World Journal of Surgery and Surgical Research

Short Communication
Published: 10 Dec, 2022

Prevalence of Virulence Genes in *Acinetobacter baumannii* Isolated from Clinical Samples in Mymensingh Medical College Hospital

Ara H*

Department of Microbiology, Mymensingh Medical College, Bangladesh

Abstract

Acinetobacter baumannii is an opportunistic bacterial pathogen that is the most important cause of hospital-acquired infections. The objective of this study was to evaluate the predominance and determination of virulence encoding genes in *A. baumannii* isolates. During study period from February 2019 to March 2020 of 380 clinical samples including endotracheal aspirates (70), wound swab/pus (175), urine (70) and blood (65) analyzed in inpatients admitted to the hospital in different unit like ICU, Surgery and Burn unit of Mymensingh Medical College Hospital. Out of 380 studied samples, 130 (34.21%) strains were yielded growth. Among 130 isolates, *Acinetobacter* spp. were 49 (37.69%). Totally, 39 (79.59%) were *Acinetobacter baumannii* which was detected by molecular technique PCR. Furthermore, the determination of virulence genes csgA and fimH detected by PCR. Among two studied virulence genes, csgA (38.46%) was the most prevalent virulent genes associated with disease severity and co-morbidity of the patient in *A. baumannii* infections.

Keywords: *Acinetobacter baumannii*; Virulence genes; Bacterial pathogen

Introduction

Acinetobacter baumannii is a ubiquitous aerobic gram-negative, oxidase-negative, catalase positive, citrate positive, non-fermentative, non-motile coccobacilli. Wide range of illness and death are the major criteria of hospital acquired infections caused by *Acinetobacter* spp., which included urinary tract, skin and soft tissue infections, pneumonia and bacteremia especially in patients with severe health condition [1-3]. *Acinetobacter* is well-thought-out to be an organism of low virulence [4,5]. Disease pathogenesis caused by *A. baumannii* is resulting from the presence of latent virulence genes [6,7]. Bacterial attachment is alleged to be an initial step in gram-negative nosocomial pneumonia [8]. Type 1 fimbriae (fimH) is adhesive virulence factor and non-fimbria adhesive virulence factor is curli fibers (csgA) are take part in adherence and biofilm formation and provide bacteria, the capacity to be concealed of host immune system and triggering infections more and take nutrition from the host [9,10]. And so, the study was done in order to evaluation of prevalence of virulence genes of *A. baumannii* isolated from various clinical samples in Mymensingh Medical College Hospital.

Materials and Methods

In this cross-sectional study, from March 2019 to February 2020 (a one-year period) totally 39 *Acinetobacter baumannii* were isolated at Mymensingh Medical College Hospital in the department of microbiology, MMC. The isolates were collected from different clinical samples such as endotracheal aspirates, wound swab/pus and urine. Primary identification done by standard bacteriological method [11] and confirmed by the PCR amplification for targeting *OXA-51* gene [12]. Finally, all the isolates were evaluated with the PCR technique to detect the virulence genes of interest [13]. The confirmed bacterial isolates were stored at -80°C.

Molecular technique

The bacterial DNA was extracted by boiling method [14]. For PCR amplification of the *OXA-51* (Table 1) gene and virulence genes specific primer were used [12,13]. PCR amplification procedure was performed using 25 µ/L master mix containing DNase free water 18.25 µ/L, buffer 2.5 µ/L, dNTP 2 µ/L, Taq polymerase 0.25 µ/L, primer 1 µ/L and DNA 1 µ/L. PCR amplification was done in thermal cycler device [12,13]. After that gel electrophoresis of the amplified DNA done to determine targeted band 353 bp, 200 bp and 508 bp respectively [12,13].

OPEN ACCESS

Correspondence:
Hosne Ara, Department of Microbiology, Mymensingh Medical College, Bangladesh, Tel: 01722521309; E-mail: ava.hosneara@gmail.com

Received Date: *08 Nov 2022*
Accepted Date: *07 Dec 2022*
Published Date: *10 Dec 2022*

Citation:
Ara H. Prevalence of Virulence Genes in Acinetobacter baumannii Isolated from Clinical Samples in Mymensingh Medical College Hospital. World J Surg Surgical Res. 2022; 5: 1429.

Table 1: Primers used for PCR amplification of the studied genes.

Primer name	Primer sequence	Product size (bp)	Reference
OXA- 51	TAATGCTTGATCGGCCTTG	353	12
	TGGATTGCACTTCATCTTG		
csgA	ACTCTGACTTGACTATTACC	200	13
	AGATGCAGTCTGGTCAAC		
fimH	TGCAGAACGGATAAGCCGTGG	508	13
	AGATGCAGTCTGGTCAA		

Table 2: Isolated organisms from different samples (N= 380).

