Original Article

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Microbiology Section

Prevalence of Quorum Sensing Regulator/ Autoinducer Synthase (*abaR/abaL*) genes among the Multi-drug resistant strains of *Acinetobacter baumannii*: A Cross-sectional Study

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ABSTRACT

Introduction: *Acinetobacter baumannii (A. baumannii)* is a significant Multi Drug Resistant (MDR) opportunistic pathogen, documented as a priority pathogen due to its persistence in hospital environments and its ability to cause nosocomial infections.

Aim: To investigate the prevalence of Quorum Sensing (QS) mediating *abaR/abaL* genetic determinants in MDR strains of *A. baumannii* isolated from wound infections.

Materials and Methods: This cross-sectional study was conducted in the department of Microbiology, at Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Institute (SIMATS), Chennai, Tamil Nadu, India, from January to April 2024. A total of 20 clinical isolates of *A. baumannii* were obtained from patients with deep wound infections. The isolates were identified using the VITEK 2 system, and their antibiotic susceptibility profiles were evaluated. Biofilm formation was assessed using a microtiter plate assay, while *abaR/abaL* genes were detected via Polymerase Chain Reaction (PCR). Mutations were identified through Sanger sequencing. Fisher's exact test was performed in Statistical Package for the Social Sciences (SPSS) with $p<0.05$ considered statistically significant.

Results: A total of 20 isolates were identified as *A. baumannii* using the VITEK 2 system followed by their antibiotic susceptibility profiles. The findings revealed that antibiotic susceptibility testing indicated high resistance rates to common antibiotics, with complete susceptibility to tigecycline and colistin. Sixty percent of the isolates demonstrated strong biofilm-forming capabilities, and 100% of the isolates acquired the *abaL* gene, while 90% possessed the *abaR* gene. Sequence analysis showed significant genetic variability in both genes, with several mutations potentially linked to enhanced resistance and virulence.

Conclusion: This study highlights the relationship between QS systems, biofilm formation, and antibiotic resistance, emphasising the importance of targeting the QS pathways as a novel therapeutic approach for controlling MDR *A. baumannii* infections. Further investigations into the molecular mechanisms underlying QS regulation are essential for developing effective antimicrobial strategies against this formidable pathogen.

Keywords: Antibiotic resistance, Health, Opportunistic pathogen, Sanger sequencing, Wound infection

INTRODUCTION

Acinetobacter baumannii is a Gram-negative, highly successful opportunistic pathogen that poses a significant threat to public health, predominantly in healthcare settings [1]. This bacterium is notorious for its capability to endure various environments and rapidly acquire resistance to multiple antibiotics, leading to the emergence of MDR strains [2]. Over recent decades, the prevalence of *A. baumannii* has increased significantly, presenting substantial challenges in healthcare systems worldwide [3]. The exceptional adaptability of *A. baumannii* to adverse conditions is attributed to a combination of intrinsic resistance mechanisms and vital genetic factors that aid its persistence under antibiotic pressure [4]. *A. baumannii* is a particularly challenging pathogen in clinical settings, especially in wound infections. Its implications are severe due to its ability to cause persistent and hard-to-treat infections, often in immunocompromised patients or those with extensive wounds, such as burns, surgical sites, or traumatic injuries [5]. *A. baumannii* can lead to chronic wound infections, significantly delaying the healing process. The bacterium's ability to form biofilms on wound surfaces shields it from the host immune response and antimicrobial treatments, complicating eradication and management strategies in healthcare settings [6].

One of the crucial factors influencing the pathogenicity and resistance of *A. baumannii* is its QS system, which regulates gene expression based on bacterial population density [7]. The QS system plays a key role in MDR *A. baumannii*, enabling cell-to-cell communication through the production and detection of small signaling molecules [8]. Specifically, by using signaling molecules like Acyl-Homoserine Lactones (AHLs), bacteria coordinate group behaviors that enhance persistence and resilience, particularly in unfavorable environments [9]. This system has been associated with increased biofilm formation, which serves as a protective barrier against antibiotics and the host immune system [10].

Biofilms represent significant barriers to antibiotic resistance, as they form a physical obstruction that blocks drug infiltration and protects bacterial cells from host immune responses [11]. The QS system also regulates numerous virulence factors, including motility, the secretion of toxins, and the production of enzymes, all of which are essential for survival in hostile environments [12]. Furthermore, specific genetic determinants, including the QS-associated *abaR/abaL* genes, play an important role in antibiotic resistance and biofilm formation. These genes contribute to resistance mechanisms such as efflux pumps, enzymatic degradation of antibiotics, and modifications of target sites, further complicating the treatment of *A. baumannii* infections [13,14].

