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RESEARCH ARTICLE **OPEN ACCESS**

Prevalence of ESBL Genes Among *Pseudomonas aeruginosa* and *Acinetobacter baumannii* Isolated from Female UTI Patients in Dharwad, Karnataka, India

Nivedita R. Bashetti[1], Renukaradhya K. Math[2], Ramesh S. Gani[3], Rajendra Nerli[4], Chandrashekhar Unakal[5] and Murigendra B. Hiremath[1,*]

[1]*Department of Microbiology and Biotechnology, Karnatak University, Dharwad, Karnataka, India*
[2]*SDM Research Institute for Biomedical Sciences, Shri Dharmasthala Manjunatheshwara University, Dharwad, Karnataka, India*
[3]*Department of Industrial Chemistry, Mangalore University, Mangalore, Karnataka, India*
[4]*Department of Urology, KLES Kidney Foundation, KLES Dr. Prabhakar Kore Hospital, Belgaum, Karnataka, India*
[5]*Department of Para-clinical Sciences, University of the West Indies, St. Augustine, Trinidad and Tobago*

Abstract:

Background: The emergence of ESBL-producing non-fermenters, such as *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, is a growing concern, particularly in Urinary Tract Infections (UTIs). This study investigates the prevalence and distribution of ESBL genes among these pathogens isolated from female UTI patients in Dharwad, Karnataka, India.

Methods: A total of 713 urine samples were processed. Bacterial identification was conducted using culture and molecular methods. Phenotypic ESBL detection and quantitative PCR (qPCR) were employed to identify specific ESBL genes.

Results: Out of 713 samples, 665 were culture positive. *Pseudomonas aeruginosa* (56 isolates) and *Acinetobacter baumannii* (29 isolates) were identified. ESBL production was phenotypically confirmed in 69.6% and 48.3% of *P. aeruginosa* and *A. baumannii*, respectively. Genotypic screening revealed the high prevalence of bla_{SHV}, bla_{TEM}, and bla_{OXA-23} genes. bla_{KPC} was only observed in *P. aeruginosa*.

Discussion: The study demonstrates a high burden of ESBL-producing non-fermenters in UTI patients. Molecular surveillance is critical for effective antibiotic stewardship.

Conclusion: High proportions of ESBL genes, particularly blaSHV (84.6%) and blaTEM (69.2%), were detected in Pseudomonas aeruginosa, indicating strong resistance potential. In Acinetobacter baumannii, the predominance of blaOXA-23 (78.6%) confirms its major role in carbapenem resistance.

Keywords: ESBL, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, Prevalence, Antibiotic resistance, qPCR.

*Address correspondence to this author at the Department of Microbiology and Biotechnology, Karnatak University, Dharwad, Karnataka; E-mail: murigendra@gmail.com

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1. INTRODUCTION

Urinary Tract Infections (UTIs) remain one of the most common bacterial infections affecting individuals globally, with a significantly higher prevalence among women due to anatomical and physiological factors [1]. Although *Escherichia coli* is traditionally regarded as the predominant uropathogen, the role of non-fermenting Gram-negative bacilli (NFGNB), particularly *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, in complicated and healthcare-associated UTIs is becoming increasingly significant [2, 3]. These opportunistic pathogens are associated with Multidrug Resistance (MDR), prolonged hospital stays, and increased morbidity and mortality. A major mechanism underlying antibiotic resistance in these pathogens is the production of Extended-Spectrum β-Lactamases (ESBLs), enzymes capable of hydrolyzing a wide range of β-lactam antibiotics, including penicillins, cephalosporins, and aztreonam [4]. While ESBL production is well documented in Enterobacteriaceae, there is growing evidence of ESBL gene dissemination among NFGNB, including *P. aeruginosa* and *A. baumannii*, largely mediated by mobile genetic elements such as integrons, plasmids, and transposons [5, 6]. In the Indian context, the burden of ESBL-producing uropathogens is especially concerning due to high rates of antibiotic misuse, over-the-counter availability of antimicrobials, and suboptimal infection control practices [7]. Regional surveillance studies from various parts of India have reported alarming levels of MDR *P. aeruginosa* and *A. baumannii* isolates harboring ESBL genes such as bla_{TEM}, bla_{SHV}, and $bla_{CTX\text{-}M}$ [8, 9]. However, data on the prevalence of these resistance determinants among UTI isolates, particularly from female patients in Tier-II cities such as Dharwad, Karnataka, remain limited. Although *Escherichia coli* is considered the most prevalent uropathogen in community-acquired UTIs, *Acinetobacter baumannii* and *Pseudomonas aeruginosa* have emerged as clinically significant nosocomial pathogens, especially in complicated and catheter-associated UTIs. In immunocompromised and hospitalized patients, these organisms are frequently isolated [10]. They exhibit both intrinsic and acquired resistance mechanisms, particularly against β-lactams and carbapenems, which often results in ineffective treatment [11]. In addition, the World Health Organization (WHO) identifies *P. aeruginosa* and *A. baumannii* as critical-priority pathogens due to their ability to acquire and disseminate resistance genes *via* mobile genetic elements [12]. Given the critical public health implications of rising ESBL-mediated resistance, localized epidemiological studies are essential for guiding empirical therapy and implementing targeted antimicrobial stewardship strategies. This study aims to determine the prevalence of ESBL genes among *P. aeruginosa* and *A. baumannii* isolated from female UTI patients in Dharwad, Karnataka, India, and to assess their resistance patterns in the context of recent antimicrobial resistance trends.

2. MATERIALS AND METHODS

2.1. Study Design and Sample Collection

The prospective study was conducted in the Department of Biotechnology and Microbiology, Karnatak University Dharwad (Karnataka, India), between June 2021 to January 2023. Samples were collected from local hospitals, outpatient clinic and private diagnostic laboratories.

