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Indian Journal of Microbiology Research

Journal homepage: www.ijmronline.org

Original Research Article

Phenotypic detection of virulence attributes in MDR *Acinetobacter* from a tertiary care hospital, South India

Monisha Basavaraju[1], Rashmi P Mahale[1]*, M Raghavendra Rao[1], Suchitra Shenoy[2]

[1]Dept. of Microbiology, JSS Medical College & Hospital, Mysuru, Karnataka, India
[2]Dept. of Microbiology, Kasturba Medical College & Hospital, Manipal, Karnataka, India

Abstract

Background: The nosocomial pathogen *Acinetobacter baumannii* is found all over the world and has a number of virulence characteristics that enable them to render resistance to antibiotics, making them multidrug resistant.
Objectives: To detect the virulence attributes of MDR *Acinetobacter baumannii* isolated from a tertiary care hospital in South India.
Materials and Methods: This prospective study, conducted from August 2023 to October 2024, involved 55 clinical and 55 environmental multidrug-resistant (MDR) *Acinetobacter baumannii* isolates. The isolates were evaluated for virulence factors using phenotypic assays, which included siderophore production assessed on Chrome Azurol S (CAS) agar, hemolytic activity on blood agar, proteolytic activity on milk agar, and biofilm formation using the tissue culture plate method.
Results: All clinical and environmental MDR *A. baumannii* isolates exhibited intermediate susceptibility to colistin. Tigecycline sensitivity was observed in 78% of the clinical isolates and 52.27% of the environmental isolates. Of the 55 clinical isolates of MDR *A. baumannii*, 34 (61.81%) showed biofilm production, 20 (36.36%) showed haemolytic activity, 29(52.72%) showed proteolytic activity and 30 (54.54%) showed siderophore production. Similarly, among the 55 Environmental isolates, 24 (43.63%) showed biofilm production, 15 (27.27%) showed haemolytic activity 31(56.36%) showed proteolytic activity and 19 (34.54%) showed siderophore production.
Conclusion: This study shows that the clinical MDR *A. baumannii* strains were more virulent than the environmental strains, thereby highlights its evolving virulence and resistance. This study also stresses the importance of continued surveillance, infection control practices and responsible antibiotic use in clinical setting.

Keywords: Antimicrobial resistance, *Acinetobacter*, Biofilm, Multidrug Resistance, Virulence.

Received: 07-04-2025; **Accepted:** 07-06-2025; **Available Online**: 20-09-2025

1. Introduction

The genus *Acinetobacter* comprises Gram-negative coccobacilli that are strictly aerobic, non-motile, non-fermentative, catalase-positive, and oxidase-negative.[1] It is frequently associated with healthcare-associated infections (HAIs) such as bloodstream infections, ventilator-associated pneumonia (VAP), wound infections, and urinary tract infections, particularly in intensive care units (ICUs).[2,3]

The emergence of multidrug-resistant (MDR) *A. baumannii* strains complicates treatment strategies and is linked to increased rates of morbidity and mortality. Thus, forcing clinicians to rely on last-resort antibiotic such as colistin (polymyxin E). However, resistance to colistin has also been reported, further increasing the challenge of treating infections caused by MDR *A. baumannii*.[4,5]

The pathogenicity of *A. baumannii* is linked to a variety of virulence factors. The biofilm consists of microbial community attached to the surface by an extracellular matrix[6,7] that involves complex regulatory networks which

*Corresponding author: Rashmi P Mahale
Email: basavarajumonisha@gmail.com

https://doi.org/10.18231/j.ijmr.89406.1758357916

coordinate the gene expression like bap, ompA, epsA, csuE and bfmS.[7,8] Pilis production is also required for biofilm formation.[9] Therefore, biofilm enhances the colonization which provides bacteria with the ability to survive and acts against the antimicrobial agents thus complicating the treatment options. The primary genes associated with haemolytic activity in *Acinetobacter baumannii* are the phospholipase C (PLC) genes, specifically "plc1" and "plc2", which encode enzymes that can lyse red blood cells by breaking down the phospholipid membrane component phosphatidylcholine.[10,11] The gene associated with proteolytic activity is "ctp" (carboxy-terminal processing protease), which plays a key important role in maintaining membrane integrity, adapting to environmental stress, and controlling virulence by regulating protein processing at the cell membrane.[12] Siderophore production, is mainly through the biosynthesis of acinetobactin, helps the bacterium in iron uptake using a set of genes (*basA-J*), efflux (*barAB*) and uptake (*bauA-E*), which was also identified as a virulence determinant in *A. baumannii*.[13,14]