Remarkably, the aba genes and QS systems, such as *abal/abaR*, and abaM, are frequently found in MDR strains of *A. baumannii*,

linking them to biofilm formation and resistance mechanisms [15]. Comparative genomic studies have shown significant differences in the frequency of QS and antibiotic resistance genes between MDR and non-MDR strains, with MDR strains harboring a higher abundance of *abaR* determinants [16]. This suggests a strong correlation between MDR and the presence of QS and antimicrobial resistance.

The increasing occurrence of QS systems and *abaR* genetic determinants among MDR *A. baumannii* strains highlights the need for novel therapeutic strategies to target QS pathways and biofilm-associated mechanisms. Understanding the role of these systems in pathogenesis and resistance is crucial for developing effective interventions against MDR *A. baumannii* infections. In this study, we investigate the frequency of QS-mediating *abaR*, and abaL genetic determinants among MDR strains of *A. baumannii*.

MATERIALS AND METHODS

This cross-sectional study was conducted in the department of Microbiology, at Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Institute (SIMATS), Chennai, Tamil Nadu, India, between January and April 2024, with approval from the Institutional Ethics Committee (IEC) (Ref No: SRB/SDC/UG-2222/24/MICRO/014).

Characterisation of *A. baumannii* from Clinical Specimens: A total of 20 clinical isolates of *A. baumannii* were collected from patients with deep wound infections, derived from 81 wound samples, including wound tissue, pus, and swabs.. The collected specimens were cultured on blood agar and MacConkey agar plates, and suspected colonies were identified based on morphology and confirmed using the VITEK 2 system.

Inclusion and Exclusion criteria: The inclusion criteria consisted of patients with clinically diagnosed deep wound infections who had not received antibiotic therapy in the past 48 hours, while the exclusion criteria involved patients with superficial wound infections and duplicate samples from the same patient.

Detection of Biofilm Formation

The ability of *A. baumannii* isolates to form biofilms was assessed using a crystal violet staining method, modified from Kannan KP and Smiline Girija AS [17]. Briefly, overnight cultures of each strain were diluted to an optical density at 600 nm (OD600) of 0.1. Subsequently, 180 µL of Brain Heart Infusion (BHI) broth (Himedia, Mumbai, India) and 20 µL of the bacterial suspension were added to 96-well polystyrene microtiter plates (Thermo Fisher Scientific, USA) in triplicate. Following a 24-hour incubation period at 37°C, the plates were gently washed three times with phosphate-buffered saline (PBS) and stained with 200 µL of 0.1% crystal violet (Himedia, Mumbai, India) for 20 minutes. The plates were then washed three more times with PBS and solubilized with 200 µL of 95% ethanol, followed by gentle shaking at room temperature for 20 minutes. The absorbance was measured at 570 nm (OD570).

DNA Extraction

A. baumannii isolates from skin and wound infection samples contained 20 MDR strains. These strains were selected for further molecular study based on CLSI breakpoint criteria and the work of Priyadharshini [18]. According to the Qiagen DNA extraction kit protocol, genomic DNA was extracted and stored at -20°C for subsequent molecular assays.

Molecular Detection of *abr* by PCR

The PCR reaction mixture was prepared using a 2x master mix from Takara, 5.6 µL of double-distilled water, and specific primers for the *abaR*/L gene obtained from Eurofins Genomic India Pvt. Ltd., Bangalore [19]. The PCR conditions are provided in [Table/Fig-1], and the amplification was performed in an Eppendorf thermocycler (Germany) for 35 cycles. The resulting amplicon was analyzed using a 100 bp DNA ladder in 1% agarose gel electrophoresis (1 µL/10 mL of agarose gel) and visualised using a gel documentation system.