2.2. Isolation and Identification of Uropathogens

Urine samples were cultured on Blood agar, Luria-Bertani agar, Cysteine Lactose Electrolyte-Deficient (CLED) agar, and MacConkey agar using the calibrated loop streak method. Plates were incubated aerobically at 37°C for 24 hours. Significant bacteriuria was defined as growth of $\geq 10^5$ CFU/mL. Isolates were identified using standard microbiological techniques and confirmed with the VITEK 2 Compact system (bioMérieux, France). Isolates were further subjected to molecular identification by 16S rRNA sequencing using the Sanger sequencing method.

2.3. Statistical Analysis

The association between various categorical risk factors and UTI positivity was analyzed using the Chi-square test of independence. P-values were calculated in GraphPad Prism version 10 (GraphPad Software, San Diego, CA, USA). A p-value <0.05 was considered statistically significant.

2.4. Antibiotic Susceptibility Testing

Antimicrobial susceptibility testing was performed by the Kirby-Bauer disk diffusion method on Mueller-Hinton agar, following Clinical and Laboratory Standards Institute (CLSI) guidelines [13]. The antibiotics tested included ampicillin (10µg), ciprofloxacin (30µg), imipenem (10µg), gentamicin (10µg), cefuroxime (30µg), ceftazidime (30µg), ceftriaxone (30µg), cefotaxime (30µg), cephalexin (30µg), cefepime (30µg), and nitrofurantoin (300µg). The isolates were distributed uniformly with a sterile, dry cotton swab on the Mueller-Hinton agar plate. Antibiotic discs were placed using sterile forceps. After incubating the plates at 37°C for 24 hrs, the results were interpreted according to Clinical Laboratory Standard Institute guidelines. For phenotypic detection of ESBL-producing isolates, isolates were selected based on resistance patterns to third-generation cephalosporins.

2.5. Screening of ESBL Producers

ESBL phenotypic detection was performed using the disc diffusion test. The Phenotypic Confirmatory Disc Diffusion Test (PCDDT) combines ceftazidime and clavulanic acid (30/10 µg), cefotaxime and clavulanic acid (30/10 µg), and ceftazidime (30 µg) and cefotaxime (30 µg) discs on Muller-Hinton agar with respective isolates. We kept the plates at 37 °C for 24 hours. An ESBL producer is defined as a zone diameter increase of greater than 5 mm for either ceftazidime/clavulanic acid disc or cefotaxime/clavulanic acid disc, compared to ceftazidime or cefotaxime alone.

2.6. Molecular Detection of Resistance Genes

Genomic DNA was extracted from isolates using a commercial DNA extraction kit. Quantitative PCR was used to detect ESBL and Carbapenemase genes, including bla_{TEM}, bla_{SHV}, bla_{GES}, bla_{KPC}, bla_{OXA-23}, bla_{OXA-48}. The specific primer sets used for each gene are listed in Table **1**. A loopful of bacterial isolates was suspended in 50 µL of ultrapure water.The suspension was centrifuged at 14000 rpm for 5 minutes, and the pellet was heated for 10 minutes at 95 °C to lyse the cells. The supernatant (30 µl) was collected and used as the DNA template for the reaction. qPCR was performed using the CFX96™ Real-Time PCR Detection System (Bio-Rad, USA). Each 20 µL reaction mixture contained 1 µL of DNA template, 0.8 µL each of forward and reverse primers (10 µM), 10 µL of 2× SYBR Green Master Mix (Bio-Rad), and nuclease-free water to make up the final volume. qPCR cycling conditions include Initial denaturation at 95 °C for 5 minutes, followed by 35 cycles of denaturation at 95 °C for 15 seconds and 60 °C for 30 seconds (combined annealing and extension), with fluorescence acquisition at the end of each cycle. A melt curve analysis was performed at the end of the run to confirm amplification specificity. qPCR was used to detect the presence or absence of the genes, and also ct values are qualitatively interpreted. All reactions were run in triplicate to ensure consistency and produce reliability of results. *Klebsiella pneumoniae* ATCC 700603 was used as a positive control for $bla_{SHV,}$ and for other genes, standard lab controls were used. Amplified products were visualized using gel electrophoresis, and melt curve analysis was performed to confirm gene specificity (Fig. **S2**).

3. RESULTS

3.1. Prevalence of Isolates and Patient Demographics

Out of 713 urine samples collected from female UTI patients, 665 (93.3%) yielded positive urine cultures. Among these, *Pseudomonas aeruginosa* was isolated in 56 cases (8.4%) and *Acinetobacter baumannii* in 29 cases (4.4%). The remaining isolates were predominantly Enterobacteriaceae (data not shown). The prevalence of *P. aeruginosa* and *A. baumannii* infections was highest among patients aged 41–50 years, followed by those aged 61–70 years, as illustrated in Fig. (**1**). The 16S rRNA gene sequences for representative *P. aeruginosa* (accession no. OQ363380) and *A. baumannii* (accession no. OQ363378) isolates were deposited in GenBank for reference. Among risk factors, only catheter use showed a statistically significant association ($p < 0.05$) with both species, suggesting it may be a significant risk factor for *A. baumannii* infections in our study (Table **2**).

Table 1. List of primers used for qPCR amplification of ESBLs.

Genes	Primer Sequence (5′ - 3′) FW: Forward RV: Reverse	Product Size (bp)	Annealing Temp (°C)
bla_{TEM}	FW: GTATCCGCTCATGAGACAATAACC RV: CCAATGCTTAATCAGTGAGGCACC	918	42.5
bla_{SHV}	FW: CGCCTGTGTATTATCTCCCTG RV: TTGCCAGTGCTCGATCAGCG	842	44.5
bla_{GES}	FW: CTATTACTGGCAGGGATCG RV: CCTCTCAATGGTGTGGGT	594	51
bla_{KPC}	FW: CATTCAAGGGCTTTCTTGCTGC RV: ACGACGGCATAGTCATTTGC	538	51
bla_{OXA-23}	FW: GTGGTTGCTTCTCTTTTTCT RV: ATTTCTGACCGCATTTCCAT	736	50.3
$bla_{OXA-448}$	FW: AACGGGCGAACCAAGCATTTT RV: TGAGCACTTCTTTTGTGATGGCT	585	50.3

Table 2. Association of the various risk factors of UTI.