Thus, this study aims to assess the phenotypic characterization of virulence factors in MDR *A. baumannii* isolates from both clinical and environmental sources which is essential to gain insights into the organism's pathogenic potential and its ability to persist and spread within healthcare settings.

2. Material and Methods

This prospective study was conducted in a tertiary care hospital, South India. Sample collection took place over a 15-month period, from August 2023 to October 2024. Clinical specimens—including sputum, endotracheal aspirates, pus, blood, urine, and other body fluids—were processed following standard protocols. Multidrug-resistant *A. baumannii* isolates were identified using the VITEK-2 system (BioMérieux, India), in accordance with the manufacturer's guidelines[15] (Multidrug resistance – resistance of an organism to at least one antimicrobial drug in three or more antimicrobial categories).

The study included 55 clinical MDR *A. baumannii* isolates along with 55 corresponding environmental isolates were isolated.

Patient's surroundings from whom MDR *Acinetobacter* were isolated clinically, were screened for the presence of MDR *Acinetobacter spp*. From each patient 5 environmental samples (Wall, trolley, floor, bathroom, bed and bed rails) were obtained. MDR *Acinetobacter* isolated from all or any one patient environmental site were considered.

Informed consent was obtained from the study participants and the study was approved by the Institutional Ethics Committee with the corresponding approval number JSS|MC|PG|0040|2022-2023|Dated 05-04-2023.

2.1. Biofilm production

Biofilm detection using the tissue culture plate method was carried out according to the protocol described by Christensen *et al.*[16] The test organism was adjusted to a 0.5 McFarland standard and subsequently diluted 1:100 in fresh tryptic soy broth. A volume of 200 µL of this diluted suspension was added to sterile, flat-bottomed 96-well polystyrene microtiter plates and incubated at 37°C for 24 hours. After incubation, each well was washed three times with phosphate-buffered saline (PBS, pH 7.2) and gently tapped to remove non-adherent bacteria and residual contents. The plates were then air-dried in an inverted position at room temperature. Biofilms were fixed using 2% sodium acetate and stained with 0.1% crystal violet (CV) solution for 10–15 minutes. Excess stain was removed by washing with PBS three times. To quantify biofilm formation, the bound CV was solubilized using 30% acetic acid for 30 minutes, and the optical density (OD) was measured at 570 nm using an ELISA reader.

The interpretation of biofilm production was classified as follows:

Optical Density	Biofilm Production
>0.68	Strong biofilm producer
0.35-0.68	Moderate biofilm producer
0.17-0.34	Weak biofilm producer
<0.17	Non biofilm producer

Note: Weak biofilm producers were also considered as nonbiofilm producer.[17] (**Figure 1**)

Test organism adjusted to 0.5 McFarland standard.

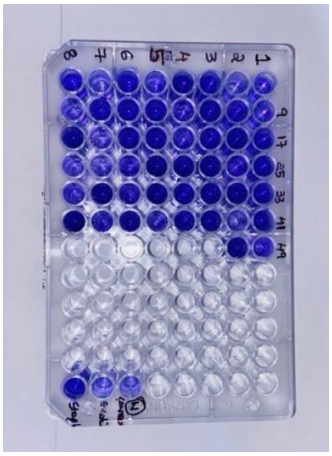

Figure 1: Biofilm production assessed using tissue culture plate method

Test organism was inoculated on 5% sheep blood agar and observed for haemolysis pattern, a clear zone of haemolysis surrounding the bacterial colony.

