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BACTERIOLOGICAL PROFILE AND ANTIMICROBIAL RESISTANCE PATTERNS IN URINE SPECIMENS OBTAINED FROM SICKLE CELL DISEASE PATIENTS

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ABSTRACT:

Patients with sickle cell disease (SCD) have a substantially high risk of urinary tract infections (UTIs) owing to functional asplenia, renal medullary pathology, and compromised immunological defenses. Characterizing regional pathogen profiles and their resistance phenotypes is a prerequisite for rational therapeutic decision-making in this vulnerable population. To recover and identify uropathogens from urine specimens of patients with SCD, characterize their antimicrobial susceptibility phenotypes, and formulate evidence-based treatment recommendations applicable to the regional clinical context. A descriptive cross-sectional study conducted at the Civil Hospital in Chandrapur involved 20 participants who have been confirmed to have sickle cell disease (SCD). The ages of these participants ranged from 5 to 40 years. Midstream urine specimens were cultured on CLED, MacConkey, and Mueller-Hinton agar plates. Found the isolated samples by applying Gram staining, observing how they moved, and performing a regular set of biochemical tests. (IMViC, oxidase, catalase). Antimicrobial Susceptibility was assessed using the Kirby-Bauer disk diffusion method in line with the CLSI 2024 guidelines breakpoints. Eight distinct uropathogens were identified: *Escherichia coli, Enterobacter* spp., *Pseudomonas aeruginosa*, *Klebsiella* spp., *Proteus* spp., *Staphylococcus aureus*, *Enterococcus faecalis* and *S. saprophyticu*, Gram-negative bacilli comprised 62.5% of the total isolates. Ampicillin, penicillin-G, and erythromycin elicited the highest resistance rates in the study. Amikacin and ciprofloxacin demonstrated the broadest efficacy against gram-negative organisms. Microbiologically guided therapy is indispensable for UTI management in patients with SCD. Aminoglycosides and fluoroquinolones are the most dependable empirical choices. Formal antimicrobial stewardship initiatives and universal urine culture protocols are urgently needed for this high-risk cohort.

Keywords:- Sickle cell disease; urinary tract infection; uropathogens; antimicrobial resistance; disk diffusion; CLSI 2024; antimicrobial stewardship; ESBL; Chandrapur; Maharashtra

INTRODUCTION:

Sickle cell disease (SCD) is a monogenic hemoglobinopathy attributable to a missense point mutation (Glu→Val) at position 6 of the beta-globin chain, producing structurally aberrant hemoglobin S (HbS). Under hypoxic or acidotic conditions, HbS molecules aggregate into polymeric fibers, imposing a rigid, sickled morphology on erythrocytes. The ensuing pathophysiological cascade encompasses chronic intravascular hemolysis, repeated vaso-occlusive episodes, endothelial injury, and progressive dysfunction of multiple organ systems, positioning SCD among the most clinically demanding single-gene disorders encountered worldwide (Délicat-Loembet et al., 2023).

Bacterial infections, particularly UTIs, are a recurrent and clinically significant source of morbidity in SCD. The mechanistic basis for this susceptibility is multifactorial: functional asplenia from repeated splenic infarction abrogates opsonophagocytic clearance of encapsulated organisms; defective complement activation impairs humoral innate immunity; and SCD-specific renal pathology, including papillary necrosis, medullary hypoxia, and hyposthenuria,

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creates a microenvironment conducive to bacterial ascent and persistence in the collecting system (Cumming et al., 2006; Donkor et al., 2017).

