

Molecular Detection of Virulence-Associated, Regulatory, and Antimicrobial-Resistance Genes in *Serratia marcescens* Isolated from Urinary Tract Infections

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ABSTRACT

Objective: *Serratia marcescens* is an opportunistic, gram-negative bacterium related to healthcare-associated and other clinically important infections. The aim of this work was to evaluate some virulence, regulatory and antimicrobial resistance genes of *S. marcescens* strains obtained from UTI patients in Mosul city, Iraq.

Methods: During February – June 2024, 150 samples were taken from Midstream urine of UTI patients. Conventional culture and biochemical testing were used to characterize presumptive isolates and VITEK-2 Compact system was used for identification; and 16S rRNA gene sequencing used for additional molecular identification. The obtained isolates were screened for *flhD*, *pigP*, *shlA*, *phlA*, *aac(6')-Ib-cr*, *blaIMP* and *blaOXA-48* by conventional PCR.

Results: *S. marcescens* was isolated from 150 urine samples with 8 isolates recovered (5.3%). 100% of all isolates were positive for the virulence associated genes *shlA*, *phlA*, *flhD* and *pigP*, as well as the regulatory genes. The resistance-associated gene *aac(6')-Ib-cr* was found in 100% of the eight isolates. Both of the carbapenemase associated genes, *blaIMP* and *blaOXA-48*, were present in 7 of 8 isolates (87.5%). The results revealed high prevalence of the examined virulence- and resistance-associated genetic determinants, and they were often found in the same organisms. The results are the molecular detection of the targeted genes and not necessarily the expression of these genes or the corresponding phenotypic characteristics.

Conclusion: This small number of urinary *S. marcescens* isolates contained multiple virulence-associated/regulatory and antimicrobial-resistance determinants. These observations are at the molecular level and should not be interpreted as population level resistance or functional virulence or phenotypic antimicrobial resistance, as only eight isolates were examined and no such testing was performed. Additional studies with larger numbers of patients incorporating phenotypic susceptibility testing and functional assays with more sophisticated molecular typing will be necessary to determine the clinical and epidemiological importance of the results.

Introduction

Serratia marcescens is an opportunistic Gram-negative bacterium that is seen as a cause of infections in adult and neonatal patients, especially in intensive-care units (ICUs) [1] [2]. It was previously considered to be not a significant pathogen, but in recent years has been identified as a pathogen in neonatal intensive care units (NICUs), intensive care units (ICUs), and other hospital wards [3]

The impact of *S. marcescens* infection can be underestimated(4). Infected or colonized neonates can be a source of transmission in neonatal units(5), and bacteremia has been reported in neonates and in all age groups(6). The organism can survive in hospital environment and outbreaks showing more than one clone (more than one strain) and an unknown source are hard to investigate [7] [8] [9].

Pathogenicity of *S. marcescens* is multifactorial. The targets in the current study reflect different virulence-related and/or regulatory functions. ShlA is a pore-forming hemolysin and PhlA a phospholipase involved in membrane damage, both of which are related to cytotoxicity. FlhD belongs to the flagellar biogenesis and motility control FlhDC transcriptional regulatory complex. PigP is a pleiotropic transcriptional regulator which is involved in production of prodigiosin, serratamolide, swarming and hemolysis. While the presence of PigP can be correlated with quorum-sensing regulated networks in *Serratia*, this should not be equated with direct measurement of quorum-sensing activity. Other factors, such as fimbrial and adhesion factors, are also important to attachment, colonization, biofilm formation, tissue injury and dissemination [10][11][12].

ShlA has been experimentally correlated with hemolytic/cytotoxic effects including in a mouse lung-infection model [13]. It also has been associated with inflammatory signaling, epithelial cell injury and inter-host-cell spread of bacteria [3]. ShlA is important for these activities when evaluating the pathogenic potential of clinical isolates.

PhlA activity is another way of causing damage to the membrane. Due to its phospholipase activity, PhlA produces lysophospholipid products which may cause the destabilization of target-cell membranes and affect the cytotoxic and hemolytic activity [3]. In this regard its presence in combination with other virulence factors may represent an additional virulence factor to the invasive potential of *S. marcescens*.