Test organism was inoculated on skim milk agar and observed for proteolytic activity, yellow coloured colonies with or without a clear zone surrounding the bacterial colony. (**Figure 2**)

Test organism was inoculated on chrome azurol S agar plates procured from HiMedia Laboratories, India,[18] incubated at 37°C overnight and siderophore production with colour change from blue to orange or presence of yellow to light orange halo surrounding the bacterial colony. (**Figure 3**)

Figure 2: Proteolytic activity assessed using milk agar. Yellow coloured colonies with clear zone indicating proteolysis

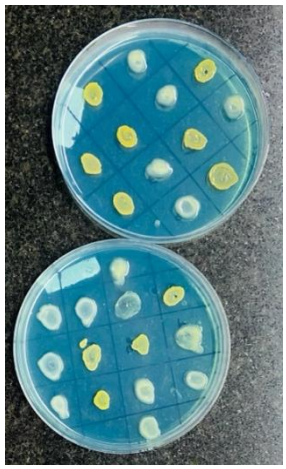

Figure 3: Siderophore production assessed using Chrome Azurol S (CAS) agar. Orange or presence of yellow to light orange halo surrounding the bacterial colony indicating siderophore production

3. Results

A total of 110 MDR *A. baumannii* isolates were included in the present study, of which 55 were clinical isolates and 55 were patient environmental isolates in whom MDR *A. baumannii* were isolated with antibiotic susceptibility pattern as shown in (**Table 1**).

Of the 55 Clinical MDR *A. baumannii* isolates, 37 (67.27%) isolates were from male patients and 18 (32.72%) were from female patients accounting to a male to female ratio of 3:1. Nineteen (34.54%) of the clinical isolates were from patients in the age group of 61-80 years followed by 15 (27.27%) isolates in the age group of 41-60 years and 14 (30.90%) isolates in the age group of 21-40 years.

Fifty-one (93%) isolates were from in-patients**,** of which 42 (82.35%) isolates were from various ICUs, Critical Care Medical ICU being the predominant ICU from where 12 (28.57%) isolates were isolated. Only 9 (17.64%) isolates were isolated from various wards in the hospital (private ward being the predominant ward with 6 (66.66%) isolates (**Table 2**).

Majority of the clinical isolates 35 (63.63%) were from endotracheal aspirate followed by pus sample with 20% (9) and sputum sample with 7.27% (3) isolates.

Patient's surroundings from whom MDR *Acinetobacter* were isolated were screened for the presence of MDR *Acinetobacter spp.* From each patient 5 environmental samples (Wall, trolley, floor, bathroom, bed and bed rails) were collected accounting to a total of 275 environmental samples.

Of the 275 environmental samples, 55 MDR *A. baumannii*, 42 Sensitive strain of *Acinetobacter*, 37 *E. coli*, 41 *Klebsiella* and 36 *Pseudomonas* and 39 isolates of MDR *E. coli* were isolated. 25 samples did not yield the growth of any bacteria.

Of the 55 environmental MDR *A. baumannii*, 24 (43.63%) isolates were isolated from bed and bed rails, 16 (29.09%) isolates were isolated from floor, 8 (14.54%) isolates were isolated from wall, 5 (9.09%) from trolley and 2 (3.63%) from patient's bathroom. (**Table 3**).

3.1. Detection of virulence factors of MDR A. baumannii.

1. Among the 55 clinical isolates, 34 (61.81%) were strong biofilm producers, 20 (36.36%) isolates were haemolytic, 29 (52.72%) isolates were proteolytic and 30 (54.54%) isolates were siderophore producers.
2. Among the 55 environmental isolates, 6 (10.90%) were strong biofilm producers, 15 (27.27%) isolates were haemolytic, 31 (56.36%) isolates were proteolytic and 19 (34.54%) isolates were siderophore producers **(Table 4).**

The phenotypic virulence determination between clinical and environmental isolates of MDR *Acinetobacter* was ($P<0.05$) statistically significant with demonstration that the clinical isolates are more virulent compared to the environmental isolates (**Table 4**).