Risk factors	Frequency (%)	
	Pseudomonas aeruginosa	*Acinetobacter baumannii*
Cathetar use	8 (16.1)	14 (48.3)
Menopause	13 (23.2)	11 (37.9)
History of UTI	12 (21.4)	5 (17.2)
Diabetes	9 (16.1)	8 (27.6)
Sexual activity	8 (16.1)	7 (24.1)

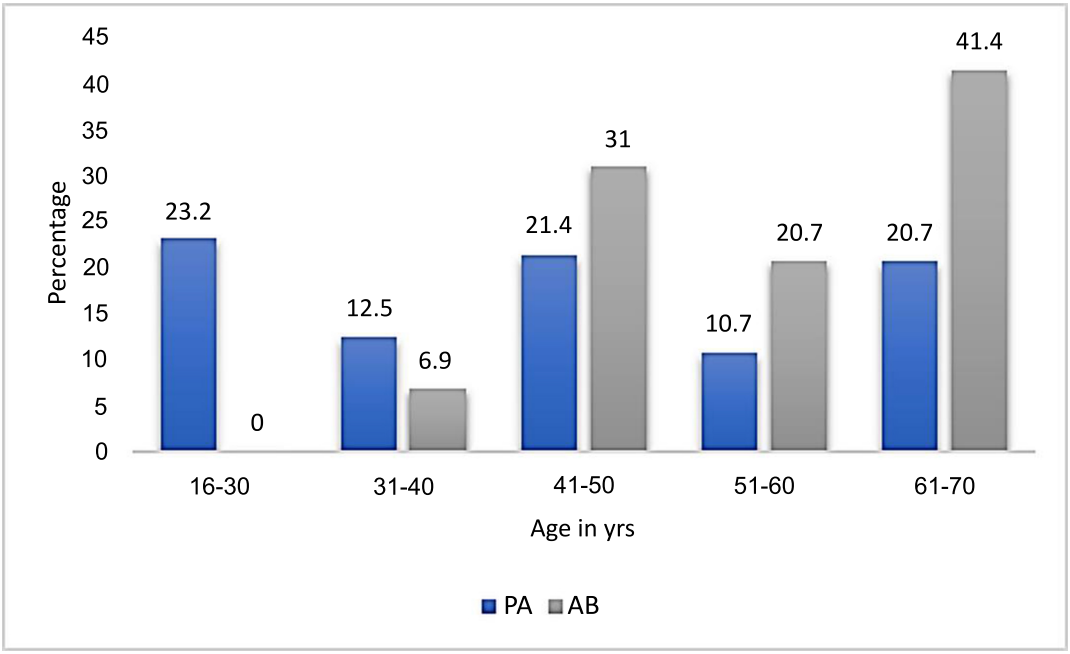


Fig. (1). Prevalence of uropathogens in female patients with UTI. (Data reproduced from Bashetti *et al.*, 2024) [27].

3.2. Antimicrobial Susceptibility Patterns

Both *P. aeruginosa* and *A. baumannii* isolates exhibited high levels of antimicrobial resistance, albeit with differing profiles. *P. aeruginosa* showed nearly universal resistance to certain first-line UTI drugs (notably 100% resistance to nitrofurantoin) and to third-generation cephalosporins (*e.g.*, 80.3% of isolates were resistant to ceftazidime). However, this species retained relatively higher susceptibility to a few antibiotics: only 16.1% of *P. aeruginosa* isolates were resistant to ciprofloxacin, followed by low resistance rates to gentamicin (21.4%) and imipenem (23.2%). Resistance to cefotaxime was moderate (observed in 57.2% of isolates). In contrast, *A. baumannii* demonstrated extreme drug resistance to many tested agents. Notably, 75% of *A. baumannii* isolates were resistant to ampicillin, and similarly high resistance frequencies were observed for the oral cephalosporin cefalexin and the parenteral cephalosporins ceftazidime and ceftriaxone (each with over half of the isolates being non-susceptible). All *A. baumannii* isolates were also resistant to nitrofurantoin. These antimicrobial resistance patterns for both organisms are summarized in Table **3**.

Table 3. Antibiotic resistance pattern of the uropathogens.

Antibiotics	Resistance (%)	
	***P.aeruginosa* (n=56)**	***A. Baumannii* (n=29)**
Ampicillin	47(83.9)	22(75.9)
Ciprofloxacin	9(16.1)	7(24.1)
Imipenam	13(23.2)	4(13.8)
Gentamicin	12(21.4)	11(37.9)
Cefuroxime	35(62.5)	13(44.8)
Ceftazidime	45(80.3)	17(58.6)
Ceftriaxone	29(51.8)	17(58.6)
Cefotaxime	32(57.1)	14(48.3)
Cephalaxein	43(76.8)	19(65.5)
Cefepime	39(69.6)	9(31)
Nitrofurantoin	56(100)	29(100)

Note: *p*-value - <0.01 for all antibotics tested.

Table 4. Prevalence of the ESBL genes among *P. aeruginosa* and *A. baumanii*.

Genotype	*Pseudomonas aeruginosa* (n=39)	*Acinetobacter baumannii* (n=14)
bla_{TEM}	27 (69.2)	9 (64.3)
bla_{SHV}	33 (84.6)	8 (57.1)
bla_{GES}	11(28.2)	6 (42.8)
bla_{KPC}	19 (48.7)	-
bla_{OXA-23}	32 (82)	11 (78.6)
bla_{OXA-48}	28 (71.8)	5 (35.7)
$bla_{TEM + SHV}$	13 (33.3)	7 (50)
$bla_{OXA-23 + OXA-48}$	9(23.1)	-

3.3. Phenotypic Detection of ESBL Production

Phenotypic screening tests identified a subset of the isolates as Extended-Spectrum Β-Lactamase (ESBL) producers. Among the 56 *P. aeruginosa* isolates, 39 (69.6%) were confirmed to be ESBL producers, and among the 29 *A. baumannii* isolates, 14 (48.3%) were ESBL-positive. Fig. (**S1**) shows a representative phenotypic confirmatory test for ESBL production in these organisms. All ESBL-producing *P. aeruginosa* and *A. baumannii* isolates were selected for further molecular characterization.