One more clinical concern of *S. marcescens* is its antimicrobial resistance. Important β -lactam agents may lose activity through acquired β -lactamases or carbapenemases, and the plasmid-mediated *aac*(6')-Ib-cr may usually result in decreased susceptibility to aminoglycosides and fluoroquinolones. However, intrinsic and acquired resistance mechanisms can significantly limit therapeutic options [14] [15] [16] [17]. *S. marcescens* has been reported in various clinical and geographic locations resistant to carbapenems [18][19]. In this context, the present study aimed to investigate the presence of seven selected molecular targets identified in urinary isolates from Mosul; the *flhD*, *pigP*, *shlA*, and *phlA* genes involved in virulence; the *aac*(6')-Ib-cr gene for plasmid mediated quinolone-resistance; and the *blaIMP* and *blaOXA-48* genes for the production of carbapenemases.

Methods

Sample Collection

During the period of February to June 2024, 150 midstream urine samples were obtained from people with clinically diagnosed UTI. The specimens were collected from Al-Salam Teaching Hospital and Mosul general hospital, in Mosul city, Iraq.

Inclusion and Exclusion Criteria

The samples were eligible for use in this study if they were "midstream" urine samples obtained from patients with clinical evidence of UTI and appropriate clinical/specimen information and consent was available. Specimens were not collected or handled to ensure sterility, or clinical information or consent was not available, and culture was either polymicrobial or could not be used to recover a pure presumptive isolate. After culture and identification only pure *S. marcescens* isolates were proceeded on to molecular analyses described below. When available, information regarding collections before antibiotic treatment was taken into account.

Ethical Considerations

The Ethics Committee, College of Medicine, University of Nineveh, gave ethical approval. Only specimens for which a consent was available for use in studies was included. Studies on human clinical specimens were conducted according to the study protocol and institutional guidelines. This manuscript does not report any personally identifying information.

Cultivation of Urine Samples

Midstream urine was sent to the lab in a sterile container and inoculated into blood, MacConkey and nutrient agars. Discrete colonies were isolated by streaking and further incubated for 24-h(20) at 37 °C.

Morphological Identification and Biochemical Characterization

Primary culture was followed by subculture of colonies morphologically compatible with *Serratia spp.* to get pure culture. Cell morphology, arrangement and Gram reaction was done from gram stained smears under 100× oil-immersion. Presumptive isolates were identified using catalase, oxidase, urease, indole, methyl red, Voges-Proskauer, citrate and triple sugar iron tests. Final identification of the route was done by the bioMérieux Vitek-2 Compact system with Gram negative organisms identification card as per manufacturer's instructions.

Molecular Identification

DNA Extraction and PCR Screening

The extraction of DNA and conventional PCR assays were done at the College of Science, University of Mosul.

Genomic DNA was isolated by a DNA extraction kit provided by Foregene (China) following the manufacturer's protocol. A PCR Master Mix from Promega (USA) was used according to the manufacturer's instructions to do the PCR amplification. An Applied Biosystems PCR thermal cycler, (Applied Biosystems, USA) was used for amplification. The isolates identified were then screened using conventional PCR for the presence of *flhD*, *pigP*, *shlA*, *phlA*, *aac(6')-Ib-cr*, *blaIMP* and *blaOXA-48*.

Each molecular target was amplified by PCR using the thermal cycling conditions outlined in the original paper referenced in Table 1, as provided by the molecular target. The first denaturation, number of amplification cycles, denaturation, annealing, extension and final extension conditions were then used as described in the published protocol. In the present study, the gene-specific annealing temperatures are presented in Table 1.

The sequences of the primers, the annealing temperature, the expected amplicon sizes and the supporting references are summarized in Table 1. The presence of the targeted DNA sequence was not assumed to indicate gene expression or phenotypic antimicrobial susceptibility; it was used as PCR detection of the DNA sequence.