Epidemiological investigations from endemic regions, including Sub-Saharan Africa, Caribbean island nations, and South Asian countries, consistently document a markedly elevated burden of both symptomatic UTIs and asymptomatic bacteriuria (ASB) among patients with SCD compared to the general population. *Escherichia coli* was the dominant uropathogen in these studies, followed by *Klebsiella* spp. and other *Enterobacteriaceae* members (Opoku-Asare et al., 2023; Sangeda & Manyanga, 2024). There is a growing problem with antimicrobial resistance, especially with the rise of strains that produce extended-spectrum β-lactamase (ESBL). These strains are able to resist certain types of antibiotics, making infections harder to treat. Which is capable to inactivating ampicillin and co-trimoxazole, the historically available first-line agents in low-resource settings, presents a mounting therapeutic challenge (Mohammed et al., 2024; Abayneh et al., 2018).

Despite SCD being hyperendemic among tribal communities across Maharashtra, Chhattisgarh, and Odisha in India, rigorous locally derived data on uropathogen speciation and resistance phenotypes in affected patients are scarce. This investigation addresses this evidence gap by characterizing the bacteriological spectrum and antibiotic susceptibility patterns in urine samples obtained from patients with SCD attending Civil Hospital, Chandrapur, a tertiary referral center serving one of India's highest-prevalence SCD districts. The overarching aim is to provide regionally actionable data that inform empirical prescribing, guide stewardship interventions, and improve patient outcomes.

MATERIALS AND METHODS

2.1 Study Design and Ethical Framework

A descriptive cross-sectional investigation was conducted at the Post Graduate Department of Microbiology, Sardar Patel Mahavidyalaya, Chandrapur, in collaboration with the Civil Hospital, Chandrapur, Maharashtra, India. The study protocol was conducted in accordance with the institutional ethical guidelines. Everyone involved, or their legal guardians if they were minors, gave written permission before any samples were taken. Twenty individuals having a laboratory-confirmed SCD diagnosis (hemoglobin electrophoresis-verified HbSS or clinically significant compound heterozygous genotype), aged 5–40 years, were prospectively enrolled. Both symptomatic (dysuria, urinary frequency, or hematuria) and asymptomatic patients undergoing periodic surveillance were eligible for inclusion.

2.2 Specimen Collection and Transport

Midstream clean-catch urine specimens were procured from each participant according to a standardized aseptic technique, following the procedural specifications detailed in the CLSI (2023) document for urine culture. Each specimen was collected in a sterile, wide-mouthed, leak-proof container pre-labelled with a unique participant identifier. To preserve microbial viability and prevent overgrowth artifacts, The samples were moved to the microbiology lab at room temperature and were examined within two hours after being collected.

2.3 Bacterial Isolation and Phenotypic Identification

An aliquot of each urine specimen was first inoculated into Nutrient Broth and incubated aerobically at 37°C for 18–24 hours to enrich any organism present at sub-threshold concentrations. The enriched broth was then streaked onto four primary isolation media using the standard quadrant streak technique: Cystine Lactose Electrolyte-Deficient (CLED) agar for colony morphology and lactose fermentation assessment, MacConkey agar for selective gram-negative isolation and lactose differentiation, King's B Medium for fluorescent pigment detection in non-fermenting organisms, and cetrimide agar for

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selective recovery of Pseudomonas species. Following 24 hours of incubation at 37 degrees Celsius, the plates were inspected.

Representative colonies of distinct morphotypes were subcultured to purity and characterized by: (i) gram staining to determine gram reaction and cellular morphology; (ii) the hanging-drop wet-mount method for motility determination; and (iii) a standardized biochemical panel comprising the IMViC series (indole production, Methyl Red reaction, Voges-Proskauer test, citrate utilization), along with Oxidase and Catalase assays. All procedures followed the protocols described in Bailey and Scott's Diagnostic Microbiology (Tille, 2021). The complete morphological and biochemical profiles of each isolate are presented in Table 1.