3.4. Genotypic Characterization of ESBL Genes by qPCR

All phenotypic ESBL-positive isolates were subjected to quantitative PCR analysis targeting ESBLs and carbapenemase genes (bla_{TEM}, bla_{SHV}, bla_{GES}, bla_{KPC}, bla_{OXA-23}, and bla_{OXA-48}). Genotypic results revealed that in *P. aeruginosa* (n = 39), the most prevalent ESBL gene was bla_{SHV}, detected in 84.6% of isolates, followed by bla_{TEM} (69.2%) and bla_{GES} (28.2%). The bla_{KPC} gene was also detected in *P. aeruginosa* (in nearly half of the ESBL-positive *P. aeruginosa* isolates), whereas bla_{KPC} was not found in any *A. baumannii* isolate. In *A. baumannii* (n = 14), the carbapenemase gene bla_{OXA-23} was the most common, present in 78.6% of isolates, and bla_{SHV} was identified in 57.1%. The bla_{OXA-48} gene appeared at a lower frequency in *A. baumannii* (detected in roughly one-third of isolates) compared to *P. aeruginosa*. Notably, bla_{TEM} was also frequently detected in *A. baumannii*, often co-occurring with bla_{SHV} in the same strains (Table **4**). In our collection, neither species showed a high prevalence of the bla_{GES} gene in *A. baumannii* (this gene was either absent or present at very low frequency in the *A. baumannii* isolates). Multiple ESBL genes were common among the tested strains. One-third (33.3%) of the ESBL-producing *P. aeruginosa* carried more than two of the targeted resistance genes concurrently, and fully half (50%) of the ESBL-producing *A. baumannii* harbored at least two or more ESBL genes (often including both bla_{TEM} and bla_{SHV}). The detailed prevalence of each bla gene and its co-occurrence in these isolates is presented in Table **3**. Cycle threshold (Ct) values for the amplification of each gene target (indicative of gene abundance) are provided in Table **S1**. Finally, the qPCR amplification and melt curves confirming the specific gene amplifications are shown in Fig. (**2**) demonstrating successful amplification of the target ESBL genes in the representative *P. aeruginosa* and *A. baumannii samples*.

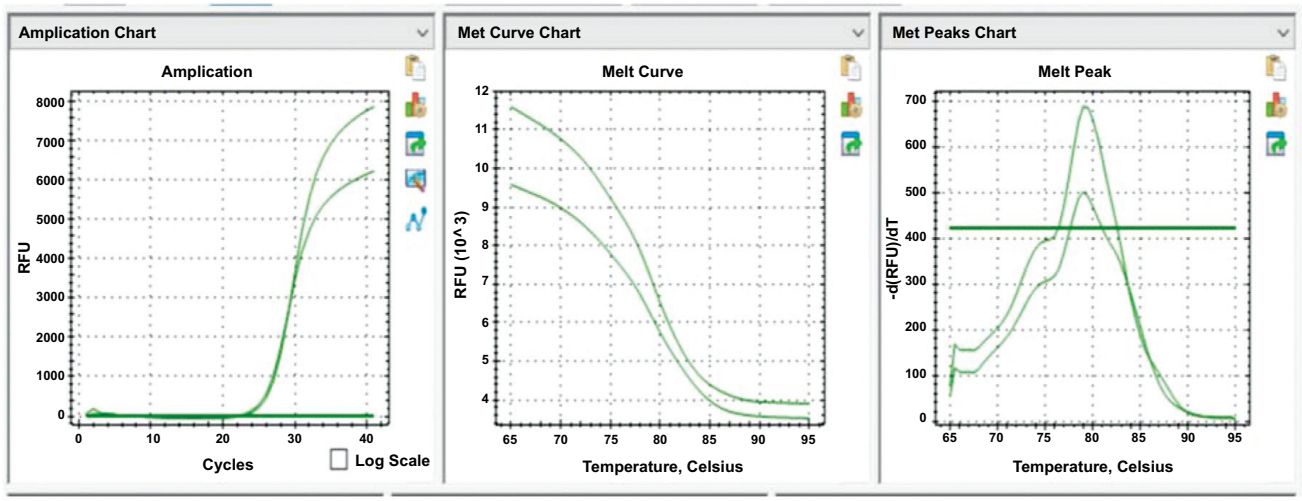
4. DISCUSSION

The rise of Multidrug-Resistant (MDR) uropathogens, particularly among non-fermenting Gram-negative bacteria such as *P. aeruginosa* and *A. baumannii*, represents a significant clinical challenge both globally and within hospital settings in India. This study reveals a substantial prevalence of ESBL-producing *P. aeruginosa* and *A. baumannii* among female UTI patients in Dharwad, Karnataka, highlighting both organisms as critical nosocomial threats. Notably, ESBL production was phenotypically confirmed in 69.6% of *P. aeruginosa* and 48.3% of *A. baumannii* isolates, a pattern consistent with previous reports suggesting increasing resistance among non-fermenters [5, 14].