Table 1. Primers used in the study

Primer name	Gene function	T _m (°C)	Amplicon size (bp)	Primer sequence (5'-3')	References
16S rRNA	Amplification of 16S rRNA	55	1495 bp	F-AGAGTTTGATCMTGGCTCAG R-AAGGAGGTGATCCARCCGCA	[21]
<i>FlhD</i>	Flagellar transcriptional regulator	57	307 bp	F-TGTCGGGATGGGGAATATGG R-CGATAGCTCTTGACGTAATGG	[22]
<i>PigP</i>	Quorum-sensing regulator	53	207 bp	F-GAACATGTTGGCAATGAAAA R- ATGTAACCCAGGAATTGCAC	[23]
<i>ShlA</i>	Hemolysin A	55	217 bp	F-GCGGCGATAACTATCAAAAT R-ATTGCCAGGAGTAGAACCAG	[24]
<i>PhlA</i>	Phospholipase A	55	207 bp	F-GGGGACAACAATCTCAGGA R-ACGCCAACAACATACTGCTTG	[25]
<i>blaIMP</i>	Carbapenemase gene	55	587 bp	GGAATAGAGTGGCTTAAYTC TCGGTTTAAYAAAACAACCACC	[26]
<i>blaOXA-48</i>	Carbapenemase gene	63	438 bp	TTGGTGGCATCGATTATCGGF GAGCACTTCTTTTGTGATGGC	[26]
<i>aac(6)-Ib-cr</i>	Plasmid-mediated quinolone-resistance determinant	55	519 bp	GGCAATAACGGAGGCGCTTCAAA TTCCAGGCATCGGCATCTCATACG	[27]

Agarose-Gel Electrophoresis

PCR products were run on 1.5% agarose gel electrophoresis at 80 V for 45 min and the sizes of the amplicons were determined by comparison to a 100-bp DNA ladder.

DNA Sequencing

Amplified 16S rRNA gene products were analyzed by agarose-gel electrophoresis and then sequenced by Psomagen (USA). The sequences obtained were compared to reference sequences available in NCBI database using BLAST. The sequence results were considered supplementary molecular evidence and added to the Vitek-2 and phenotypic identification for interpretation, and due to the potential for limited discriminatory resolution at 16S rRNA between closely related bacterial taxa, the 16S rRNA sequencing was not used alone for definitive species identification [28].

Results

Eight isolates were identified as *S. marcescens* by culture and biochemical tests, in addition to VITEK-2 Compact system. The colonies on Macconkey agar were light in colour and non lactose fermenting, while the colonies on nutrient agar were circular, convex, smooth and light. 100% of the isolates were oxidase negative and catalase positive. Phenotypic observations were carried out as initial characterization, but not as a reliable basis for definitive species identification.

An approximately 1495-bp PCR product of the 16S rRNA gene was obtained from all eight isolates (Figure 1). Amplicons were purified with PCR clean-up system (Foregene, China), sequenced at Psomagen (USA) and compared with sequences in NCBI database with BLAST. Sequences had an average similarity of ~98% with deposited sequences of *S. marcescens*.