2.4 Antimicrobial Susceptibility Testing

Antibiotic susceptibility was determined using the Kirby-Bauer disk diffusion assay performed on Mueller-Hinton Agar (MHA) plates, adhering in full to CLSI (2024) performance standards. A direct colony suspension in sterile saline was adjusted to a turbidity equivalent to the 0.5 McFarland standard (approximately 1.5×10^8 CFU/mL) and applied uniformly to the MHA surface using a sterile swab. Commercial antibiotic disks were placed at standardized inter-disk distances: Amikacin (AK, 30 µg), Ciprofloxacin (CIP, 5 µg), Ampicillin (AMP, 10 µg), Erythromycin (E, 15 µg), Penicillin-G (P, 10 U), Streptomycin (S, 10 µg), Tetracycline (TE, 30 µg), and Gentamicin (GM, 10 µg). Following incubation at 35 ± 2°C for 16–18 h, inhibition zone diameters were measured in millimeters with a calibrated ruler and classified as susceptible (S), intermediate (I), or resistant (R) against the breakpoints tabulated in CLSI M100, 34th Edition (2024), reproduced in Table 3.

2.5 Statistical Analysis and Percentage Calculation

Descriptive statistics were used to summarize the distribution of isolates and susceptibility outcomes. Frequency data were expressed as absolute counts and percentages. All proportions reported in this study were derived using the following standard percentage formula:

General Percentage Formula

Percentage (%) = (Number of Observations in Category / Total Number of Observations) × 100

where the numerator is the count of observations in the specific category of interest, the denominator is the total count of all observations in the dataset, and the multiplier is 100 (converts decimal fraction to percentage scale).

Table 5. All Percentage Values Reported in This Study Formula Application and Worked Calculations

Parameter/ Observation	n (Numerator)	N (Denominator)	Formula Applied	Calculated %
Culture-positivity rate (specimens yielding isolates)	8	20	(8/20)×100	40.00%
Gram-negative bacilli among total isolates	5	8	(5/8)×100	62.50%
Gram-positive cocci among total isolates	3	8	(3/8)×100	37.50%
Each individual species as a share of isolates (for example E. coli)	1	8	(1/8)×100	12.50%
Gram-neg. isolates resistant to Ampicillin	5	5	(5/5)×100	100.00%
Gram-neg. isolates resistant to Penicillin-G	4	5	(4/5)×100	80.00%
Gram-neg. isolates resistant to Erythromycin	5	5	(5/5)×100	100.00%
Gram-neg. isolates susceptible to Amikacin	5	5	(5/5)×100	100.00%
Gram-neg. isolates susceptible to Ciprofloxacin	5	5	(5/5)×100	100.00%

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n = numerator count for specific category; N = denominator (total observations; context-dependent: N = 20 patients for culture rate; N = 8 total isolates for species/gram category proportions; N = 5 gram-negative isolates for resistance/susceptibility rates). All values were rounded to two decimal places

Table -2 Overview of key diversity measures showing species composition and distribution in the sampled quadrats.

RESULTS AND DISCUSSION:

3.1 Recovery and Characterisation of Isolates

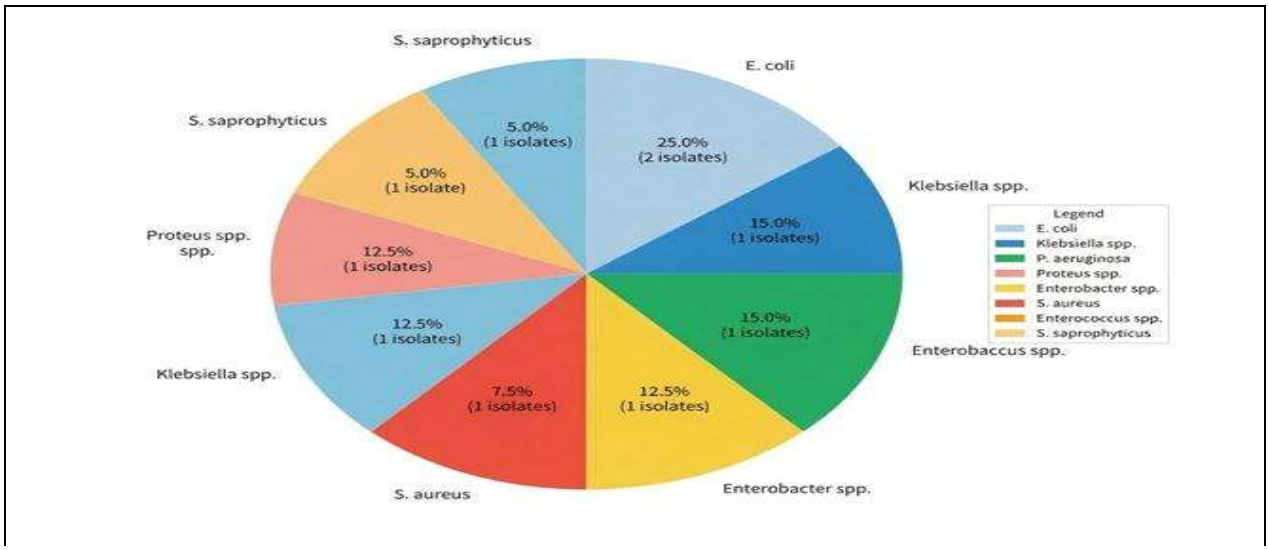
Percentage Calculations Section 3.1

Culture Positivity Rate: (8/20)×100 = 40.00%

Gram-Negative Proportion: (5/8)×100 = 62.50%

Gram-Positive Proportion: (3/8)×100 = 37.50%

Bacterial growth yielding pure isolates was obtained from eight of the 20 urine specimens processed. Applying the standard percentage formula [% = (n/N) × 100], the culture positivity rate was (8/20) × 100 = 40.0%. The eight organisms recovered (designated S1 through S8) comprised five gram-negative bacillary species (S1–S5) and three gram-positive coccal species (S6–S8). The Gram-negative proportion was (5/8) × 100 = 62.5%, and Gram-positive organisms constituted (3/8) × 100 = 37.5% of the isolate pool (Figure 1; Table 5). Each of the eight species accounted for (1/8) × 100 = 12.5% of the total. Lactose-fermenting organisms produced yellow colonies on CLED agar and distinctive pink colonies on MacConkey agar, whereas non-lactose fermenters produced colorless to blue-green growth. The complete morphological and biochemical profiles of each isolate are presented in Table 1. Notably, *P. aeruginosa* (S4) was distinguished by pyocyanin pigmentation on King's B Medium and a strongly positive oxidase reaction, whereas *Enterococcus faecalis* (S8) was differentiated from coagulase-positive *staphylococci* by its catalase-negative result and chain arrangement on Gram staining.



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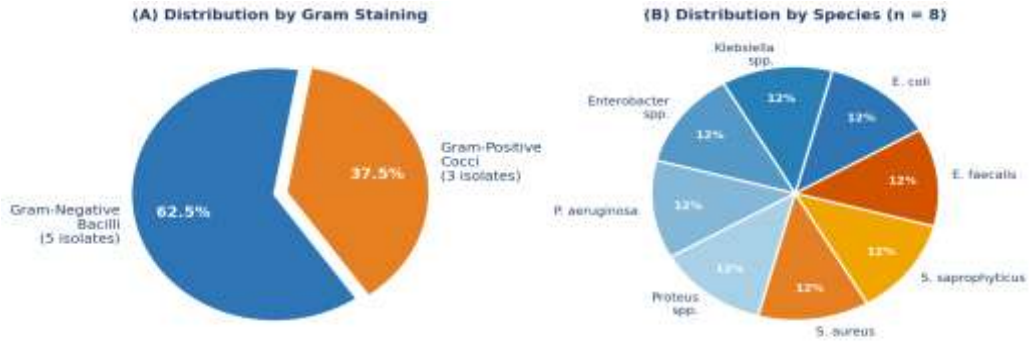