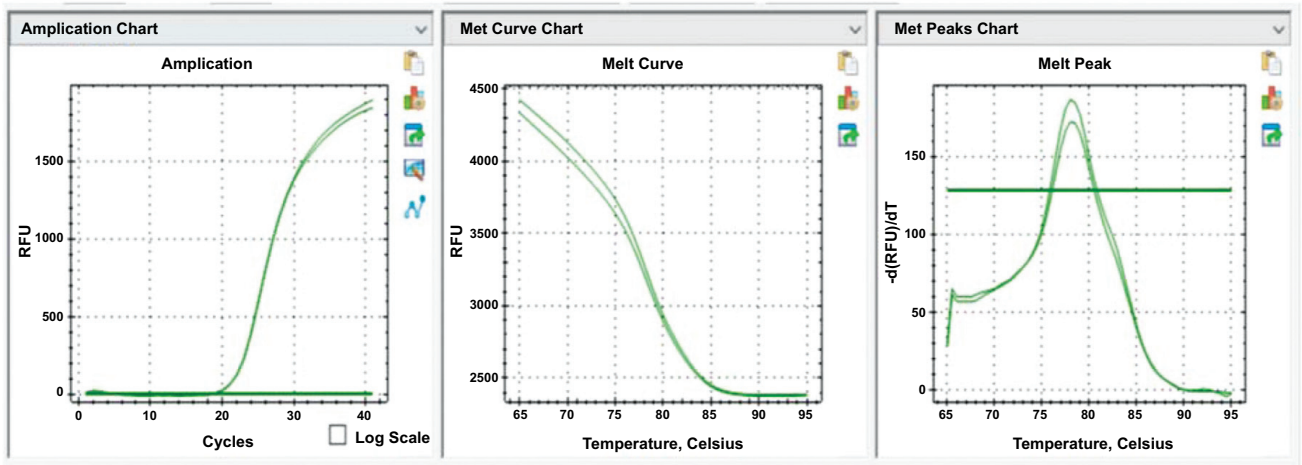
The high proportion of isolates harboring ESBL genes, particularly bla_{SHV}(84.6%) and bla_{TEM} (69.2%) in *P. aeruginosa*, underscores the genetic adaptability and resistance potential of this pathogen. Our detection rates exceed those reported in similar studies from the Middle East and South Asia, which found bla_{SHV} and bla_{TEM} in fewer than 50% of clinical *Pseudomonas* isolates [15, 16]. In *A. baumannii*, the predominance of bla_{OXA-23} (78.6%) aligns with findings from Algeria and Iran, where this gene is frequently associated with carbapenem resistance [17, 18]. The detection of bla_{OXA-48} in 71.8% of *P. aeruginosa* and 35.7% of *A. baumannii* is alarming and represents a potential reservoir for pan-drug resistance, as OXA-48-like enzymes contribute to carbapenem hydrolysis while remaining undetectable by many phenotypic tests [19]. Of concern, nearly one-third of *P. aeruginosa* isolates and half of *A. baumannii* isolates harbored two or more ESBL genes, suggesting a strong potential for horizontal gene transfer and co-selection of resistance traits. Such gene combinations may facilitate survival under a broad spectrum of antibiotic classes, especially in hospital environments where antimicrobial pressure is high [20].

2A. *Pseudomonas aeruginosa*

a. TEM



b. SHV



c. GES

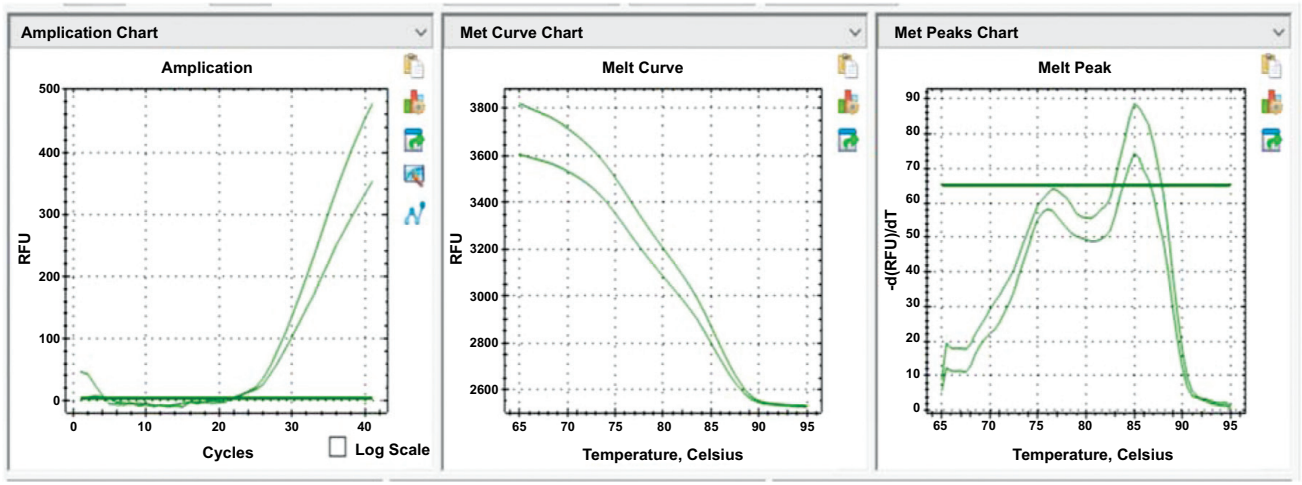
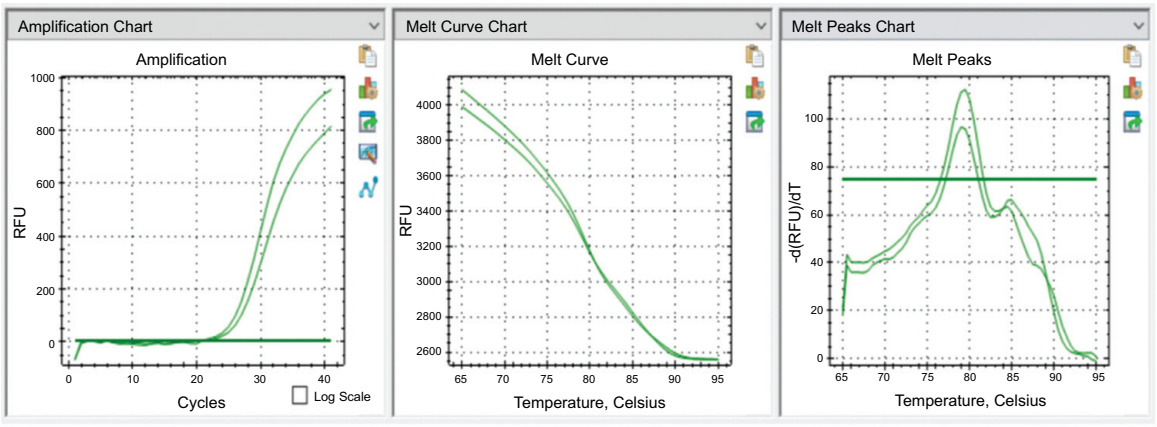
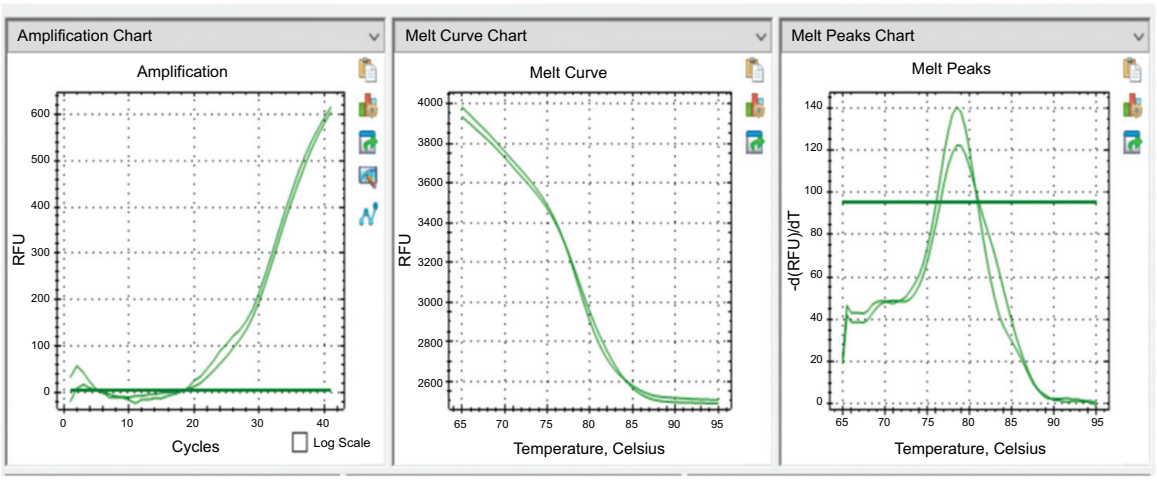