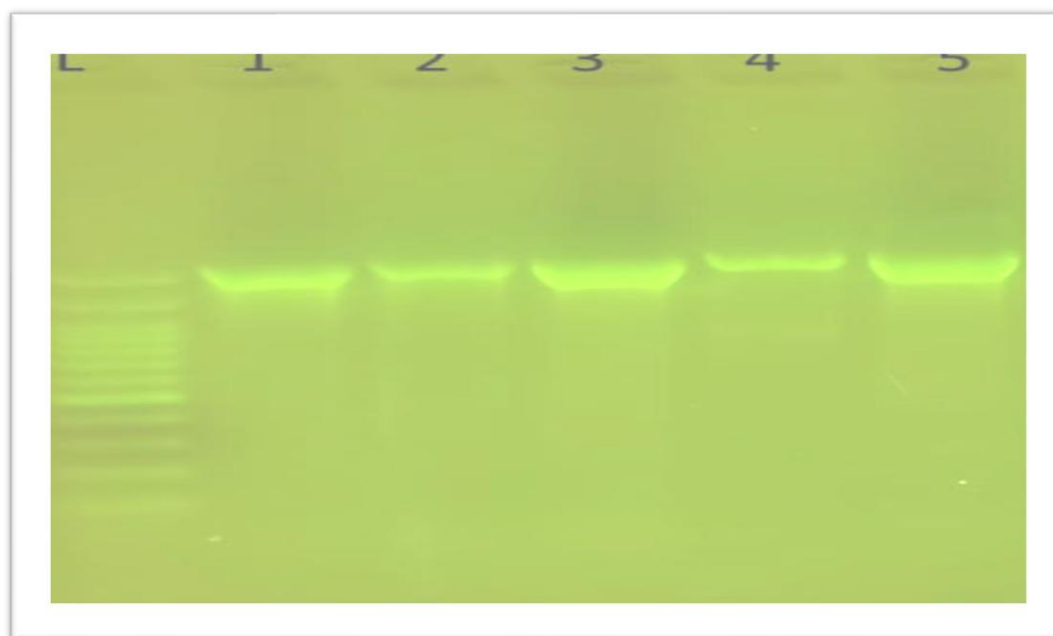


Figure 1. Agarose-gel profile of 16S rRNA PCR products (1495 bp) from *S. marcescens* isolates. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

A total of 150 urine samples were tested, and *S. marcescens* was recovered from eight samples, which is a recovery rate of 5.3%. All of the eight urinary isolates (100%) were positive for the genes *shlA*, *phlA*, *flhD*, and *pigP* when tested by PCR (Figures 2 and 3).

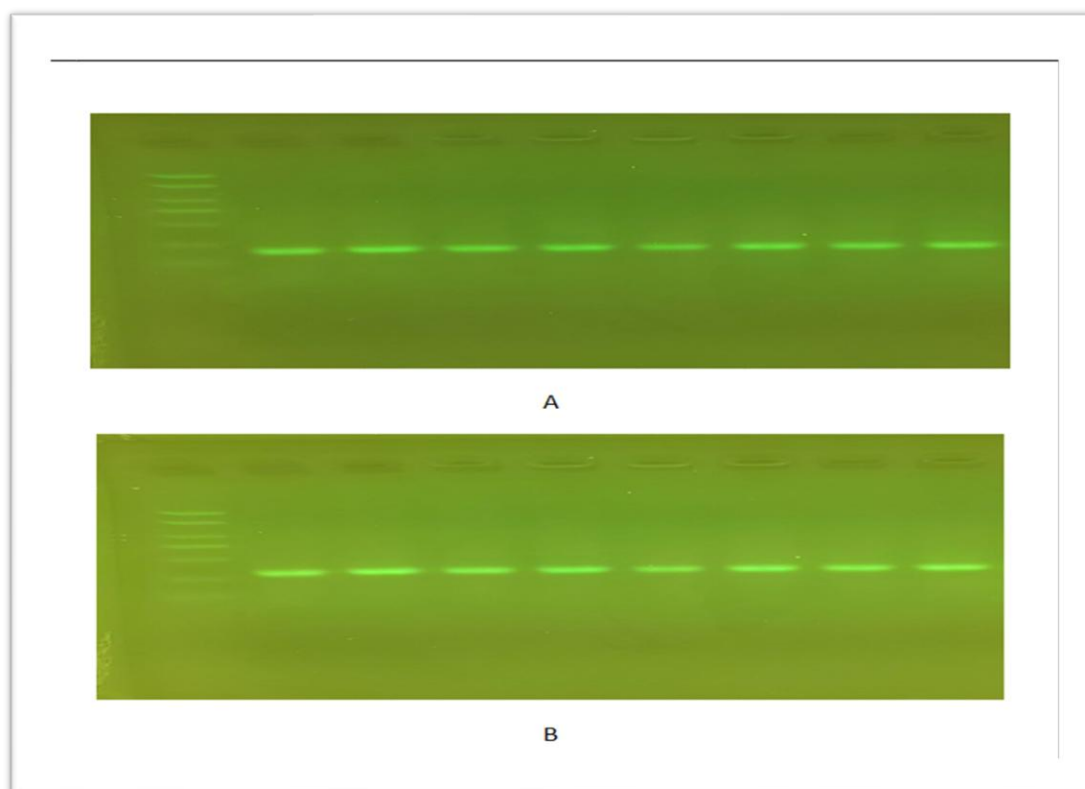


Figure 2. (A) Agarose-gel electrophoresis of the 217-bp ShlA PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

(B) Agarose-gel electrophoresis of the 307-bp *flhD* PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

