OTJHS
Online Turkish Journal of Health Sciences

e-ISSN: 2459-1467

OTSBD
Online Türk Sağlık Bilimleri Dergisi

Online Turkish Journal of Health Sciences 2025;10(3):203-210 *Online Türk Sağlık Bilimleri Dergisi 2025;10(3):203-210*

Determining the Risk Factors for Nosocomial Multidrug-Resistant Acinetobacter Infections in Patients in Intensive Care Units and Genotyping of Isolates

Yoğun Bakım Ünitesinde Nosokomiyal Çoklu İlaç Dirençli Acinetobacter Enfeksiyonları için Risk Faktörlerinin Belirlenmesi ve İzolatların Genotiplendirilmesi

[1]Şirvan ELMAS DAL, [2]Funda MEMİŞOĞLU, [2]Barış OTLU, [3]Çiğdem KUZUCU, [2]Yasemin ERSOY

[1]Malatya Turgut Özal University Faculty of Medicine, Department of Infectious Diseases and Clinical Microbiology, Malatya, Türkiye
[2]İnönü University Faculty of Medicine, Department of Infectious Diseases, Malatya, Türkiye
[3]İzmir Tınaztepe University Faculty of Medicine, Department of Clinical Microbiology, İzmir, Türkiye

Şirvan Elmas Dal: https://orcid.org/ 0000-0003-2048-0796
Funda Memişoğlu: https://orcid.org/0000-0003-3905-1182
Barış Otlu: https://orcid.org/0000-0002-6220-0521
Çiğdem Kuzucu: https://orcid.org/0000-0003-4507-5914
Yasemin Ersoy: https://orcid.org/0000-0001-5730-6682

ABSTRACT

Objective: This study aimed to identify the risk factors for hospital-acquired *Acinetobacter baumannii* infections in an intensive care unit (ICU), delineate the antibiotic resistance profiles of isolates, and elucidate the clonal relationships among strains through genotypic analysis.

Materials and Methods: This prospective case-control study was conducted between April 2011 and March 2012 and included 213 patients. The antibiotic susceptibility of *Acinetobacter* strains was evaluated using the Kirby–Bauer disk diffusion method. The DiversiLab system was used to determine the clonal relationships among *A. baumannii* strains.

Results: The key independent risk factors for hospital-acquired *A. baumannii* infections included age, mechanical ventilator use, tracheotomy, percutaneous endoscopic gastrostomy (PEG), carbapenem administration, and non-use of cephalosporins. High multi-antibiotic resistance was observed in 94% of the isolates. Furthermore, a substantial clonal closeness with an 86% clustering rate was observed among the isolates, with the largest cluster (P5) comprising 24 isolates persisting for approximately 14 months in the hospital setting.

Conclusions: The findings indicate the need for targeted preventive measures against specific risk factors for *A. baumannii* infections in ICUs. The genotypic analysis revealed significant clonal spread, necessitating enhanced infection control strategies.

Keywords: *Acinetobacter* infections, genotype, intensive care units

ÖZ

Amaç: Bu çalışma, Yoğun Bakım Ünitesi 'nde (YBÜ) hastane kaynaklı *Acinetobacter baumannii* enfeksiyonları ile ilişkili risk faktörlerini belirlemeyi, izolatların antibiyotik direnç profillerini çizmeyi ve genotipik analiz yoluyla suşlar arasındaki klonal ilişkileri aydınlatmayı amaçlamaktadır.

Materyal ve Metot: Bu çalışma Nisan 2011 ile Mart 2012 tarihleri arasında prospektif vaka-kontrol çalışması olarak yürütülmüş olup 213 hastayı kapsamaktadır. *Acinetobacter* suşlarının antibiyotik duyarlılıkları, Kirby–Bauer disk difüzyon yöntemi kullanılarak değerlendirilmiştir. *A. baumannii* suşları arasındaki klonal ilişkilerin belirlenmesi için DiversiLab sistemi kullanılmıştır.