Fig. 2 contd.....

d. KPC



e. OXA - 48



f. OXA - 23

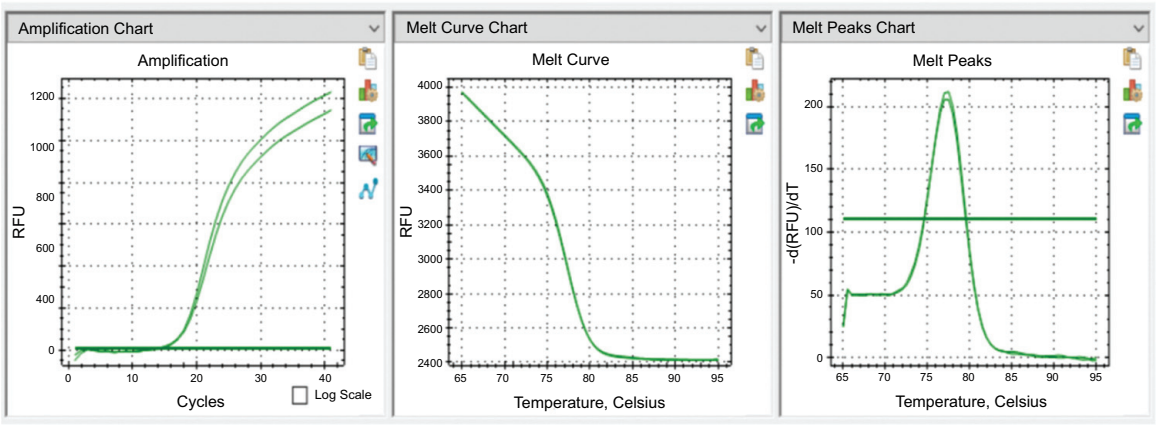
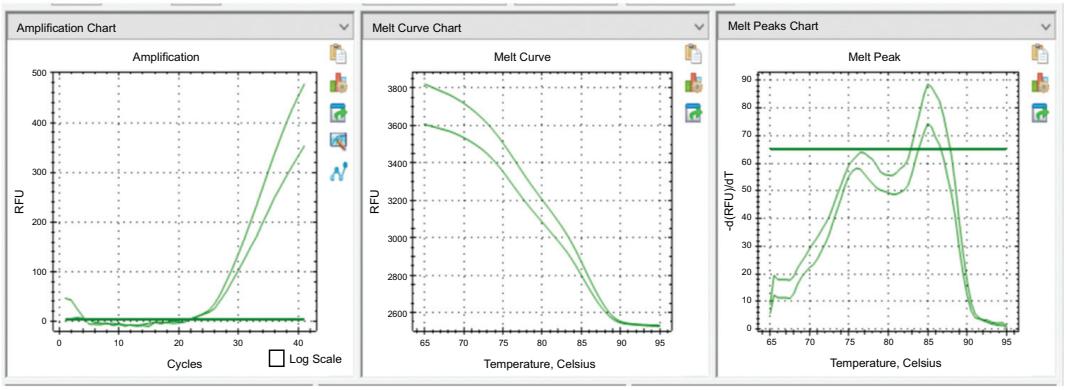


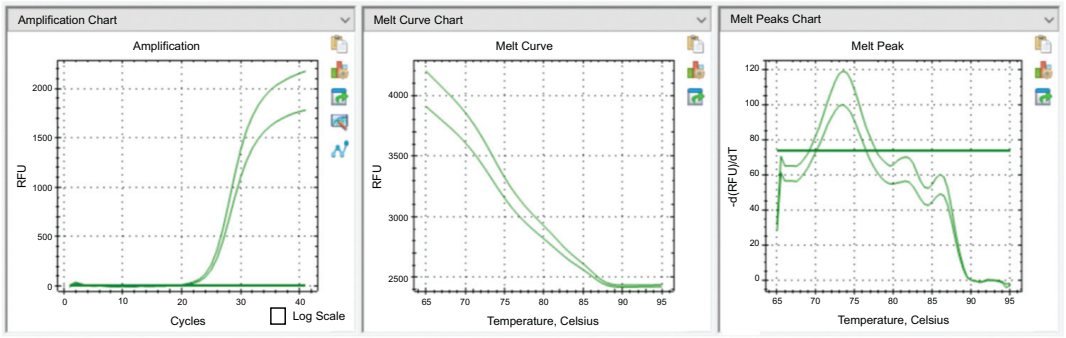
Fig. 2 contd.....