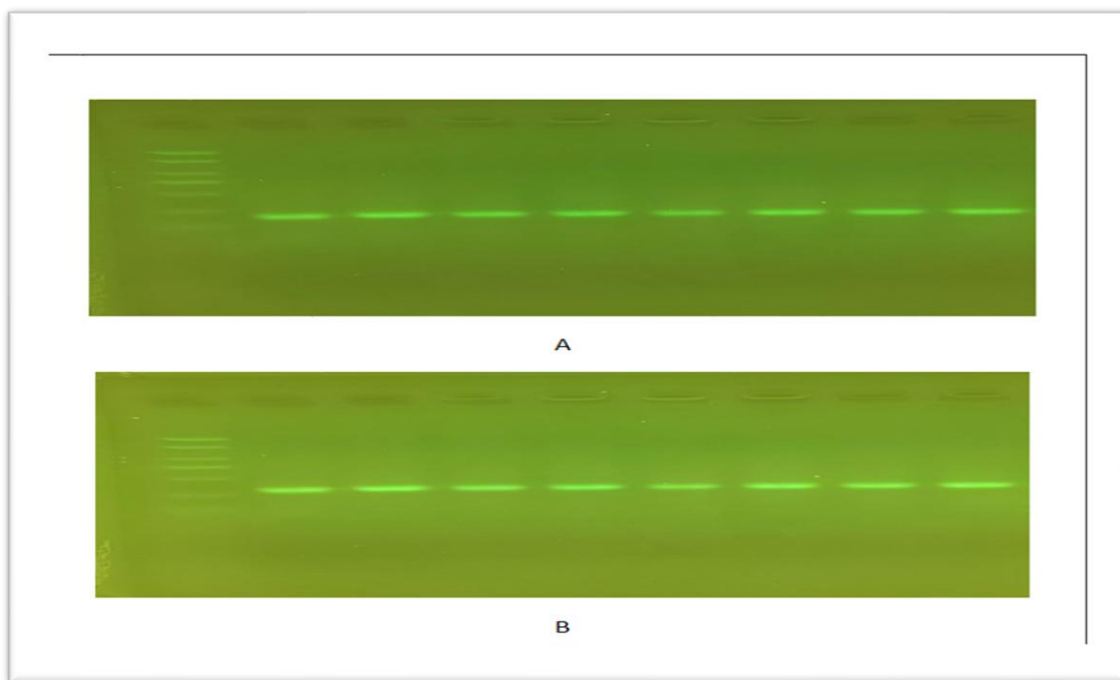


Figure 3. (A) Agarose-gel electrophoresis of the 207-bp PigP PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

(B) Agarose-gel electrophoresis of the 207-bp PhlA PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

All eight isolates (100%) were positive for *aac(6')-Ib-cr* and *blaIMP* was detected in seven of eight (87.5%) while *blaOXA-48* was detected in seven of eight (87.5%) (Figures 4-6). So, seven isolates have been found to have both of the carbapenemase-associated targets tested and all eight have the plasmid-mediated quinolone-resistance determinant included in the PCR panel.