Figure 1. *Distribution of bacterial isolates recovered from the urine specimens of patients with SCD: (A) gram-staining category breakdown (gram-negative, 62.5% vs. gram-positive, 37.5%); (B) species-level proportional distribution across all eight isolates (n = 20 enrolled participants).*

3.2 Antimicrobial Susceptibility Patterns

All five gram-negative isolates (S1–S5) exhibited complete resistance to ampicillin and penicillin-G, either producing no measurable inhibition zone or generating zones below the CLSI-designated resistance thresholds. Erythromycin resistance was similarly pervasive, extending across all gram-negative rods and *S. aureus* (S6). In contrast, amikacin and ciprofloxacin consistently produced the largest zones of inhibition among gram-negative isolates and yielded susceptible classifications for most of them. *P. aeruginosa* (S4) demonstrated intrinsic resistance to all penicillins and macrolides tested, retaining susceptibility exclusively to amikacin and ciprofloxacin, consistent with its recognized inherent resistance mechanisms. Among Gram-positive organisms, *E. faecalis* (S8) displayed intrinsic aminoglycoside resistance and variable fluoroquinolone responses, whereas *S. aureus* (S6) remained ampicillin-susceptible but demonstrated intermediate sensitivity to amikacin. Comprehensive isolate-level susceptibility data are provided in Table 2, CLSI breakpoints in Table 3, and a visualization of all isolate–antibiotic combinations in Figures 2 and 3.

Table 2. Antimicrobial Susceptibility Profiles of Uropathogens (Kirby-Bauer Disk Diffusion Method, CLSI 2024 Breakpoints)

Iso.	*Organism*	AK	CIP	AMP	ERY	PEN-G	STR	TE
S1	*E. coli*	**S**	**S**	**R**	**R**	**R**	**S**	**I**
S2	*Klebsiella spp.*	**S**	**S**	**R (ESBL)**	**R**	**V**	**S**	**R**
S3	*Enterobacter spp.*	**S**	**S**	**R**	**R**	**R**	**S**	**I**
S4	*P. aeruginosa*	**S**	**S**	**R**	**R**	**R**	**R**	**R**
S5	*Proteus spp.*	**S**	**S**	**R**	**R**	**S**	**S**	**I**
S6	*S. aureus*	**I**	**V**	**S**	**R**	**V**	**S**	**I**
S7	*S. saprophyticus*	**S**	**V**	**S**	**V**	**V**	**S**	**V**
S8	*E. faecalis*	**V**	**I**	**S**	**R**	**V**	**R**	**R**

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AK, Amikacin; CIP = Ciprofloxacin; AMP = Ampicillin; ERY = Erythromycin; PEN-G = Penicillin-G; STR = Streptomycin. Color coding: S = Susceptible (green); I = Intermediate (amber); R = Resistant (red); V = Variable strain-dependent (blue). ESBL: extended-spectrum β-lactamase.

Table 3. CLSI (2024) Disk Diffusion Breakpoints Employed for Susceptibility Interpretation (Inhibition Zone Diameters in mm)

Source: CLSI M100 Performance Standards for Antimicrobial Susceptibility Testing, 34th Edition (2024). Plates were incubated at 35 ± 2°C for 16–18h. Zone diameters were measured in millimeters (mm) using a calibrated Vernier caliper.