Type of Sample	No. of isolate	Percentage%
Endotracheal aspirate	37	75
Wound swab	5	10.2
Catheterized urine	5	10.2
Blood	2	4.08

Table 3: Distribution of isolated *Acinetobacter* spp (n=49) among different type of samples.

Type of Sample	No. of isolate	Percentage (%)
Endotracheal aspirate	37	75
Wound swab	5	10.2
Catheterized urine	5	10.2
Blood	2	4.08

Showing distribution of isolated *Acinetobacter* spp. (n=49) among different type of samples. Majority of the isolates were obtained from endotracheal aspirate

Table 4: Distribution of *Acinetobacter baumannii* among *Acinetobacter* spp. isolates (n=49).

Acinetobacter baumannii	39 (79.59%)
Other than *A. baumannii*	10 (20.41%)

Showing among 49 *Acinetobacter* spp. 39 (79.59%) were *Acinetobacter baumannii* which was identified by PCR targeting *OXA-51* like gene and 10 (20.41%) were other than *Acinetobacter baumannii*

Discussion

Nowadays controlling infections caused by gram negative pathogen bacteria such as *Acinetobacter baumannii* has grown into a challenge [15]. This study was led on critically ill patients of intensive care unit, Surgery and Burn Unit and sample size was 380. Amongst them, 130 (34.21%) yielded growth correlates with the findings from BIRDEM [16] hospital. In India, reported 38.41% [17] and 23.15% [18] respectively. Degree of culture positivity and isolation number of organisms from various samples vary by hospitals and country.

In the current study, *Acinetobacter baumannii* was the most commonly isolated pathogen, 49 (37.69%) out of 130 strains, which relates with the finding of Bangladesh that was 37.5% [19]. Another study from Bangladesh reported 7.24% [20] and 34% [21]. In India, reported 42.02% [18]. The frequency of Acinetobacter from clinical samples varies by hospital, patient population, exposure to antibiotics, types of patient and changes over time.

Most of the *Acinetobacter* 37 (75%) was obtained from endotracheal aspirates in the present study that was similar to the study from Italy [22] and was 72.2%. Another study from India, reported 63.15% [18] and 53.39% [23] respectively. Greater isolation rate from endotracheal aspirates perhaps due to the fact that most patients either had previous respiratory difficulties or were in air duct or because of low resistance or serious illness.

Table 5: Shows distribution of virulence genes in *Acinetobacter baumannii* isolates (n=39).

Type of samples	Number of virulence gene (%)		Co-morbidity
	csgA	fimH	
Endotracheal aspirate (30)	10 (33)	4 (10.25)	CVD, DM, COPD
Catheterized urine (5)	3 (60)	3 (60)	CKD, UTI
Wound swab (2)	1 (50)	0 (0)	DM
Blood (2)	1(50)	0 (0)	Septicemia
Total (39)	15 (38.46)	7 (17.95)	

Shows distribution of virulence genes in *Acinetobacter baumannii* isolates (n=39). csgA (38.46%) was the most prevalent virulence gene and fimH were (17.95%) mostly were detected from endotracheal aspirates with associated co-morbidity were CVD, DM and COPD

CVD: Cerebrovascular Disease; DM: Diabetes Mellitus; COPD: Chronic Obstructive Pulmonary Disease; CKD: Chronic Kidney Disease; UTI: Urinary Tract Infection

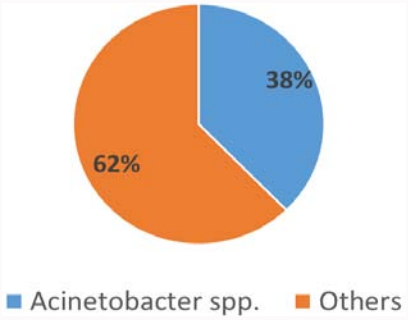

Figure 1: Distribution of *Acinetobacter* spp. isolated from different samples (n=130).

In the current study, among 49 *Acinetobacter* spp., *A. baumannii* were 39 (79.59%) that harboring the *OXA-51* like gene. This finding was correlates with the study from South Africa [24] reported 83% positive for *OXA-51* like gene while from Saudi Arabia [23] reported 72.7% (Figures 1-3).

Adhesive virulence factors are deliberated an important factor in adhesion, biofilm formation and existence of most microbes and their pathogenicity in the host [25]. In our study, csgA gene were 38.46% (15/39) and fimH gene were 17.95 (7/39). Study Iran [26], reported that prevalence of csgA and fimH gene were 54% and 60% respectively which was higher than the present study. Another study from Iran [27] reported prevalence of csgA and fimH genes were 12.39% and 74.38% respectively. Prevalence of csgA virulence gene among the samples of UTIs were 55% [28] which was similar to our findings. This two-virulence gene were prevalent in patients with previous co-morbidity such as Diabetes Mellitus, Chronic Obstructive Pulmonary Disease and patient with devices.

