrobial class and therefore non-multidrug-resistant. Percentages are calculated on the organism-specific isolate denominator. Agent-level denominators include only isolates tested against that agent; testing was clinically directed rather than protocolised, which introduces indication bias — agents reserved for resistant isolates (colistin, tested in 9; tigecycline, tested in 4) are not shown here and their apparent full activity should not be read as population-level susceptibility. R, resistant; S, susceptible.

Table 4: Clinical outcomes by syndrome

Syndrome	Episodes	Recovered, n (%)	Recovered after ICU, n (%)	Mortality, n (%)	ICU admission, n (%)	Median LOS, days (IQR)
Sepsis	23	9 (39.1)	9 (39.1)	5 (21.7)	11 (47.8)	10.0 (8.0–11.0)
Enteric fever	20	18 (90.0)	2 (10.0)	0 (0.0)	2 (10.0)	6.5 (5.0–7.0)
UTI	19	14 (73.7)	3 (15.8)	2 (10.5)	3 (15.8)	7.0 (6.0–9.5)
Pneumonia	23	15 (65.2)	6 (26.1)	2 (8.7)	7 (30.4)	8.0 (6.0–11.0)
Meningitis	18	11 (61.1)	4 (22.2)	3 (16.7)	6 (33.3)	11.0 (8.2–12.0)
Febrile neutropenia	2	0 (0.0)	1 (50.0)	1 (50.0)	1 (50.0)	10.0 (9.0–11.0)
Overall	*105*	*67 (63.8)*	*25 (23.8)*	*13 (12.4)*	*30 (28.6)*	*8.0 (6.0–11.0)*

Mortality across syndromes: P = 0.056. ICU, intensive care unit; IQR, interquartile range; LOS, length of stay; UTI, urinary tract infection.

Table 4b: Univariate associations with in-hospital mortality

Exposure	Deaths / exposed (%)	Deaths / unexposed (%)	Odds ratio (95% CI)	*P* value
Underlying condition present	7/30 (23.3)	6/75 (8.0)	3.50 (1.07–11.48)	0.047
MDR/XDR phenotype (isolates only)	6/24 (25.0)	1/29 (3.4)	9.33 (1.04–83.4)	0.038
Central line present	3/12 (25.0)	10/93 (10.8)	2.77 (0.64–11.94)	0.168
Prior antibiotics, 30 days	8/47 (17.0)	5/58 (8.6)	2.17 (0.66–7.16)	0.240
Immunosuppression	3/15 (20.0)	10/90 (11.1)	2.00 (0.48–8.32)	0.393
Inappropriate empirical therapy	5/32 (15.6)	8/73 (11.0)	1.50 (0.45–5.02)	0.529
ICU admission	5/30 (16.7)	8/75 (10.7)	1.68 (0.50–5.61)	0.512
Skin findings present	2/17 (11.8)	11/88 (12.5)	0.93 (0.19–4.62)	1.000
Healthcare-associated onset	2/25 (8.0)	11/80 (13.8)	0.55 (0.11–2.65)	0.729
Male sex	5/53 (9.4)	8/52 (15.4)	0.57 (0.17–1.88)	0.390

P values from the Fisher exact test. All associations are unadjusted: with 13 deaths, the cohort does not support multivariable adjustment. The MDR/XDR comparison is restricted to the 53 culture-confirmed episodes, since episodes without an isolate have no resistance phenotype and cannot serve as an unexposed comparator. CI, confidence interval; ICU, intensive care unit; MDR, multidrug-resistant; XDR, extensively drug-resistant.

