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Prevalence, Antibiotic Resistant Pattern and Genotypic Detection of Acinetobacter baumannii Isolated from Different Clinical Specimens of Patients Admitted at a Tertiary Care Hospital in Bangladesh

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Abstract

Background: *Acinetobacter baumannii* infection treatment has become a clinical challenge due to the increasing resistance of the bacteria to different classes of antimicrobial agents and it needs to be identified before exposure to patients. **Objective:** This study aimed to determine the prevalence of *Acinetobacter baumannii* with its antibiotic-resistant patterns. **Methodology:** The study was a cross-sectional study that was done from 1st January 2019 to 31st December 2019 at the Microbiology Department of Dhaka Medical College. Clinical samples including endotracheal aspirates, blood, urine, sputum, wound swabs, and pus were collected from the patients from the intensive care unit, burn unit, general wards, and outpatient department of Dhaka Medical College and Hospital. A*cinetobacter baumannii* organisms were identified by biochemical tests and Gram staining, and the resistance pattern was determined by using the disc diffusion method for all antibiotics except for colistin, which was determined by minimum inhibitory concentration. **Results:** Among 500 clinical samples, 13.31% *Acinetobacter baumannii* were identified. Most of these showed resistance to fluoroquinolones, carbapenem, amikacin, the extended spectrum of cephalosporin, β-lactam, and β-lactamase inhibitors. However, the least resistant drug was tigecycline (8.88%). About 62.22% MDR, 28.89% A. baumannii were identified. **Conclusion:** Acinetobacter baumannii is showing increasing resistance to colistin and tigecycline.

Keywords: *Acinetobacter baumannii*; Colistin; Extensively Drug Resistant; Tigecycline; Pan Drug Resistant

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Introduction

Acinetobacter baumannii has become a nightmare because of its serious infections that are associated with higher mortality and morbidity[1]. This bacterium has been associated with endocarditis, septicemia, skin and soft tissue infection, meningitis, wound infection, and respiratory and urinary tract infections[2]. These are non-fastidious, non-fermentative, oxidase-negative, catalase-positive, strictly aerobic, non-motile, and gram-negative coccobacilli[3]. Among the Acinetobacter species, *A. baumannii* is mostly associated with nosocomial infections[4].

Acinetobacter baumannii is referred to as "Iraqibacter", as many *A. baumannii* infections have been reported from the soldiers who worked in Afghanistan and Iraq[5]. Therefore, Multi-Drug Resistant (MDR) *Acinetobacter baumannii* has risen due to cross-infection from war zones[6]. Moreover, the community-acquired infections of *Acinetobacter*

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baumannii have been rising gradually[4]. Generally, patients who are immunocompromised like premature neonates, older, have recently experienced major trauma, undergone surgery, or were previously admitted to contaminated Intensive Care Units (ICU) are at risk[7]. Patients who smoke and drink excessively are more likely to get community-acquired *Acinetobacter baumannii* pneumonia, mainly in tropical regions[8].

Several virulence factors, including lipopolysaccharides, outer membrane porins, phospholipases, proteases, capsular polysaccharides, iron-chelating systems, protein secretion systems, and biofilm formation, have been discovered in *Acinetobacter baumannii* through genomic and phenotypic investigations[9]. These virulence traits aid in the organism's adhesion and desiccation on inanimate surfaces for more than 14 days[10].

According to the World Health Organization, *Acinetobacter baumannii* is among the most dangerous ESKAPE pathogens (Enterococcus faecium, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter species*) that may successfully evade the effects of antibiotics[11]. As a result of the indiscriminate and widespread use of antibiotics, Multi-Drug and Extensively drug-resistant (XDR) *Acinetobacter baumannii* have now become serious issues on a global scale and are being reported more frequently[12].