Results

An over-all of 380 samples were examined which included tracheal aspirates (70), wound swab/pus (175), catheterized urine (70) and Blood (65). Growth was obtained in 34.21% of the samples yielding 130 organisms. Common were isolated from endotracheal aspirates.

Conclusion

In conclusion, we identified high prevalent and virulent strains of *A. baumanniiin* the respiratory and urinary tract infections of co-morbid patients hospitalized in Mymensingh Medical College Hospital. Entirely, respiratory infections had the highest prevalence

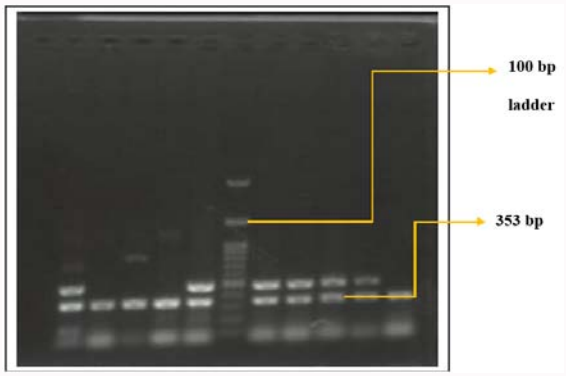

Figure 2: Amplification product (353bp) for *OXA-51* like gene for *Acinetobacter baumannii.*
PCR was done to detect the *OXA-51* like gene for *Acinetobacter baumannii* are showing bands of the amplified product of 353 bp regions and is indicated by arrow with 100 bp ladder

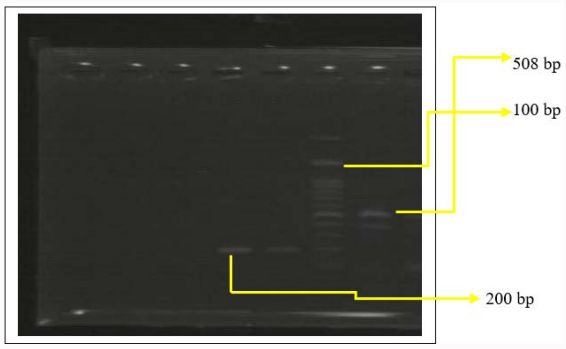

Figure 3: Amplification product 200bp csgA and 508bp fimH virulence genes for *Acinetobacter baumannii*.
PCR was done to detect the csgA and fimH virulence genes *Acinetobacter baumannii* are showing bands of the amplified product of 200bp & 508bp regions and is indicated by arrow with 100 bp ladder

isolated bacteria and also csgA were the most commonly detected virulence gene associated with co-morbidity and disease severity of the patient.