Gene	Sequence	Annealing	Amplicon size
abaR F *abaR* R	TCCTCGGGTCCCAATA TAAATCTACCGCATCAA	52°C	310bp
abaL F *abaL* R	AAAGTTACCGCTACAGGG CACGATGGGCACGAAA	58°C	435bp

[Table/Fig-1]: Primer sequences and PCR specifications for *abaR/abaL* gene types used in the study.
F-Forward primers; R- Reverse primers

Sequencing and Genomic Analysis of *abaR* Genes

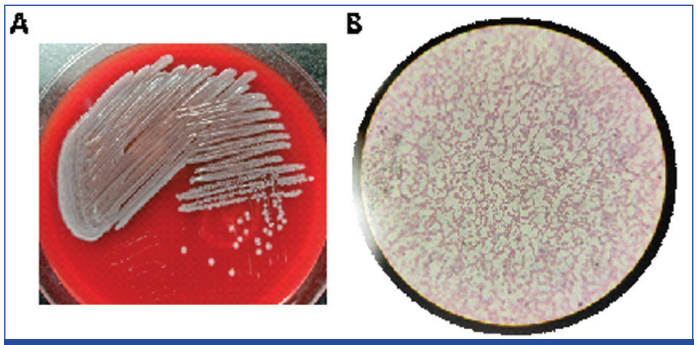
The PCR amplicons were sequenced using the Big-Dye Terminator v3.1 Cycle Sequencing Kit (Applied Biosystems, USA) and analyzed on a 3730XL genetic analyzer. The resulting sequences were processed using Bio-Edit Sequence Alignment Editor version 7.2.5. Nucleotide similarity and mutation analysis were conducted using the BLAST (Basic Local Alignment Search Tool) platform, and multiple sequence alignments were carried out using ClustalW software version 1.83.

STATISTICAL ANALYSIS

The association between the presence of *abaR/abaL* genes and strong biofilm formation was evaluated using Fisher's exact test. A p-value<0.05 was considered statistically significant, indicating a strong association between the presence of QS genes and biofilm formation. Statistical analysis was performed using SPSS software version 25.0.

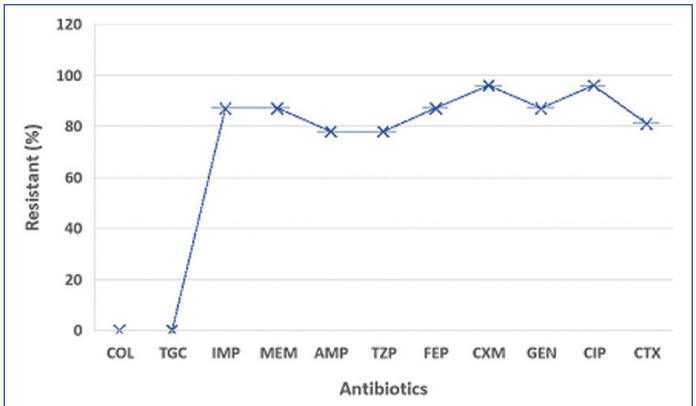
RESULTS

Isolation and Identification of *A. baumannii*: A total of 20 isolates (24.69%) were identified as *A. baumannii* using the VITEK 2 system, followed by the determination of their antibiotic susceptibility profiles [Table/Fig-2]. These 20 isolates were recovered from skin and wound infections of patients irrespective of gender. *A. baumannii* strains were found to be more prevalent in skin and wound infections. The antimicrobial susceptibility of *A. baumannii* (N=20) was evaluated against 11 antibiotics [Table/Fig-3]. The isolates demonstrated complete sensitivity to tigecycline and colistin (100%). Resistance was observed in 81% of the isolates against cefotaxime, 78% against both ampicillin-sulbactam and piperacillin-tazobactam, and 87% against imipenem, meropenem, cefepime, and gentamicin. Additionally, 96% of the isolates showed resistance to cefuroxime and tetracycline.

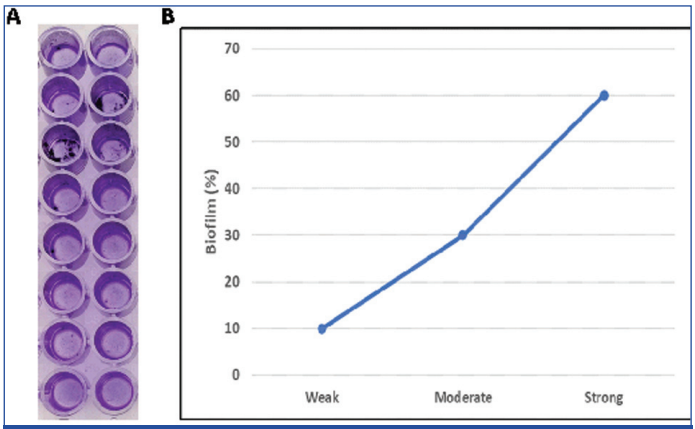


[Table/Fig-2]: Isolation and identification of *A. baumannii* from the patients with wound infections. A. Typical *A. baumannii* colonies on the blood agar plate. B. Gram staining presenting the typical gram-negative coccobacilli forms of *A. baumannii*.