Table 1: Antimicrobial susceptibility of 55 clinical and 55 environmental MDR *A. baumannii*

Drug	**(Clinical) Sensitive No.**	**(Clinical) Sensitive %**	**(Environmental) Sensitive No.**	**(Environmental) Sensitive %**
Colistin	12	21.82	18	32.72
Tigecycline	43	78.18	26	47.27
Minocycline	10	18.18	11	20.00
Gentamycin	6	10.91	6	10.91
Ceferazone/sulbactam	4	7.27	8	14.55
Amikacin	0	0.00	0	0.00
Cefepime	0	0.00	0	0.00
Levoflaxacin	1	1.82	0	0.00
Cotrimoxazole	2	3.64	1	1.82
Piperacillin/tazobactam	0	0.00	0	0.00
Ceftazidime	0	0.00	0	0.00
Ceftriaxone	0	0.00	0	0.00
Imipenem	0	0.00	0	0.00
Meropenem	0	0.00	0	0.00
Ciprofloxazin	0	0.00	0	0.00

Table 2: Distribution of 51 clinical in-patient isolates of MDR *A. baumannii* in various hospital locations

Respiratory ICU	3 (7.14%)
Surgery ICU	7 (16.66%)
Paediatric ICU	2 (4.76%)
Burns ICU	2 (4.76%)
Critical Care Medicine ICU	12 (28.57%)
Medicine ICU	6 (14.28%)
Neuro ICU	8 (19.04%)
Neonatal ICU	2 (4.76%)
Total	**42**
Private ward	6 (66.66%)
Male surgery ward	2 (22.22%)
General medicine - male ward	1 (11.11%)
Total	**9**
Grand Total	**51**

Table 3: Distribution of 55 MDR *A. baumannii* from patient surrounding

Bed & Rail	22 (40%)
Floor	18 (32.72%)
Wall	4 (7.27%)
Trolley	9 (16.36%)
Bathroom	2 (3.63%)
Grand Total	55

Table 4: Virulence distribution of 55 clinical and 55 environmental MDR *A. baumannii*

Virulence	Biofilm producer	Haemolytic	Proteolytic	Siderophore producer
Clinical MDR *A. baumannii*	37 (67.26%)	20 (36.36%)	29 (52.72%)	30 (54.54%)
Environmental MDR *A. baumannii*	25 (45.44%)	15 (27.27%)	31 (56.36%)	19 (34.54%)
Chi-Square Test				
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.716	1	.017	
Fisher's Exact Test				.022

4. Discussion

In this study, the phenotypic detection of virulence markers in MDR *A. baumannii* isolates from clinical and environmental sources was investigated.

The age distribution of MDR *A. baumannii* isolates in this study showed that the highest percentage of infections (34.54%) were in the 61-80 year age group. This pattern aligns with the study by Yadav *et al*[20] with 22.4% isolates from the age group above 65 years of age, likely due to a combination of immune suppression and the presence of chronic comorbidities such as cardiovascular diseases, diabetes, and respiratory disorders.

In our study, majority of the clinical MDR *A. baumannii* isolates were isolated from ICUs (82.35%) which was very similar to the study conducted by Boulesnam *et al*[19] where all the samples (100%) were isolated from the ICUs and also a study of Yadav *et al*[20] showed 49.6% ICU isolates.

In our study, majority of the clinical MDR *A. baumannii* isolates were isolated from endotracheal aspirate (63.63%) samples which was similar to the study of Yadav *et al*[20] with 47.2% isolates from respiratory samples, remarking that were majorly suffering from respiratory diseases.

In our study, majority of the environmental MDR *A. baumannii* were isolated from bed and bed rails (63.63%) of the patient surrounding, which is very similar to the study conducted by Boulesnam *et al*,[19] where 60% of the environmental MDR *A. baumannii* were isolated from beds of the patient surrounding.