2B. *Acinetobacter baumannii*

a. TEM



b. SHV



c. GES

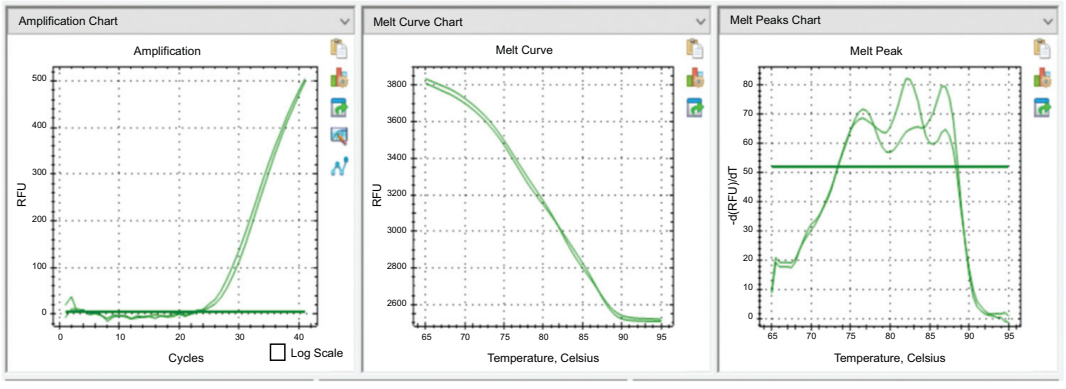
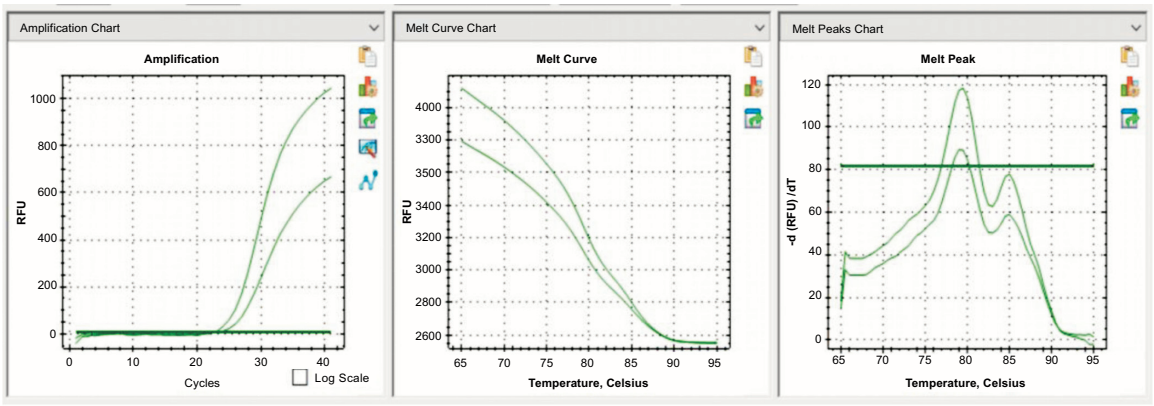
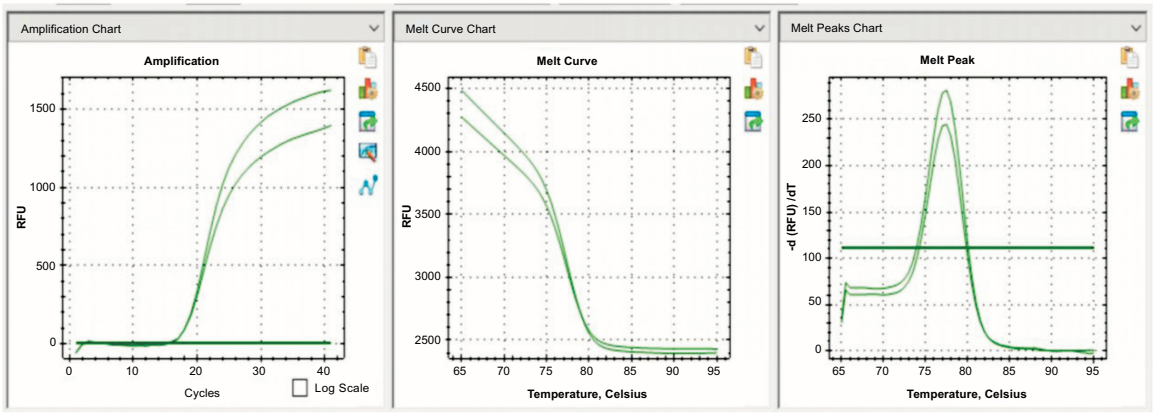


Fig. 2 contd.....