The number of antibiotic classes used in the treatment of *Acinetobacter baumannii* infections in clinical practice has gradually decreased as a result of the accumulation of various resistance mechanisms in this organism, including β-lactamases, efflux pumps aminoglycoside-modifying enzymes, modifications of target sites, and permeability defects[4]. *Acinetobacter baumannii* is intrinsically resistant to various antibiotics, including amoxicillin, ertapenem, narrow-spectrum cephalosporins, chloramphenicol, and trimethoprim[13]. Additionally, by transferring plasmids, integrons, and transposons from other gram-negative bacteria, *Acinetobacter baumannii* acquired resistance genes against numerous antibiotics[14].

MDR *Acinetobacter baumannii* infections can currently be treated with limited pharmacological candidates and therapeutic approaches[15]. Colistin is currently used as a first-line antimicrobial against MDR *Acinetobacter baumannii*, either alone or in combination with other medications. They typically have in vitro potential activity against *Acinetobacter baumannii* strains, but they have a very narrow therapeutic spectrum and cause serious side effects such as neurotoxicity, and nephrotoxicity[16]. This study was aimed to determine the prevalence of *Acinetobacter baumannii* with its antibiotic-resistant patterns.

Methodology

Study Design and Population: In the Microbiology Department of Dhaka Medical College and Hospital (DMCH), Dhaka, Bangladesh, a cross-sectional investigation on 500 samples was conducted from January to December 2019 for a period of one year. Endotracheal aspirates (ETA) were collected from patients having suspected clinical infections and mechanical ventilation for more than 48 hours in the ICU. Adult patients with clinically suspected infections who were admitted to DMCH or received wound swabs (WS) for culture and sensitivity were included in the study regardless of sex or antibiotic use. These samples included blood, urine, sputum, wound swabs (WS), and pus.

Sample Collection and Processing

Endotracheal Aspirates (ETA): A 25- to 26-cm portion of a 50 cm long 14 FR sterile suction catheter (Medi Plus, India) was gently inserted through the endotracheal tube aseptically. Without using saline, ETA was collected via suction. Suction catheter cut tips were placed inside sterile test tubes, and 1 ml of sterile 0.9% normal saline was used to liquefy the tips. This solution was then homogenized by vertexing with sterile 1-2 glass beads for 1 minute, then centrifuged at 3000 rpm for 10 minutes.

Blood: After adhering to the aseptic protocol, A sterile disposable syringe was used to draw 5 ml of venous blood and place it into a blood culture vial with 50 ml of trypticase soy broth.

Wound Swab and Pus: Samples were aseptically collected from patients using sterile cotton-tipped swab sticks from clinically deep areas of the wound site before any cleansing. In the case of the collection of samples from dry surfaces, the swab was moistened with sterile normal saline. 5 ml of pus were aseptically removed from a drainage tube and placed in a sterile, leak-proof container.

Urine: About 10 to 12 ml of midstream clean catch urine was collected in a dry, sterile, wide-necked, leakproof container from patients after explaining the collection procedure. In the case of catheterized

patients, urine was collected after clamping the catheter. Further, the supernatant of the urine's centrifugation at 1000 g for five minutes was discarded. Pus cells were checked using microscopy.

Sputum: The samples were collected into a sterile container early in the morning before any mouthwash was used.

Isolation and Identification of *Acinetobacter baumannii*: Both blood agar and MacConkey agar media were used to inoculate the ETA, WS, and pus. They were then incubated at 37°C aerobically for 24 hours. Blood was cultured for 72 hours at 37 in trypticase soy broth, then sub-cultured for 24 hours at 37°C on blood agar and MacConkey agar media. Gram-stained sputum samples with more than 25 polymorphonuclear cells (PMNs)/LPF (10X) and 10 squamous epithelial cells/LPF (10X) were added to blood agar and MacConkey agar media, respectively, and incubated for 24 hours at 37°C aerobically. Polymorphonuclear cells (PMNs)/ HPF more than 5 in urine samples were inoculated in chromogenic agar media and incubated at 37°C for 24 hours aerobically.