Antibiotic (Disk Load)	Class	Susceptible (S) ≥ mm	Intermediate (I) mm	Resistant (R) ≤ mm
Amikacin (AK, 30 µg)	Aminoglycoside	**≥17**	15–16	**≤14**
Ciprofloxacin (CIP, 5 µg)	Fluoroquinolone	**≥21**	16–20	**≤15**
Ampicillin (AMP, 10 µg)	Penicillin (β-Lactam)	**≥17**	14–16	**≤13**
Erythromycin (E, 15 µg)	Macrolide	**≥23**	14–22	**≤13**
Penicillin-G (P, 10 U)	Penicillin (β-Lactam)	**≥29**	—	**≤28**
Streptomycin (STR, 10 µg)	Aminoglycoside	**≥15**	12–14	**≤11**
Tetracycline (TE, 30 µg)	Tetracycline	**≥19**	15–18	**≤14**
Gentamicin (GM, 10 µg)	Aminoglycoside	**≥15**	13–14	**≤12**

Table 4. Observed Inhibition Zone Diameters (mm) and Susceptibility Classifications All Isolate–Antibiotic Combinations

Iso.	Organism	AK	CIP	AMP	ERY	PEN-G	STR	TE	GM
S1	*E. coli*	22 (S)	25 (S)	8 (R)	9 (R)	10 (R)	18 (S)	16 (I)	16 (S)
S2	*Klebsiella*	19 (S)	23 (S)	7 (R)	10 (R)	14 (V)	17 (S)	14 (R)	15 (S)
S3	*Enterobacter*	20 (S)	24 (S)	9 (R)	8 (R)	10 (R)	16 (S)	15 (I)	15 (S)
S4	*P. aeruginosa*	21 (S)	22 (S)	6 (R)	6 (R)	7 (R)	12 (R)	10 (R)	12 (R)
S5	*Proteus*	20 (S)	23 (S)	8 (R)	9 (R)	20 (S)	17 (S)	17 (I)	16 (S)
S6	*S. aureus*	15 (I)	17 (V)	19 (S)	10 (R)	15 (V)	18 (S)	16 (I)	14 (S)
S7	*S. saprophyticus*	18 (S)	16 (V)	20 (S)	15 (V)	16 (V)	16 (S)	17 (I)	15 (S)
S8	*E. faecalis*	13 (V)	16 (I)	21 (S)	11 (R)	14 (V)	10 (R)	14 (R)	13 (S)

Values represent the zone of inhibition diameter in mm; classification in parentheses. AK, Amikacin; CIP = Ciprofloxacin; AMP = Ampicillin; ERY = Erythromycin; PEN-G = Penicillin-G; STR = Streptomycin; TE = Tetracycline; GM = Gentamicin. R = Resistant; I = Intermediate; S = Susceptible; V = Variable.

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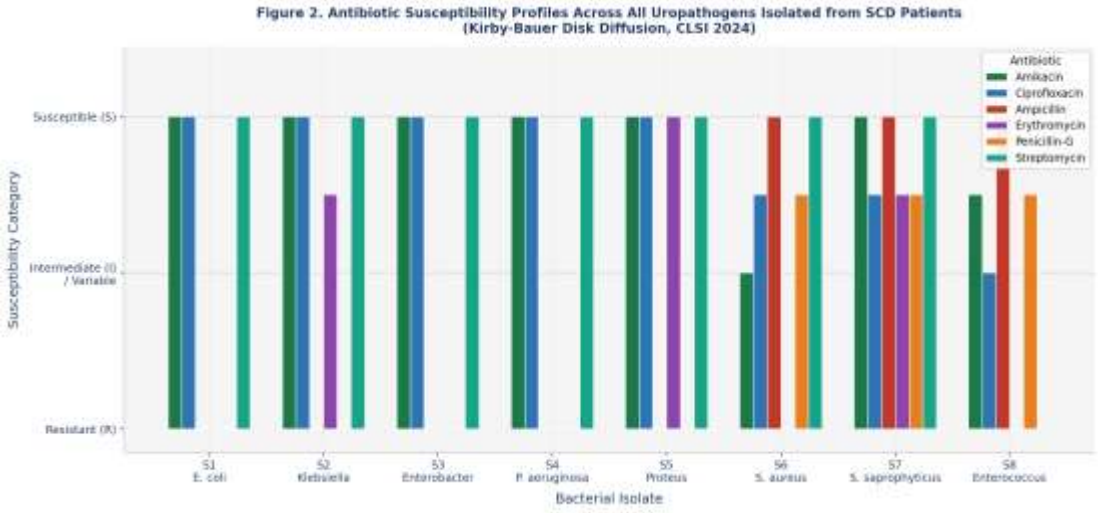