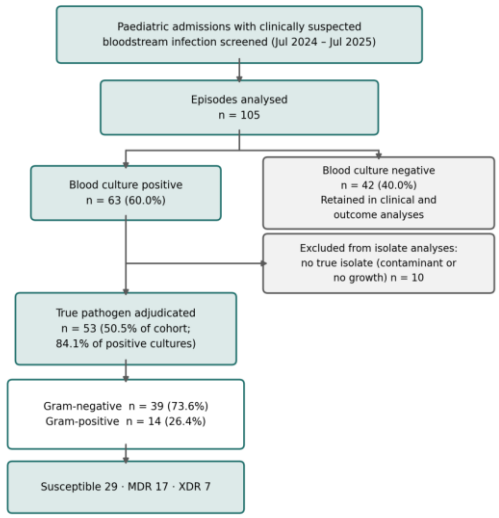

Figure 1: STROBE participant flow diagram. Of 105 analysed episodes of clinically suspected paediatric bloodstream infection, 63 (60.0%) yielded a positive blood culture and 53 (50.5%) a true pathogen on consensus adjudication. Culture-negative episodes were retained in clinical and outcome analyses.

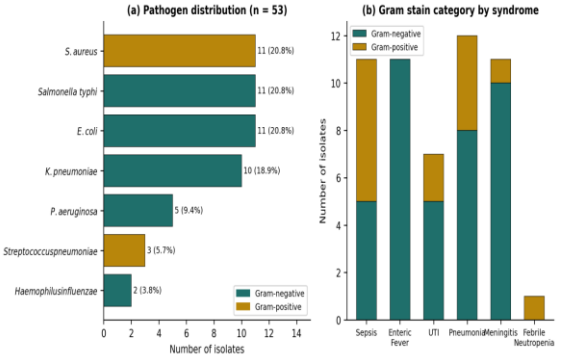

Figure 2: Pathogen distribution among 53 true pathogens. (a) Organism frequency, coloured by Gram stain category. (b) Gram stain category by clinical syndrome, showing Gram-negative predominance in all syndromes except sepsis and febrile neutropenia.

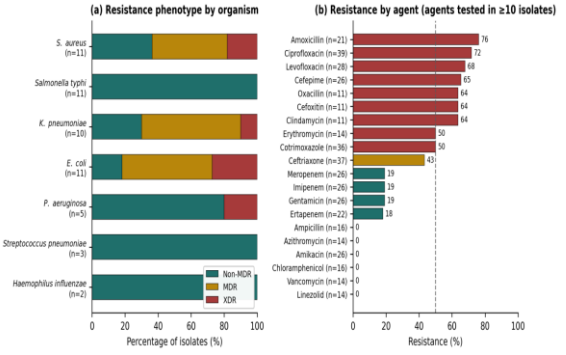

Figure 3: Antimicrobial resistance. (a) Resistance phenotype distribution by organism, expressed as a percentage of that organism's isolates. (b) Resistance percentage by agent, restricted to agents tested in at least 10 isolates; the dashed line marks 50% resistance. Agent denominators differ because susceptibility testing was clinically directed.

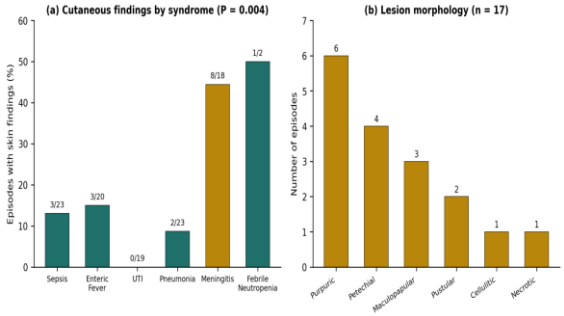

Figure 4: Cutaneous manifestations. (a) Proportion of episodes with skin findings by clinical syndrome (P = 0.004); fractions above each bar are episodes with findings / total episodes. (b) Lesion morphology among the 17 episodes with cutaneous involvement.

DISCUSSION

Three findings from this cohort bear on empirical practice. Gram-negative organisms accounted for nearly three-quarters of isolates; almost half of all isolates were multidrug- or extensively drug-resistant; and ceftriaxone — the default empirical agent for much of paediatric practice in this setting — was inactive against 43.2% of isolates tested against it. Set beside a culture yield of 60.0% and a true-pathogen yield of 50.5%, these figures describe a situation in which the clinician commits to therapy under substantial and quantifiable uncertainty.