Semi-Quantitative Culture of ETA17: Processed ETA was streaked using a sterile wire loop of 4 mm diameter 0.001 ml of fluid, inoculated on MacConkey and, blood agar media in three consecutive sectors, and incubated overnight at 37°C. According to the number of colonies in each of the three sectors, as indicated in Table 1, the growth was categorized as rare, light, moderate, and heavy. Significant growth was defined as heavy to moderate growth.

Table 1: Semi-Quantitative Reporting of Microbial Growth

Report	Number of colonies		
	1st sector	2nd sector	3rd sector
Rare	<10	0	0
Light	≥10	<5	0
Moderate	≥10	≥5	<5
Heavy	≥10	≥5	≥5

Phenotypic identification: Colony morphology on MacConkey agar (colorless), blood agar (cream-colored, non-pigmented, smooth, mucoid colonies, non-hemolytic, 1-2 mm in diameter), Gram staining (gram negative coccobacilli) and biochemical tests like- catalase tests (positive), oxidase test (negative), citrate utilization test (positive), indole test (negative), urease production (variable), and motility (non-motile) was done[18].

Genotypic identification: $bla_{oxa\text{-}51\text{-}like}$ gene was used[19].

Antimicrobial Susceptibility Test: All identified *A. baumannii* susceptibility to antimicrobial drugs was assessed using the modified Kirby-Bauer disc diffusion method on Mueller-Hinton agar plates, and zones of inhibition were measured following CLSI[20] guidelines. The zone of inhibition of tigecycline was interpreted using standards set by the United States Food and Drug Administration[21]. *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853 were used as control strains[22].

Antibiotic agents: Ceftazidime (30 mg), ceftriaxone (30 mg), cefepime (30 mg), amoxicillin 20µg and clavulanic acid 10µg, cefoxitin (30 mg), aztreonam (30 mg), ciprofloxacin (5 mg), imipenem (10 mg), amikacin (30 mg), gentamycin (10 mg), doxycycline (30 mg), tigecycline (15 mg), and colistin (8 mg/ml) (Oxoid Ltd, UK).

Interpretation: ESBL producers were defined as organisms resistant to penicillin, the first, second, and third generations of cephalosporins, and aztreonam (but not to cephamycins or carbapenems). These organisms were also inhibited by beta-lactamase inhibitors such as clavulanic acid[23]. MDR was defined as acquired resistance to at least one agent in three or more antibiotic categories. XDR was defined as resistance to at least one agent in all but two or fewer antibiotic categories. Pan drug-resistant (PDR) was defined as resistance to all agents in all antimicrobial categories[24].

Statistical Analysis: The data was analyzed using SPSS, version 21.0 (IBM SPSS Statistics for Windows, Version 21.0, Armonk, NY: IBM Corp.). Results were interpreted using mean, median, minimum, and maximum values together with standard deviation and were reported as frequency and percent. When there are gaps in the data, every effort is made to fill such gaps by using the denominator. The level of statistical significance was defined as a p-value of less than 0.05.

Ethical Clearance: The protocol was approved by the Research Review Committee (RRC) of the Microbiology Department, and the Ethical Review Committee (ERC) of Dhaka Medical College (DMC). Helsinki Declaration was followed after explaining the purpose and methods of research and information was kept confidential. All samples were collected after obtaining written informed consent from the patients. Data were collected anonymously and analyzed by a coding system.

Results

Among the 500 samples, 338 (67.67%) samples were culture positive of which, 40 (80%) were ETA, 51 (72.85%) were blood, 106 (70.66%) were urine, 124 (62%) were WS & pus, and 17 (56.67%) were sputum samples (Table 2).

Table 2: Culture positivity of clinical samples (n=500)

Samples	Culture Positive	Culture Negative	P value
ETA	40 (80.00%)	10 (20.00%)	
Blood	51 (72.85%)	19 (27.15%)	
Urine	106 (70.67%)	44 (29.33%)	
WS & pus	124 (62.00%)	76 (38.00%)	0.0001
Sputum	17 (56.67%)	13 (43.33%)	
Total	**338 (67.67%)**	**162 (32.32 %)**	

cinetobacter species isolated from various samples after genotypic identification by PCR using blaoxa-51-like gene. Among them isolated Acinetobacter baumannii and other species of Acinetobacter were, 14(82.35%) and 3(17.64%) from WS and pus samples; 13(92.8%) and one (7.14%) from ETA, 12(70.58%) and 5(29.41%) from blood samples; 4(66.7%) and 2(33.3%) from sputum samples, 2 (50%) and 2(50%) from urine samples respectively (Table 3).