The results of this study showed that a majority of the clinical isolates (61.81%) were strong biofilm producers, compared to only 10.90% of environmental isolates. But in the study conducted by Boulesnam *et al*,[19] 56% of environmental isolates and 50% of clinical isolates have a moderate to high potential for biofilm formation. Also in the Bardbari *et al*[21] study, the prevalence of strong biofilm producers in clinical and environmental isolates were 31.2% and 58.7% respectively, remarking significant differences in both the studies. Strong biofilm producers, especially among clinical isolates (61.81%), can adhere to surfaces like catheters, ventilators, and other medical devices. This increases the risk of healthcare-associated infections (HAIs). Implementation of aggressive cleaning protocols for surfaces and devices, routine surveillance, and possibly anti-biofilm coatings on medical equipment is recommended.[21]

In this study, 36.36% of clinical isolates exhibited haemolytic activity, whereas only 27.27% of environmental isolates were haemolytic. A study by Pournaras *et al*[22] also observed that clinical MDR *A. baumannii* strains exhibited higher haemolytic activity (45.5%), indicating the ability to lyse host cells, facilitating deeper tissue invasion and immune system evasion thereby causes severe infections, especially in immunocompromised patients. Should consider integrating haemolytic activity as a marker for identifying high-risk isolates.[22]

In the current study, both clinical and environmental isolates showed similar rates of proteolytic activity, with 52.72% of clinical isolates and 56.36% of environmental isolates being proteolytic. A study by Martínez *et al.*[23] found proteolytic activity in 50-60% of clinical isolates, which is in line with the clinical isolates in our study. Proteolytic enzymes help degrade host tissues and extracellular matrices, aiding in bacterial dissemination and infection establishment thereby exacerbate inflammation and tissue damage, prolonging patient recovery. Similar rates in both clinical and environmental isolates (around 50–56%) suggest environmental strains may be equally capable of establishing infections if introduced into susceptible hosts.[23]

In this study, 54.54% of clinical and 34.54% of environmental isolates tested positive for siderophore production. Similar results were observed by Lee *et al*[24] and Zong *et al*[25] on MDR *A. baumannii* isolates reported that 58% and 55% of the clinical isolates, whereas only 30% and 33% of environmental isolates tested positive for siderophore production.

Siderophores allow bacteria to scavenge iron from the host environment, which is critical for growth and virulence. The higher prevalence among clinical isolates (54.54%) supports its role in infection persistence in iron-limited environments like human tissue. Iron-chelating

agents as adjunctive therapies under research as be considered. Infection surveillance has to be enhanced to detect siderophore-positive strains, especially in ICUs where iron-restricted conditions are common.[24]

5. Conclusion

The results of this study emphasize the higher virulence of clinical *A. baumannii* isolates compared to their environmental counterparts. While environmental isolates did exhibit certain virulence factors. Nevertheless, ongoing monitoring of MDR *A. baumannii* from both clinical and environmental sources remains essential to track the evolution of virulence traits. Such surveillance is critical for implementing effective infection control strategies and promoting responsible antibiotic use in healthcare settings to address the growing threat posed by MDR *A. baumannii*.

6. Source of Funding

Self.

7. Conflict of Interest

None to declare.

8. Ethical Committee Approval

The authors of this manuscript declare that this scientific work complies with reporting quality, formatting and reproducibility guidelines set forth by the EQUATOR Network. The authors also attest that this study was determined to require the Institutional Ethics Committee review, and the corresponding approval number is JSS|MC|PG|0040|2022-2023|Dated 05-04-2023.

9. Clinical Trial Registry

The authors have not registered this study with the Clinical Trial Registry as it is not applicable.

10. Authors Contribution

MB (Monisha B) collected the samples, performed the preliminary and main tests, RPM (Rashmi P Mahale) analyzed the data, MRR and SSM (M Raghavendra Rao and Suchitra Shenoy M) constructed the agreements and errors.

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Cite this article: Basavaraju M, Mahale RP, Rao MR, Shenoy S. Phenotypic detection of virulence attributes in MDR Acinetobacter from a tertiary care hospital, South India. *Indian J Microbiol Res*. 2025;12(3):357–363.