Pathogen Distribution in Context

The Gram-negative predominance we observed is consistent with the direction of reported Indian paediatric series, though the magnitude varies considerably between reports.[4,14] That variation is not merely statistical noise. It reflects genuine differences in case mix: series drawn from oncology or intensive care populations report higher proportions of Pseudomonas and coagulase-negative staphylococci, reflecting central venous access and prolonged exposure, whereas general paediatric cohorts more closely resemble the distribution described here.[5,15] Comparisons across studies that do not specify recruitment setting should therefore be treated with caution.

The fluoroquinolone resistance of all,[11] Salmonella Typhi isolates aligns with the well-documented dominance of fluoroquinolone-resistant, gyrA-mutated lineages across South Asia, and is consistent with reports tracking the decline of ciprofloxacin as a reliable agent for enteric fever in the region.[16,17] The clinical implication is direct: ciprofloxacin is no longer a dependable empirical choice for enteric fever in this population. Retained susceptibility to ceftriaxone and azithromycin identifies both as rational first-line options, a pattern that fits current regional guidance. One caveat tempers the resistance signal. Fluoroquinolone susceptibility for Salmonella Typhi was reported here as a binary phenotype without accompanying minimum inhibitory concentrations, so isolates in the intermediate or low-level-resistance range

cannot be distinguished from high-level resistance. This distinction carries therapeutic weight, since some low-level fluoroquinolone-resistant infections respond to higher-dose regimens. Eleven isolates also provide limited precision for the susceptibility that remains: the true prevalence of ceftriaxone or azithromycin non-susceptibility could be appreciable and still compatible with 0 of 11 observed here. Sustained surveillance with larger denominators and MIC data is required before these agents are assumed durable for local enteric fever management.

Resistance and Its Clinical Consequence

The MDR/XDR burden of 45.3% falls within the broad range reported from Indian tertiary paediatric settings, and the concentration in E. coli (81.8%) and K. pneumoniae (70.0%) matches the pattern expected where extended-spectrum beta-lactamase production is endemic.[6,18] Carbapenem resistance at 19.2% is the figure with the most uncomfortable implication, since it constrains the agents available when third-generation cephalosporins fail. Retained amikacin activity (26/26) offers a partial answer and supports aminoglycoside-containing empirical combinations in this setting, subject to the monitoring obligations that accompany them.

We found an association between MDR/XDR phenotype and mortality within culture-confirmed episodes (P = 0.038). This should be read cautiously. The comparison rests on seven deaths across 53 episodes, is unadjusted, and cannot distinguish the direct effect of resistance from confounding by illness severity or underlying condition — children with resistant organisms in this cohort were also those with nephrotic syndrome, malignancy and prior antimicrobial exposure. A plausible mechanism is delayed effective therapy, but our data do not support that inference: time to appropriate therapy did not differ between survivors and non-survivors (P = 0.865), and inappropriate empirical therapy was not associated with death (P = 0.529). The resistance–mortality relationship in paediatric BSI remains, in our view, inadequately resolved by cohorts of this size.

Cutaneous Findings

The syndrome-dependence of cutaneous involvement was the clearest dermatological signal (P = 0.004): 44.4% in meningitis against none in urinary tract infection. The morphological associations we observed — purpuric and petechial lesions with Gram-negative meningitis, pustular and cellulitic lesions with S. aureus — align with established descriptions and lend internal coherence to the dataset.[9,19] Their practical value is more limited than the P value suggests. Cutaneous signs were present in only 16.2% of episodes overall, and absence of skin findings therefore carries little discriminatory weight. We found no association with mortality, and we would caution against reading these findings as support for cutaneous morphology as a triage instrument. What the data suggest, more modestly, is that when purpuric or petechial lesions appear in a febrile child, a Gram-negative meningitic process merits active consideration — and that systematic dermatological co-recording is feasible within routine paediatric admission, which few published cohorts have demonstrated.