Table 3: Acinetobacter species Isolated by PCR using blaoxa-51-like gene (n=58)

Samples	*Acinetobacter species*	*Acinetobacter baumannii*	Other *Acinetobacter species*
WS & pus	17	14 (82.4%)	3 (17.6%)
ETA	14	13 (92.9%)	1 (7.1%)
Blood	17	12 (70.6%)	5 (29.4%)
Sputum	6	4 (66.7%)	2 (33.3%)
Urine	4	2 (50.0%)	2 (50.0%)
Total	**58**	**45 (77.6%)**	**13 (22.4%)**

Acinetobacter baumannii was mostly distributed in ETA samples 11(24.44%) from ICU, wound swab and pus samples 7(15.54%) and blood samples 6 (13.33%) from wards (Table 4).

Table 4: Acinetobacter baumannii isolated from Different Samples and Sources (n=45)

Samples	ICU	Ward	Burn	OPD
WS & pus	2 (4.4%)	7 (15.5%)	3 (6.7%)	2 (4.4%)
ETA	11 (24.4%)	2 (4.4%)	0 (0.0%)	0 (0.0%)
Blood	4 (8.9%)	6 (13.3%)	2 (4.4%)	0 (0.0%)
Sputum	0 (0.0%)	2 (4.4%)	0 (0.0%)	2 (4.4%)
Urine	0 (0.0%)	1 (2.2%)	1 (2.2%)	0 (0.0%)
Total	**17 (37.8%)**	**18 (40.0%)**	**6 (13.3%)**	**4 (8.9%)**

The antimicrobial resistance pattern of isolated A. baumannii were recorded. Here, all were resistant to amoxiclav, cefoxitin, and ceftazidime, 43(95.55%) were resistant to piperacillin-tazobactam, ceftriaxone, cefepime and ciprofloxacin, 42(93.33%) were resistant to imipenem and amikacin, 13 (28.89%) were resistant to colistin and 4(8.88%) were resistant to tigecycline (Table 5).

Table 5: Antimicrobial Resistance Pattern among Isolated *Acinetobacter baumannii*

Antimicrobial drugs	Frequency	Percent
Amoxiclav	45	100.0
Piperacillin-tazobactam	43	95.5
Cefoxitin	45	100.0
Ceftazidime	45	100.0
Ceftriaxone	43	95.5
Cefepime	43	95.5
Aztreonam	35	77.8
Ciprofloxacin	43	95.5
Imipenem	42	93.3
Tigecycline	4	8.9
Doxycycline	26	57.8
Amikacin	42	93.3
Gentamycin	35	77.8
Colistin	13	28.9

Colistin antimicrobial susceptibility test for colistin was determined by the agar dilution method. Among the isolated strains of A. baumannii, 62.22% were MDR, 28.89% were XDR and 8.88% were PDR (Table 6).

Table 6: Distribution of MDR, XDR, and PDR *A. baumannii* (n=45)

Samples	MDR	XDR	PDR
ETA	0(0.0%)	11(24.4%)	3(6.66%)
WS & pus	14(31.1%)	2 (4.4%)	1(2.2%)
Blood	12(26.7%)	0(0.0%)	0(0.0%)
Urine	2(4.4%)	0(0.0%)	0(0.0%)
Sputum	0(0.0%)	0(0.0%)	0(0.0%)
Total	**28(62.2%)**	**13(28.89%)**	**4(8.9%)**

Discussion

Acinetobacter baumannii has grown to be a significant healthcare facility-acquired bacterial infection that is linked to meningitis, wound and soft tissue infections, catheter-related bacteremia, urinary tract infections, post-surgical endocarditis, and Ventilator-associated Pneumonia (VAP) over the past few decades[25]. It can survive for a long time in hostile surroundings (walls, surfaces, and medical equipment) in hospital settings[26],

and it is Chromosomally resistant to several types of antibiotics[27].