Figure 2. *Antibiotic susceptibility category profiles for all eight uropathogens recovered from urine specimens of patients with SCD. Each cluster represents an antibiotic, with individual bars denoting the susceptibility outcome for each isolate (S1–S8). Data were derived from the Kirby-Bauer disk diffusion method (CLSI 2024).*

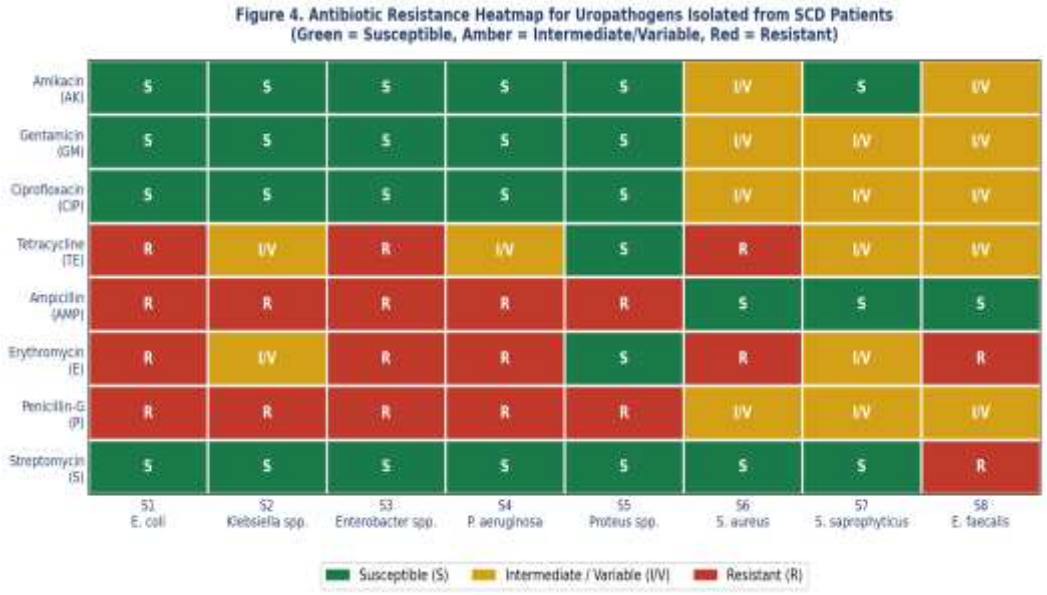

Figure 3. *Comprehensive resistance heatmap illustrating susceptibility outcomes for all eight uropathogens against each of the eight antibiotics tested (Kirby-Bauer disk diffusion, CLSI, 2024). Green = Susceptible; Amber = Intermediate/Variable; Red = Resistant.*

DISCUSSION

The present investigation documented a bacteriologically diverse array of uropathogens in patients with SCD, with gram-negative bacilli accounting for 62.5% of the isolate pool, a proportion in close agreement with globally reported figures for UTIs in SCD cohorts (Opoku-Asare et al., 2023; Ibrahim et al., 2022). The primacy of E. coli (S1) among the recovered organisms replicates a well-documented pattern across African, Middle Eastern, and Indian study populations, where this species constitutes 40–60% of uropathogens in patients with SCD (Sangeda & Manyanga, 2024; LaLhMunSangi et al., 2022). Its virulence attributes, notably type 1 and P fimbriae mediating uroepithelial adhesion and siderophore-mediated iron acquisition, confer a competitive advantage in the urinary

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microenvironment that is further accentuated by the hyposthenuria and tubular dysfunction characteristic of SCD nephropathy.