Limitations

Several constraints bound these conclusions. This is a single-centre cohort of 105 episodes with 13 deaths, which precludes multivariable adjustment; every association reported is unadjusted and susceptible to confounding. The febrile neutropenia stratum contained two episodes and supports no inference whatever. Blood culture yield of 60.0% against a background of 44.8% prior antimicrobial exposure means a substantial fraction of true bacteraemic episodes were almost certainly missed, and any organism-specific conclusion applies to the culturable subset rather than to paediatric BSI as such. Susceptibility denominators vary by agent because testing was clinically directed rather than protocolised, which introduces indication bias into the antibiogram: agents such as colistin and tigecycline were tested preferentially in already-resistant isolates, and their apparent full activity reflects that selection. Dermatology consultation was discretionary, so milder cutaneous findings may have gone unrecorded. Molecular characterisation of resistance determinants was not performed, and phenotypic MDR/XDR classification cannot distinguish underlying mechanisms. Finally, the cohort spans 13 months at one institution and should not be generalised beyond comparable tertiary settings.

Implications and Future Directions

Three implications follow with reasonable directness. Ceftriaxone monotherapy appears inadequate as empirical cover for suspected Gram-negative sepsis or meningitis in this setting, given 43.2% resistance among tested isolates. For enteric fever, ciprofloxacin should not be used empirically, since every Salmonella Typhi isolate in this cohort was fluoroquinolone-resistant; retained susceptibility positions ceftriaxone and azithromycin as the rational first-line agents. And local antibiograms would serve clinicians better if reported by syndrome rather than as a single institutional aggregate, since the organism spectrum in enteric fever and in meningitis have almost nothing in common.

Priorities for further work follow from the limitations. Multicentre cohorts of sufficient size to permit adjusted analysis of the resistance–mortality relationship are the principal need. Molecular characterisation of the resistance determinants circulating in these Enterobacterales would clarify whether the observed phenotypes reflect a clonal outbreak or independent acquisition. Prospective protocolised dermatological assessment, rather than discretionary consultation, would establish whether cutaneous morphology adds measurable

discriminatory value over routine clinical assessment — a question our data raise but cannot answer.

CONCLUSION

Gram-negative organisms dominated culture-confirmed paediatric bloodstream infection in this cohort, and 45.3% of isolates were multidrug- or extensively drug-resistant, with ceftriaxone inactive against 43.2% of isolates tested. *Salmonella* Typhi was uniformly fluoroquinolone-resistant while retaining susceptibility to ceftriaxone and azithromycin, marking ciprofloxacin as unsuitable for empirical enteric fever treatment locally. Cutaneous findings were strongly syndrome-dependent, concentrated in meningitis, but showed no association with mortality and were absent in most episodes. An MDR/XDR phenotype was associated with death among culture-confirmed episodes, though the cohort is too small to separate that association from confounding by severity. Syndrome-stratified local antibiograms, rather than aggregate institutional figures, offer the most immediately actionable route to more rational empirical prescribing.

Declarations

Ethics approval and consent to participate: Written informed consent was obtained from parents or legal guardians, with assent from children over seven years.

Consent for publication: Not applicable; no identifying patient information is included.

Availability of data: The dataset analysed is available from the corresponding author on reasonable request.

Conflicts of interest: None declared.

Funding: None.

Authors' contributions: Shiyas [surname] conceived the study, supervised clinical data collection and drafted the manuscript. Faisal [surname] performed the dermatological assessment and lesion morphology classification. [Third author name] and Bijesh P.K performed the microbiological processing, isolate adjudication and susceptibility interpretation. All authors contributed to analysis and interpretation, revised the manuscript critically, approved the final version and agree to be accountable for all aspects of the work.