Strength of Biofilm-formation by *A. baumannii* Isolates: The crystal violet biofilm assay successfully classified the 20 *A. baumannii* isolates into distinct categories based on their biofilm-forming capabilities. Specifically, two isolates (10%) were identified as weak biofilm formers, six isolates (30%) exhibited moderate biofilm formation, and 12 isolates (60%) demonstrated strong biofilm-forming abilities, as depicted in [Table/Fig-4].

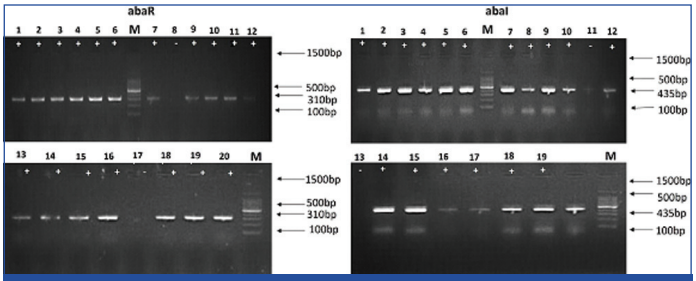


[Table/Fig-3]: Bar chart showing the susceptibility and resistance patterns of *A. baumannii* isolates to various antibiotic classes as interpreted using the CLSI guidelines, 2023.



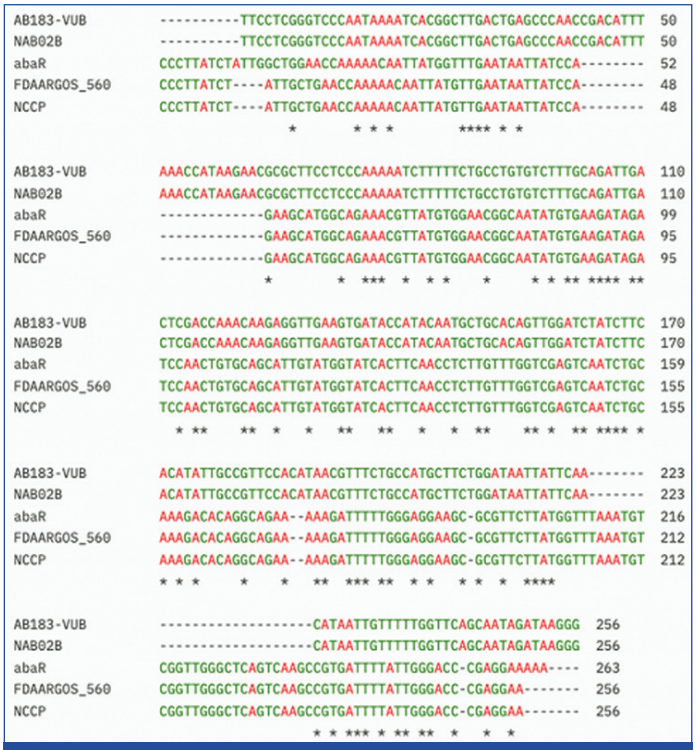
[Table/Fig-4]: A. Biofilm formation ability of *A. baumannii* isolates as determined by the crystal violet assay in a microtiter plate; b) Graph representing the grading of biofilm formation by the isolates.

Frequency of *abaR* and *abaL* genes among MDR *A. baumannii*: The *abaR* and *abaL* genes were identified in all (100%) isolates, confirming the presence of *A. baumannii*. Among the 20 isolates examined, the detection rates for *abaL* and *abaR* genes were 100% and 90%, respectively [Table/Fig-5].