The universally observed resistance to ampicillin and penicillin-G across all gram-negative isolates constitutes a clinically critical finding. Resistance rates exceeding 80% against ampicillin have been consistently documented in sub-Saharan African and Indian cohorts of patients with SCD (Abayneh et al., 2018; Sangeda & Manyanga, 2024). This resistance phenotype reflects the pervasive horizontal dissemination of plasmid-encoded β-lactamase genes, including blaCTX-M, blaTEM, and blaSHV, among commensal and pathogenic Enterobacteriaceae in these geographic contexts (Mohammed et al., 2024; Bebe et al., 2020). The practical clinical implication is unambiguous: empirical prescribing of ampicillin or co-trimoxazole for UTIs in patients can cannot be justified without prior microbiological confirmation.

The preserved susceptibility of most gram-negative isolates to amikacin and ciprofloxacin translates into directly actionable therapeutic guidance. This finding parallels evidence from Tanzania, Nigeria, and Ghana, where aminoglycosides and fluoroquinolones are similarly identified as the most dependable agents for UTIs in patients with SCD (Ibrahim et al., 2022; Donkor et al., 2017; Garga et al., 2024). The exclusive susceptibility of *P. aeruginosa* (S4) to these two antibiotic classes reflects its well-characterized. The natural resistance methods include a few key factors: the outer membrane becomes less permeable due to mutations in the OprD porin, the MexAB-OprM efflux pump is constantly active, and there is an AmpC β-lactamase that is coded in the chromosome. Together, these factors make standard treatments with β-lactam and macrolide antibiotics ineffective (Poole, 2011; Verdial et al., 2023). Identifying the specific species and testing for their resistance to treatments are essential parts of the diagnostic process.

The isolation of gram-positive organisms, particularly *E. faecalis* and coagulase-positive *staphylococci*, deserves clinical consideration. Enterococcal UTIs present a therapeutic dilemma owing to intrinsic high-level aminoglycoside resistance and frequent co-resistance to fluoroquinolones, necessitating the consideration of ampicillin or vancomycin in susceptible strains. These organisms are increasingly acknowledged as contributors to UTIs in SCD, particularly in the context of asymptomatic bacteriuria (Musonda et al., 2020; Cumming et al., 2006).

From a public health perspective, the resistance landscape mapped in this investigation underscores the pressing necessity for formalized antimicrobial stewardship programmes specifically calibrated to SCD populations. Mandatory pre-treatment urine culture, informed by regularly updated institution-specific antibiograms, would curtail injudicious empirical prescribing and decelerate resistance selection. Preventive strategies, including systematic screening for asymptomatic bacteriuria at each clinic visit and prompt treatment of all microbiologically confirmed infections, are equally critical for averting progressive renal deterioration, which is a leading cause of long-term morbidity in SCD (Cumming et al., 2006; WHO, 2023).

CONCLUSION

This study established that UTIs in patients with SCD are primarily driven by gram-negative uropathogens, with *E. coli* emerging as the predominant species. Universal resistance to ampicillin and penicillin-G renders these agents therapeutically obsolete for empirical use in this population group. Amikacin and ciprofloxacin demonstrated the broadest and most consistent antimicrobial activity across the isolate panel and represented the most defensible empirical choices pending culture and susceptibility results. The implementation of standardized urine culture protocols, routine antibiogram surveillance, and formally structured antimicrobial stewardship

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programs are essential clinical and public health imperatives to ensure effective, evidence-based UTI management in individuals with sickle cell disease in Chandrapur and comparable endemic regions.

DECLARATIONS

CONFLICT OF INTEREST: The authors declare no competing financial, personal, or professional interests.

ETHICAL APPROVAL: The investigation was conducted in strict compliance with institutional ethical guidelines. All adult participants gave their written consent, and for minors, we got their agreement along with permission from their parents or guardians before collecting any samples.

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