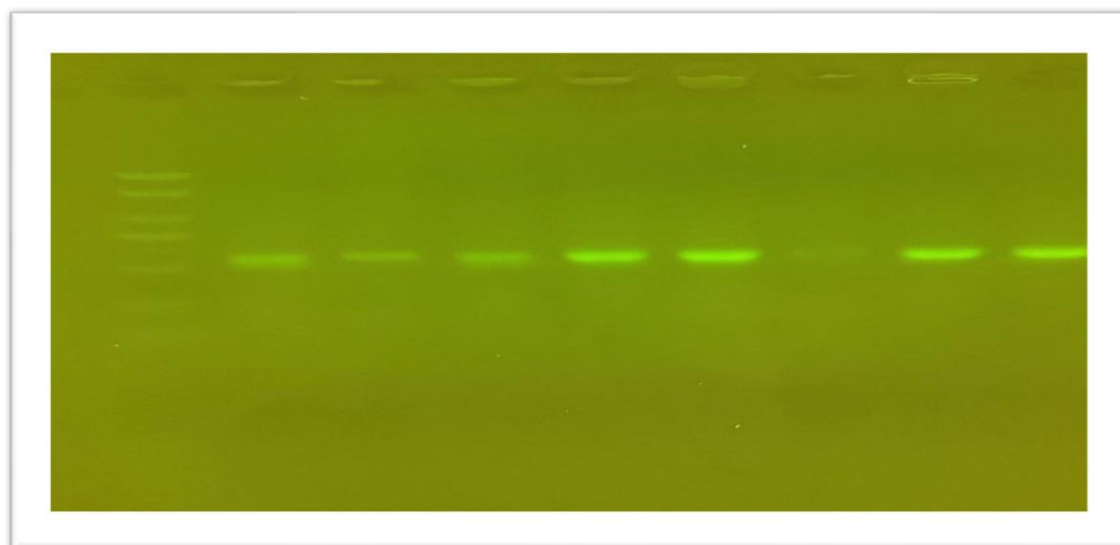


Figure 4. Agarose-gel electrophoresis of the 438-bp *blaOXA-48* PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

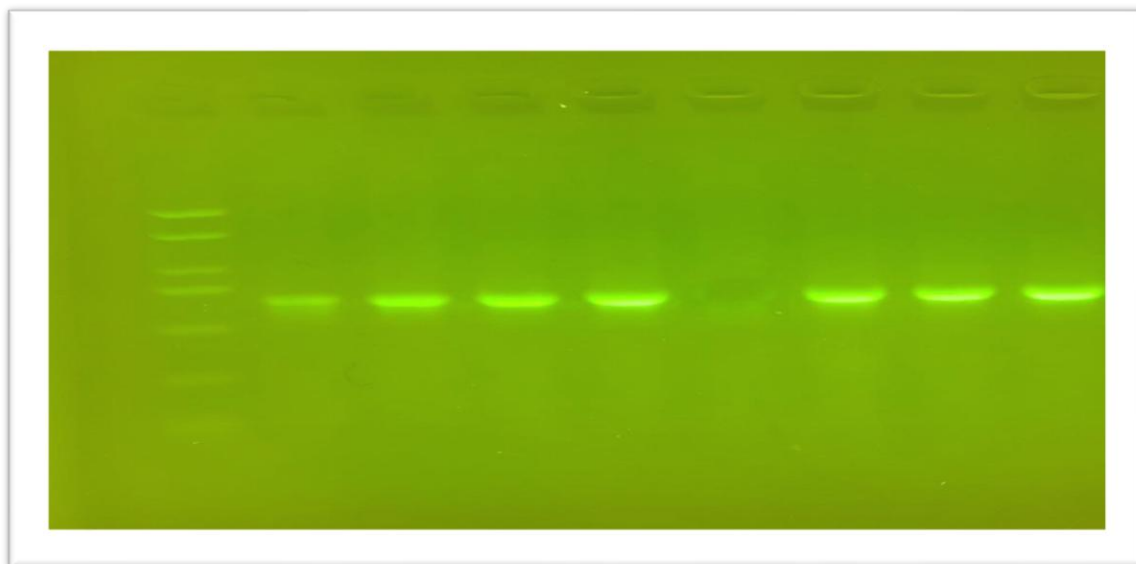


Figure 5. Agarose-gel electrophoresis of the 587-bp *blaIMP* PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