d. KPC



e. OXA -23



f. OXA-48

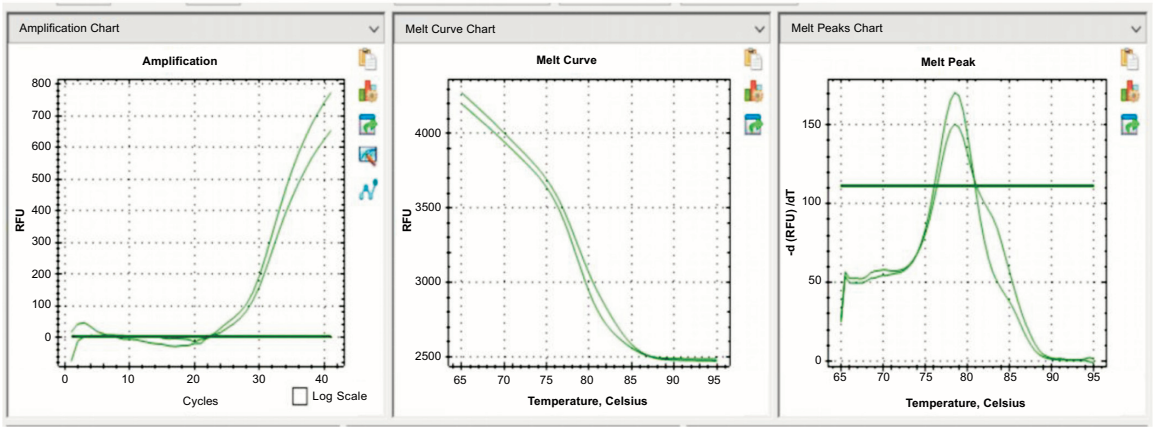


Fig. (2). PCR amplification curves of resistance genes of uropathogens.

Furthermore, greater resistance to nitrofurantoin and high resistance to β-lactams and cephalosporins, particularly to ceftazidime and cefotaxime, further reinforce the growing clinical irrelevance of these agents for empirical UTI therapy. Compared to recent data from Nepal and Poland, where ceftazidime resistance in *P. aeruginosa* ranged between 40–60%, our observed resistance rate of 80.3% suggests region-specific antimicrobial misuse or selective pressure [21, 22]. In contrast, relatively preserved susceptibility to imipenem, ciprofloxacin, and gentamicin (resistance <25%) indicates their potential role as second-line therapies, although this should be approached cautiously due to the risk of emerging carbapenem resistance [23].

Hormonal factors, such as menopause, were associated with higher rates of isolation of *P. aeruginosa* and *A. baumannii*, echoing the existing literature linking estrogen deficiency to changes in the genitourinary flora and increased infection risk [24]. The role of urinary catheterization as a prominent risk factor for *A. baumannii* infections (48.3%) further underscores the organism's opportunistic nature and its propensity to form biofilms on indwelling devices [25].

Our study demonstrates the utility of qPCR as a rapid and sensitive method for detecting ESBL genes, with Ct values indicating robust amplification efficiency across most targets. The use of melt curve analysis provided additional specificity in distinguishing amplicon identities. Taken together, these findings reinforce global and regional alerts on AMR, as outlined in the WHO priority pathogen list [26]. The presence of high-risk clones carrying multiple β-lactamase genes necessitates ongoing molecular surveillance and stringent infection control practices. Moreover, our results suggest the urgent need to revise empirical UTI treatment protocols in this region, incorporating local resistance data to avoid therapeutic failures and reduce selective pressure.

5. LIMITATION

This study has some limitations. The limited number of isolates of *P. aeruginosa* and *A. baumannii* may affect generalizability. Resource constraints hindered the incorporation of the $bla_{\text{CTX-M}}$ gene, even though they have worldwide significance. Resistance mechanisms such as efflux pumps and porin loss were not investigated. Molecular typing methods are important to study clone-relatedness and transmission of resistant strains, this will be considered in future studies. In addition, the study is limited by its single-center design and sequencing to determine the genetic context of the ESBL genes.

CONCLUSION

This study indicates that *Pseudomonas aeruginosa* and *Acinetobacter baumannii* showed high incidence of resistance genes bla_{TEM}, bla_{SHV}, and $bla_{\text{OXA-23}}$. These uropathogens represent a wide range of clinical complications due to their high rates of multidrug resistance, co-occurrence of other resistance genes, and significant association with risk factors like catheterization. Although antibiotics such as nitrofurantoin, gentamicin, ampicillin, and cefotaxime are not recommended against these uropathogens, we evaluated them in our study due to their empirical use by clinicians in our study setting, which further highlights the implications of local prescribing patterns. The use of qPCR has enhanced its importance in monitoring antibiotic resistance by enabling quick, precise identification of ESBL and carbapenemase genes. These findings emphasize the critical need for antimicrobial stewardship, routine molecular diagnostics, and infection control practices. Continuous surveillance and targeted antimicrobial therapy to manage and control the spread of resistant strains are necessary.

AUTHORS' CONTRIBUTIONS

The authors confirm their contributions to this paper as follows: C.U.: Responsible for the study conception and design; M.H. and R.G.: Contributed to conceptualization. R.N. and R.M.: Responsible for data curation; N.B.: Conducted the investigation. All authors reviewed the results and approved the final version of the manuscript.

LIST OF ABBREVIATIONS

UTIs	=	Urinary Tract Infections
qPCR	=	Quantitative Polymerase Chain Reaction
NFGNB	=	Non-fermenting Gram-negative Bacilli
MDR	=	Multidrug Resistance
Ct	=	Cycle Threshold
WHO	=	World Health Organization
ESBLs	=	Extended-Spectrum β-Lactamases
CLED	=	Cysteine Lactose Electrolyte-Deficient

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was approved by the KLES Kidney Foundation Institutional Ethics Committee, Belagavi, India with approval Number KLESKF/ IEC/23/017.

HUMAN AND ANIMAL RIGHTS

All procedures performed in studies involving human participants were in accordance with the ethical standards of institutional and/or research committee and with the 1975 Declaration of Helsinki, as revised in 2013.

CONSENT FOR PUBLICATION

Informed consent was obtained from all participants/guardians.

AVAILABILITY OF DATA AND MATERIALS

The data and supportive information are available within the article.

FUNDING

None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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SUPPLEMENTARY MATERIAL

Supplementary material is available on the Publisher's website along with the published article.

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