In this study, out of 500 clinical samples, 338 samples (67.6%) yielded culture-positive growth of which 58 (17.15%) *Acinetobacter species* were identified. Among them, 45 (13.31%) isolated *Acinetobacter species* showed the presence of blaoxa-51-like gene and were identified as *Acinetobacter baumannii*. This was similar to the study conducted by Fallah et al[28]. A recent study by Jahan[29] from DMCH reported that 14.04% of *Acinetobacter baumannii* were isolated from various clinical samples and different studies in the world as well as in India have also shown an increased prevalence of *Acinetobacter baumannii* ranging from 13 to 68% and even higher over the last two decades[30] which are in agreement with this study.

Most of the *Acinetobacter baumannii* isolates (28.9%) were found in endotracheal aspirates, of which 24.4% were found in patients who had been hospitalized in the intensive care unit. According to a recent study by Jain et al[31] in India, *Acinetobacter baumannii* accounted for around 26.2% of the cases of LRTI in patients who were admitted to the ICU, which was consistent with the results of the present study.

This study showed that the isolated *Acinetobacter baumannii* were highly resistant (95.5% to 100.0%) to the extended spectrum of cephalosporins which was very similar to the study by Nesa[32] from BSMMU reported that 93.5% to 100.0% *Acinetobacter baumannii* were resistant to the extended spectrum of cephalosporins. These findings were in agreement with the current study. No recent data regarding the use of extended-spectrum cephalosporins among *Acinetobacter baumannii* were found in Asian countries. However, a study in India by Guckan et al[33] reported that 74.8% of *Acinetobacter baumannii* were resistant to ceftazidime. The higher rate of resistance might be due to the indiscriminate use of cephalosporins in recent years.

In the present study, resistance to carbapenem was observed in 93.33% *Acinetobacter baumannii* clinical isolates. A previous study in BSMMU by Nesa (2018) reported that 94.8% and in India a study by Jain et al[31] showed that 96.4% carbapenem resistance was observed in *Acinetobacter baumannii* isolates which were in agreement with the study.

According to these findings, 62.22% MDR, 28.89% XDR, and 8.88% PDR *Acinetobacter baumannii* were clinically isolated which was closer to the study by Fragkou et al[34] who reported the isolation of 52.3% MDR, 28.7% XDR, and 19% PDR *Acinetobacter baumannii* strains from different clinical samples. A study by Nesa[32] from BSMMU showed the presence of 75.29% MDR *Acinetobacter baumannii*. However, no study was found regarding XDR and PDR *Acinetobacter baumannii* isolates in Bangladesh.

Conclusion

According to the results of this study, A*cinetobacter baumannii* is highly resistant to the majority of routinely used antibiotics, including carbapenems, lactam and lactamase inhibitors, extended-spectrum cephalosporins, aminoglycosides, and fluoroquinolones. They included 28.89% colistin-resistant and 8.88% tigecycline-resistant strains, worrying numbers that prompted the development of XDR and PDR strains of *Acinetobacter baumannii* that require alternative therapies.

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Conflict of Interest
There are no conflicts of interest.

Financial Disclosure
No funding was provided to the author(s) for this study.

Authors' contributions
Nazmun Sharmin conceived and designed the study, analyzed the data, interpreted the results, and wrote up the draft manuscript.
Md. Mahbub E Khoda contributed to the analysis of the data, interpretation of the results and critically reviewing the manuscript.
Mohammad Nazim Uddin. involved in the manuscript review and editing. All authors read and approved the final manuscript.

Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate
Ethical approval for the study was obtained from the Institutional Review Board. As this was a prospective study the written informed consent was obtained from all study participants. All methods were performed in accordance with the relevant guidelines and regulations.

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