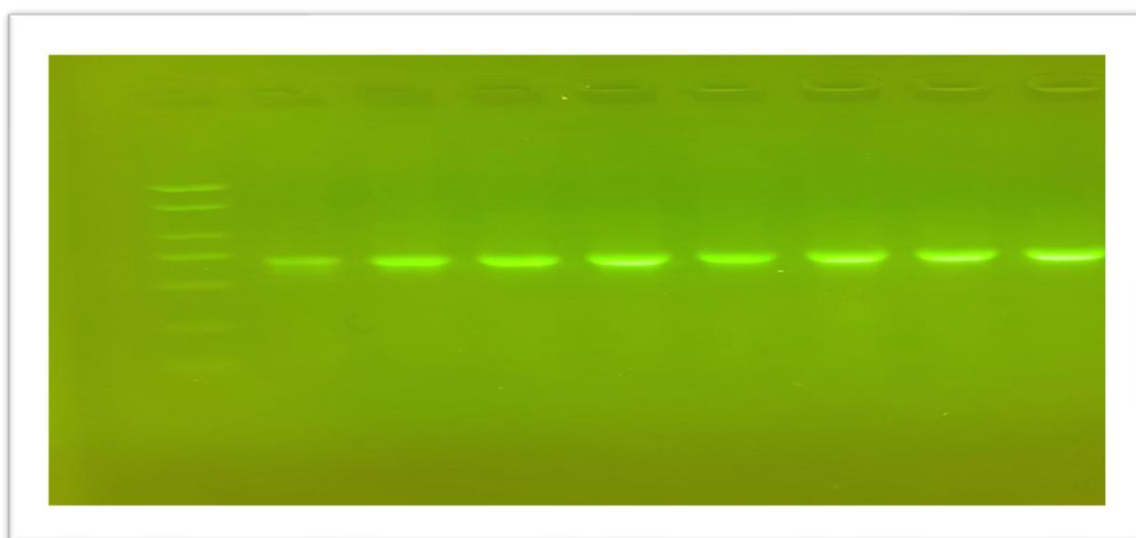


Figure 6. Agarose-gel electrophoresis of the *aac(6')-Ib-cr* PCR product from *S. marcescens*. L: 100-bp DNA marker; lanes 1-8: isolates 1-8.

Discussion

The presence of *S. marcescens* in 8/150 (5.3%) urine samples collected from 2 hospitals during a study period is only representative of the proportion of UTI samples that contained *S. marcescens* samples from the 2 hospitals studied and should not be interpreted as a prevalence proportion for Mosul or for all urine samples with UTI. *S. marcescens* can withstand antimicrobial and host-defense pressures through persistence in healthcare settings and through the ability to form biofilms on abiotic surfaces and urinary devices [30,31].

Khalil et al. [24] found *S. marcescens* in 5 out of 152 (3.28%) clinical samples. Caution should be used in making direct comparisons with the 5.3% recovery seen in the present urine-specimen collection, due to varying study populations, type of specimen collected, sampling

time, inclusion criteria, and identification methods. Ferreira et al. [26] typed 54 *S. marcescens* isolates from various clinical sources, including urine, obtained from the intensive-care and neonatal intensive-care units of a Brazilian tertiary hospital. This distribution is not directly comparable to the distribution of urine specimens that tested positive for *S. marcescens* in the current study and is merely representative of the source composition of an isolate collection. The 16S rRNA sequence similarity of approximately 98% between the eight isolates must be viewed with caution as 16S rRNA may be of limited discriminatory value for closely related members of the *Serratia marcescens* group. More housekeeping genes or genome-wide studies may be necessary for species-level resolution [32]. For this reason, the results of the 16S rRNA typing in this study were regarded as an aid to, rather than an independent basis for, the Vitek-2 and phenotypic identification.

All eight urinary isolates tested are positive for the virulence-associated and regulatory determinants *shlA*, *phlA*, *flhD* and *pigP*, confirming the presence of these determinants. These genes are linked to hemolytic/cytotoxic activity, phospholipase activity, causing membrane damage, regulation of flagellar activities, and regulation of secondary-metabolite production. However, PCR detection is only proof of the presence of the desired genetic sequences, and is not an indication of gene expression or the corresponding virulence phenotypes. The phenotypic expression and biological significance of the studied isolates would require functional assays such as hemolysis, motility and biofilm-formation assays.

In the same manner, the presence of *blaIMP*, *blaOXA-48*, and *aac(6')-Ib-cr* will indicate the presence of antimicrobial resistance determinants but not necessarily the phenotypic antimicrobial resistance. Conventional PCR can be used to detect the presence of a specific sequence, but is not able to measure its expression or contribute to the antimicrobial-susceptibility phenotype. Thus, the present study has a limitation as there is a lack of phenotypic antimicrobial-susceptibility testing.

Molecular screening and standardized phenotypic susceptibility testing should be used together in future studies to evaluate genotype-phenotype correlations and the clinical relevance of these factors.

The pattern of antimicrobial resistance genes of the present isolate collection differs from that of other *S. marcescens* and Enterobacterales populations in that significant clinical and geographic variability in the distribution of antimicrobial resistance genes has been described. Plasmid-mediated quinolone-resistance determinants are present at different prevalence rates in Enterobacterales and their prevalence can vary between bacterial populations and geographical areas [33]. Similarly, in *S. marcescens*, there are various acquired carbapenemase genes isolated from clinical and geographical sites for carbapenem resistance.