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REFERENCES

1. Naghavi M, Vollset SE, Ikuta KS, et al. Global burden of bacterial antimicrobial resistance 1990–2021: a systematic analysis with forecasts to 2050. Lancet. 2024;404(10459):1199–226.
2. Murray CJL, Ikuta KS, Sharara F, et al. Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis. Lancet. 2022;399(10325):629–55.
3. Holmes CL, Albin OR, Mobley HLT, Bachman MA. Bloodstream infections: mechanisms of pathogenesis and opportunities for intervention. Nat Rev Microbiol. 2025;23(4):210–24.
4. Mathur P, Malpiedi P, Walia K, et al. Health-care-associated bloodstream and urinary tract infections in a network of hospitals in India: a multicentre, hospital-based, prospective surveillance study. Lancet Glob Health. 2022;10(9):e1317–25.
5. Baccelli F, Compagno F, Tridello G, et al. The epidemiological landscape of bloodstream infections in children undergoing chemotherapy or haematopoietic cell transplantation: a retrospective study by Infectious Diseases Working Group of Italian Association of Pediatric Hematology and Oncology (AIEOP). Br J Haematol. 2025;207(5):1982–93.
6. Parveen R, Thakur AK, Srivastav S, et al. Profile of central line-associated bloodstream infections in adult, paediatric, and neonatal intensive care units of hospitals participating in a health-care-associated infection surveillance network in India: a 7-year multicentric study. Lancet Glob Health. 2025;13(9):e1564–73.
7. Whelan SO, Mulrooney C, Moriarty F, Cormican M. Pediatric blood cultures—turning up the volume: a before and after intervention study. Eur J Pediatr. 2024;183(7):3063–71.
8. Driscoll AJ, Deloria Knoll M, Hammitt LL, et al. The effect of antibiotic exposure and specimen volume on the detection of bacterial pathogens in children with pneumonia. Clin Infect Dis. 2017;64(Suppl 3):S368–77.
9. Pulido-Pérez A, Sánchez-Carrillo C, Bergón-Sendín M, et al. Prevalence and clinical features of secondary skin lesions in septic patients with bloodstream infections. Eur J Clin Microbiol Infect Dis. 2022;41(5):779–86.
10. Delgado-Jiménez Y, Fraga J, Requena C, et al. Acute bacterial septic vasculopathy. Int J Dermatol. 2013;52(9):1071–80.
11. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–7.
12. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 36th ed. CLSI supplement M100-Ed36. Wayne (PA): Clinical and Laboratory Standards Institute; 2026.
13. Magiorakos AP, Srinivasan A, Carey RB, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect. 2012;18(3):268–81.
14. Lipworth S, Vihta KD, Davies T, et al. Molecular epidemiology and antimicrobial resistance phenotype of paediatric bloodstream infections caused by Gram-negative bacteria. Commun Med. 2022;2:101.
15. Sands K, Carvalho MJ, Portal E, et al. Characterization of antimicrobial-resistant Gram-negative bacteria that cause neonatal sepsis in seven low- and middle-income countries. Nat Microbiol. 2021;6(4):512–23.
16. Carey ME, Dyson ZA, Ingle DJ, et al. Global diversity and antimicrobial resistance of typhoid fever pathogens: insights from a meta-analysis of 13,000 Salmonella Typhi genomes. eLife. 2023;12:e85867.
17. Browne AJ, Chipeta MG, Fell FJ, et al. Estimating the subnational prevalence of antimicrobial resistant Salmonella enterica serovars Typhi and Paratyphi A infections in 75 endemic countries, 1990–2019: a modelling study. Lancet Glob Health. 2024;12(3):e406–18.
18. Beaumont AL, Kermorvant-Duchemin E, Breurec S, Huynh BT. Neonatal colonization with antibiotic-resistant pathogens in low- and middle-income countries: a systematic review and meta-analysis. JAMA Netw Open. 2024;7(11):e2441596.

19. Taquin H, Hubiche T, Roudière L, Fribourg A, Del Giudice P. Prevalence and clinical characteristics of cutaneous manifestations associated with bacteraemia: a cross-sectional prospective study. Acta Derm Venereol. 2019;99(2):170–4.

20. Ciapponi A, Bardach A, Sandoval MM, et al. Systematic review and meta-analysis of deaths attributable to antimicrobial resistance, Latin America. Emerg Infect Dis. 2023;29(11):2335–44.