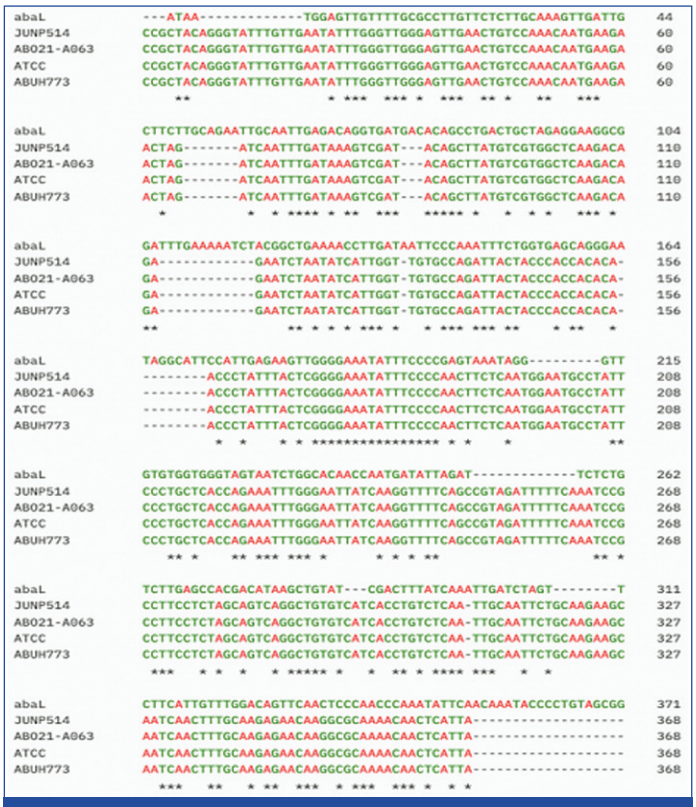


[Table/Fig-5]: Agarose gel electrophoretogram showing amplification of *abaR* (310 bp) and *abaL* (435 bp) genes along with standard DNA ladder (Lane M = 100 bp DNA marker).

Sanger sequencing analysis: Sequence analysis of the *abaR* and *abaL* genes in *A. baumannii* isolates revealed significant genetic diversity. Conserved regions, mainly within the N-terminal domains, were identified. However, various mutations, including insertions, deletions, and substitutions, were observed in specific regions. These variations may have important implications for the functional alterations of the encoded proteins, potentially inducing virulence or antibiotic resistance. [Table/Fig-6] illustrates that mutations in the *abaR* gene within the region positions 170-223 may alter protein structure or function, potentially impacting its role in antibiotic resistance. Similarly, mutations in the *abaL* gene between positions 164 and 215 [Table/Fig-7] might affect the protein's ability to regulate bacterial virulence. Additionally, the noted sequence mutations in the C-terminal region of *abaL* suggest evolutionary deviation among the isolates, potentially supporting their diverse phenotypic characteristics.



[Table/Fig-6]: A multiple-sequence alignment of *abaR* gene in *A. baumannii*. The process was performed with 4 reference strains of *A. baumannii* viz., AB183-VUB, FDAARGOS_560, NAB02B, NCCP 16006. The red colour indicates - mismatches; Green - deletions; Gray dashes (- - -) - deletions; Gaps (blank spaces) - one square lacks corresponding nucleotides found in other sequences; Astericks (*) - marks the position where all the four sequences are identical (conserved nucleotides).



[Table/Fig-7]: A multiple-sequence alignment of *abaL* gene in *A. baumannii*. The process was performed with 4 reference strains of *A. baumannii* viz., JUNP514, AB021-A063, ATCC BAA-1790, ABUH773. The red colour indicates - mismatches; Green - deletions; Gray dashes (- - -) - deletions; Gaps (blank spaces) - one square lacks corresponding nucleotides found in other sequences; Astericks (*) - marks the position where all the four sequences are identical (conserved nucleotides).

DISCUSSION

A. baumannii has the ability to colonize and infect both surgical and traumatic wounds, making it an alarming pathogen in healthcare settings. In postsurgical infections, *A. baumannii* can lead to delayed wound healing, prolonged hospital stays, and increased healthcare expenditures due to its persistence and resistance to treatment

strategies. In cases of traumatic injuries, *A. baumannii* has been recognized as a significant pathogen of deep tissue infections. These conditions are often resistant to multiple antibiotics, further complicating their treatment options [20]. The present study aimed to explore the interrelationship between antibiotic resistance, biofilm formation, and the associated genetic factors in clinical isolates of *A. baumannii*.

It is important to understand the mechanisms that control the expression of virulence factors, which is crucial for the development of novel antimicrobial tactics against antibiotic-resistant pathogens like *A. baumannii* [21]. Among these mechanisms, quorum sensing (QS) has emerged as a key target for the regulation of virulence features [22]. The QS system, specifically the *abaL/abaR* system, is known to influence biofilm formation and antibiotic resistance in *A. baumannii*. This study focused on the prevalence of the *abaL/abaR* QS system in the virulence of clinical multidrug-resistant (MDR) *A. baumannii* isolates.