References

1. Karageorgopoulos DE, Falagas ME. Current control and treatment of multidrug-resistant *Acinetobacter baumannii* infections. Lancet Infect Dis. 2008;8(12):751-62.
2. Cisneros JM, Rodríguez-Baño J. Nosocomial bacteremia due to *Acinetobacter baumannii:* Epidemiology, clinical features and treatment. Clin Microbiol Infect. 2002;8(11):687-93.
3. Kuo SC, Chang SC, Wang HY, Lai JF, Chen PC, Shiau YR, et al. Emergence of extensively drug-resistant *Acinetobacter baumannii* complex over 10 years: Nationwide data from the Taiwan Surveillance of Antimicrobial Resistance (TSAR) program. BMC Infect Dis. 2012;12:200.
4. Kurcik-Trajkovska B. *Acinetobacter* spp.-A serious enemy threatening hospitals worldwide. Maced J Med Sci. 2009;2(2):157-62.
5. Kanafani ZA, Kanj SS. Acinetobacter infection: Treatment and prevention. 2015.
6. Eraç B, Yilmaz FF, Hosgor Limoncu MH, Ozturk I, Aydemir S. Investigation of the virulence factors of multidrug-resistant *Acinetobacter baumannii* isolates. Mikrobiyol Bul. 2004;48(1):70-81.
7. Eijkelkamp BA, Stroeher UH, Hassan KA, Paulsen IT, Brown MH. Comparative analysis of surface-exposed virulence factors of *Acinetobacter baumannii*. BMC Genomics. 2014;15(1):1020.
8. Hornick DB, Allen BL, Horn MA, Clegg S. Fimbrial types among respiratory isolates belonging to the family Enterobacteriaceae. J Clin Microbiol. 1991;29(9):1795-800.
9. Barnhart MM, Chapman MR. Curli biogenesis and function. Annu Rev Microbiol. 2006;60:131-47.
10. McConnell MJ, Actis, Pachón J. *Acinetobacter baumannii*: human infections, factors contributing to pathogenesis and animal models. FEMS Microbiol Rev. 2013;37(2):130-55.
11. Constantiniu S, Romaniuc A, Iancu LS, Filimon R, Tarași I. Cultural and biochemical characteristics of *Acinetobacter* spp. strains isolated from hospital units. Prev Med. 2004;12(3-4):35-42.
12. Turton JF, Ward ME, Woodford N, Kaufmann ME, Pike R, Livermore DM, et al. The role of ISAba1 in expression of OXA carbapenemase genes in *Acinetobacter baumannii*. FEMS Microbiol lett. 2006:258(1):72-7.
13. Momtaz H, Seifati SM, Tavakol M. Determining the prevalence and detection of the most prevalent virulence genes in *Acinetobacter baumannii* isolated from hospital infections. Int J Med Lab. 2015;2(2):87-97.
14. Andriamanantena TS, Ratsima E, Rakotonirina HC, Randrianirina F, Ramparany L, Carod JF, et al. Dissemination of multidrug resistant *Acinetobacter baumannii* in various hospitals of Antananarivo Madagascar. Ann Clin Microbiol Antimicrob. 2010;9:17.
15. Braun G, Vidotto MC. Evaluation of adherence, hemagglutination, and presence of genes codifying for virulence factors of *Acinetobacter baumannii* causing urinary tract infection. Mem Inst Oswaldo Cruz. 2004;99(8):839-44.
16. Barai L, Fatema K, Haq JA, Faruq MO, Ahsan AA, Morshed MA, et al. Bacterial profile and their antimicrobial resistance pattern in an intensive care unit of a tertiary care hospital of Dhaka. Ibrahim Med Coll J. 2010;4(2):66-9.
17. Pal N, Sujatha R, Kumar A. Phenotypic and genotypic identification of *Acinetobacter baumannii* with special reference to blaoxa-51 like gene and its antimicrobial susceptibility pattern from intensive care units in Kanpur. IJCMR. 2017;4(5):1154-8.
18. Kaur T, Putatunda C, OBeroi A, Vyas A, Kumar G. Prevalence and drug resistance in *Acinetobacter* sp. isolated from intensive care units' patients in Punjab, India. Asian J Pharm Clin Res. 2018;11(14):88.
19. Ahsan AA, Fatema K, Barai L, Faruq MO, Ahmed F, Saha DK, et al. Prevalence and antimicrobial resistance pattern of blood isolates in patients of septicemia in ICU: Single center observation. Bangladesh Crit Care J. 2016;4(2):100-4.
20. Paul S, Jhora ST, Dey PP, Begum BA. Detection of Extended Spectrum Beta-Lactamase (ESBL) producing gram negative bacteria from clinical specimens of sir Salimullah Medical College and Mitford hospital. Bangladesh J Med Microbiol. 2016:10(1):8-12.
21. Khatun MN, Shamsuzzaman SM, Fardows J, Siddique AB, Joly SN. Identification of bacterial isolates from endotracheal aspirate of patients in intensive care unit and their antimicrobial susceptibility pattern. J Enam Med Col. 2018;8(2):67-73.
22. Mammina C, Palma DM, Bonura C, Aleo A, Fasciana T, Sodano C, et al. Epidemiology and clonality of carbapenem-resistant *Acinetobacter baumannii* from an intensive care unit in Palermo, Italy. BMC Res Notes. 2012;5:365.
23. Amudhan SM, Sekar U, Arunagiri K, Sekar B. OXA beta-lactamase-mediated carbapenem resistance in *Acinetobacter baumannii*. Indian J Med Microbiol. 2011;29(3):269-74.
24. Kock MM, Bellomo AN, Storm N, Ehlers MM. Prevalence of carbapenem resistance genes in *Acinetobacter baumannii* isolated from clinical specimens obtained from an academic hospital in South Africa. South Afr

J Epidemiol Infect. 2013;28(1):28-32.

25. Doughari HJ, Ndakidemi PA, Human IS, Benade S. The ecology, biology and pathogenesis of *Acinetobacter* spp.: An overview. Microbes Environ. 2011;26(2):101-12.

26. Farahami A, Khodarahmi R. Frequency of adhesive virulence factors in Carbapenemase-producing *Acinetobacter baumannii* isolated from clinical samples. Asian J Biol Sci. 2014;7(4):158-64.

27. Momtaz H, Seifati SM, Tavakol M. Determining the prevalence and detection of the most prevalent virulence genes in *Acinetobacter baumannii* isolated from hospital infections. Int J Med Lab 2015;2(2):87-97.

28. Daryanavard R, Safaei HR. Virulence genes and antimicrobial resistance properties of *Acinetobacter baumannii* isolated from pediatrics suffered from UTIs. Int J Adv Res Biol Sci. 2015;2(11):272-9.