Several resistance determinants can occur in the same background strain. Kausar et al. found *blaOXA-48*, *armA* and *aac(6')-Ib-cr* in co-existence in carbapenem-resistant *S. marcescens* from Pakistan [34]. In the current study, co-production of *blaIMP* and *blaOXA-48* was found in 7/8 isolates and *aac(6')-Ib-cr* in 8/8 isolates. The possible explanations are local dissemination of a resistance-gene pattern, related bacterial clones, plasmid mediated dissemination, horizontal transfer of bacterial resistance by mobile genetic elements and antimicrobial selection pressure in healthcare settings. These mechanisms were not explored directly in the current study, and as such, are only hypotheses and not proven mechanisms.

The finding of high prevalence of *blaIMP* and *blaOXA-48* should also be taken with a pinch of salt as compared with the international resistance pattern in this small collection of isolates. Variations in the distribution of carbapenemases between geographical regions may be explained by the variations in circulating plasmids and bacterial clones, antimicrobial use, patient populations, infection-control environments, and local healthcare epidemiology. It is noteworthy that the co-detection seen here, along with reports like that of Kausar et al. [34] suggests that the distribution of carbapenemase determinants may not be the same across the globe. The present data

cannot establish The current information does not confirm the *blaIMP* and *blaOXA-48* as the main carbapenemase genes in Mosul or Iraq. A limited number of *S. marcescens* isolates were examined and only a limited subset of the carbapenemase genes was included in the PCR panel. Also, plasmid characterization, whole genome sequencing, and phenotypic antimicrobial-susceptibility testing were not done. The results are therefore not intended to be used as population prevalence estimates, but as frequencies of the investigated molecular targets in this local isolate collection. To define whether the pattern is due to local antimicrobial selection and horizontal gene transfer, to a pattern of clonal dissemination or sampling variation, larger surveillance studies with more comprehensive resistance-gene panels, standardized phenotypic susceptibility testing, plasmid typing and high-resolution genomic typing are needed.

Study Limitations

There are a number of limitations to this study. First, only eight *S. marcescens* isolates were recovered and molecularly characterized, so the frequencies of the observed genes should be interpreted as descriptive results from this collection of isolates, and should not be extrapolated to the general population of *S. marcescens*. Second, phenotypic antimicrobial susceptibility testing was not carried out; thus, detection of *blaIMP*, *blaOXA-48* and *aac(6')-Ib-cr* does not imply the presence of the associated antimicrobial-resistance phenotypes. Third, functional virulence assays (e.g., biofilm formation, motility, hemolysis, and cytotoxicity) were not conducted, and detection of *shlA*, *phlA*, *flhD*, and *pigP* should not be taken as an indicator of gene expression or functional virulence. Fourth, the typical 16S rRNA sequence similarity (98%) does not offer sufficient species level resolution between closely related *Serratia spp.* Further determination of taxonomic information through housekeeping-gene analysis or whole genome sequencing would give more taxonomic resolution. Finally, molecular typing is not sufficiently discriminatory to allow for assessment of clonal relatedness or possible transmission relationships between isolates or to consider the plasmid context. These data should be confirmed in future studies using larger collections of isolates with standardized phenotypic antimicrobial susceptibility testing, functional virulence testing, expression profiling, and higher resolution molecular typing to assess their phenotypic, clinical, and epidemiological impact.

Conclusions

Eight *S. marcescens* were isolated from 150 samples of urine from UTI patients in two hospitals in Mosul during the study period (5.3%). All eight isolates carried *shlA*, *flhD*, *pigP*, *phlA*, and *aac(6')-Ib-cr*, while *blaIMP* and *blaOXA-48* were each detected in seven isolates (87.5%). These results reflect the co-occurrence of some of the virulence-associated/regulatory and antimicrobial-resistance determinants within a small local collection of urinary isolates. The results are not intended to be interpreted as prevalence rates of the population or as evidence for gene expression or evidence of phenotypic antimicrobial resistance. Higher resolution molecular typing, standardized antimicrobial-susceptibility testing, appropriate molecular controls, and sequence accession data should be included in larger and prospectively designed studies to assess the clinical and epidemiological importance of these observations.

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