The frequency of *A. baumannii* in wound infections is well-studied. In a military medical center in the United States, the occurrence rate of *A. baumannii* increased from 4% to 55% over eight years, with wound isolates accounting for 24% of the total cases [23]. In our study, *A. baumannii* accounted for 24.69% of wound infections (N=20), which aligns with previous reports and suggests that the prevalence of this pathogen remains high in various regions.

The VITEK 2 system, used in our study to identify and characterize *A. baumannii* isolates, offers numerous clinical advantages for the routine testing of gram-negative rods from clinical samples. Its rapid identification within three hours, high level of accuracy, and wide-ranging database make it a valuable tool in the field of clinical microbiology. Early and precise identification of pathogens, particularly MDR strains, is essential for timely and appropriate antimicrobial therapy, which can reduce both patient mortality and healthcare costs [24,25].

The rising concern of antibiotic resistance in *A. baumannii* is highlighted by our findings, which identified all isolates as multidrug-resistant (MDR) and carbapenem-resistant. These isolates exhibited resistance to a broad spectrum of antibiotics, including aminoglycosides, carbapenems, cephalosporins, fluoroquinolones, penicillins, and β-lactams, with the exception of colistin and tigecycline. Such findings are consistent with recent studies reporting similarly high resistance rates of *A. baumannii* [26].

Biofilm formation is a key virulence factor in *A. baumannii* and contributes to its persistence in both infection sites and medical devices (e.g., catheters and wound dressings). Biofilms act as a shield for bacteria, protecting them from immune responses and antimicrobial treatments, making them particularly harmful [27]. Our study found a strong correlation between the varying ranges of biofilm formation (low, moderate, and high) and antibiotic resistance in *A. baumannii* strains, consistent with previous findings [28].

Recent research has investigated the correlation between biofilm development and the QS system in *A. baumannii*, indicating that the QS system regulates not only biofilm formation but also gene expression and extracellular polysaccharide production [29]. In our study, we observed a high prevalence of *abaR* and *abaL* genes among the MDR strains of *A. baumannii*. These genes are crucial components of the QS system, regulating various factors such as biofilm formation, motility, and virulence, which are essential for the pathogen's survival and persistence in hostile environments, including clinical settings [30]. This prevalence may be attributed to the increasing resistance and virulence of *A. baumannii* strains.

The multiple-sequence alignment of the *abaR* and *abaL* genes among eight *A. baumannii* reference strains (AB183-VUB, FDAARGOS_560, NAB02B, NCCP 16006, JUNP514, AB021-A063, ATCC BAA-1790, and ABUH773) revealed significant genetic variability and conserved mutated regions. These variations could influence the regulatory role of *abaR* and *abaL* in QS and pathogenicity.

This study provides important insights into the phenotypic and genetic characteristics of *A. baumannii*; however, it has a limited number of samples, which is insufficient for comprehensive statistical analysis on the prevalence rate and frequency of the *abaL* and *abaR* genes. The study may be restricted to a specific geographic location, which limits its genetic diversity. Future studies should incorporate larger and more geographically diverse samples, along with microbiological, clinical, and environmental data, to better understand the drivers of MDR and QS in *A. baumannii*.

Limitation(s)

This study presents several limitations despite its noteworthy findings. Firstly, the limited sample size (n=20) constrains the generalizability of our results; a more extensive cohort would provide deeper insights into the prevalence and molecular diversity of the *abaR/abaL* genes. The investigation was conducted in a single geographic region, potentially overlooking genetic variations among *A. baumannii* strains from broader locales. Although this study implemented phenotypic and genetic assays, the lack of functional validation for the identified mutations in the *abaR/abaL* genes leaves their specific roles in biofilm formation and resistance mechanisms ambiguous. Lastly, while a significant association between QS genes and biofilm formation was noted, further research incorporating transcriptomic and proteomic analyses is essential to clarify the underlying regulatory mechanisms in the pathogenesis of multidrug-resistant *A. baumannii*.

CONCLUSION(S)

In this study, the QS system and *abaR/abaL* were widely distributed among the clinical isolates of *A. baumannii*, associating with drug resistance, biofilm formation, and virulence. Moreover, this study highlights the prevalence of *abaR/abaL* in the QS signaling system, which can help manage infections caused by *A. baumannii*. However, elucidation of the QS network for the regulation of antimicrobial resistance and virulence of *A. baumannii* at the molecular and cellular levels is necessary to apply this new strategy.

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