Bulgular: Hastane kaynaklı *Acinetobacter* enfeksiyonu için bağımsız anahtar risk faktörleri yaş, mekanik ventilatör kullanımı, trakeotomi, perkütan endoskopik gastrostomi (PEG) uygulaması, karbapenem verilmesi ve sefalosporin kullanılmamasıdır. İzolatların %94'ünde yüksek çoklu antibiyotik direnci gözlemlenmiştir. Ayrıca izolatlar arasında %86 oranında belirgin bir klonal yakınlık gözlenmiş, en büyük kümeyi (P5) yaklaşık 14 ay boyunca hastane ortamında süregelen 24 izolat oluşturmuştur.

Sonuç: Bulgular, YBÜ'lerinde *A. baumannii* enfeksiyonları için belirli risk faktörlerine karşı hedeflenmiş önleyici tedbirlerin gerekliliğini vurgulamaktadır. Genotipik inceleme, artırılmış enfeksiyon kontrol stratejilerini gerektiren önemli bir klonal yayılımı ortaya koymaktadır.

Anahtar Kelimeler: *Acinetobacter* enfeksiyonları, genotip, yoğun bakım üniteleri

Sorumlu Yazar / Corresponding Author:
Şirvan Elmas Dal
Malatya Turgut Özal University Faculty of Medicine, Department of Infectious Diseases and Clinical Microbiology, Malatya, Türkiye
Tel: +905053858991
E-mail: sedal44@hotmail.com

Yayın Bilgisi / Article Info:
Gönderi Tarihi/ Received: 19/12/2024
Kabul Tarihi/ Accepted: 29/08/2025
Online Yayın Tarihi/ Published: 15/09/2025

Atıf / Cited: Elmas Dal Ş and et al. Determining the Risk Factors for Nosocomial Multidrug-Resistant Acinetobacter Infections in Patients in Intensive Care Units and Genotyping of Isolates. *Online Türk Sağlık Bilimleri Dergisi* 2025;10(3):203-210. doi: 10.26453/otjhs.1604466

INTRODUCTION

Acinetobacter baumannii, recognized for its swift emergence of antibiotic resistance, frequently results in difficult-to-manage outbreaks.[1] This bacterium primarily occurs as a causative agent in bacteremia and respiratory tract diseases. According to the Centers for Disease Control and Prevention's Antibiotic Resistance Threats in the United States 2019 report, carbapenem-resistant *A. baumannii* has been reported as an urgent pathogenic threat.[2] The invasive procedures such as endotracheal mechanical ventilation and implanted invasive devices, admission to intensive care, recent surgery, use of broad-spectrum antibiotics, ineffective treatment, and septic shock at diagnosis are reported as risk factors for multidrug-resistant *A. baumannii* colonization or infection and higher mortality.[3,4] In addition, molecular techniques such as polymerase chain reaction are very useful in identifying *A. baumannii* and examining the genetic affiliation of clinical isolates of *A. baumannii* in hospitals.[5]

This study aimed to explore the risk factors for *A. baumannii* infections in intensive care units (ICUs). Furthermore, it aimed to provide insights and recommendations to reduce infection rates, formulate treatment procedures via culture antibiogram evaluations, and determine the clonal relationships of strains through genotypic analysis.

MATERIALS AND METHODS

Ethics Committee Approval: This study was approved by the ethics committee of XX University (Date: 01.03.2011, decision no: 2011/14) and was conducted in accordance with the principles of the Declaration of Helsinki.

Study Design: This prospective case-control study was conducted at XX Medical Center in the adult ICU from April 2011 to March 2012 to identify the risk factors for hospital-acquired *A. baumannii* infections. It included 213 patients who were categorized into 108 cases and 105 controls. During the study period, the patients who were followed up in the adult ICU and developed a hospital-acquired *Acinetobacter* infection attack were included as the case group, and the patients who did not develop an infection attack were included as the control group. Each participant completed an "Epidemiological Patient Study Form" and the "Apache II Form."[6] The diagnostic criteria established by the Centers for Disease Control and Prevention in the United States[7] were used for hospital infection identification.

Clinical specimens from hospitalized patients were grown on blood and eosin–methylene blue agar. Traditional techniques were employed for the gram staining of colonies classified as *Acinetobacter*. The antibiotic susceptibility of *Acinetobacter* strains obtained from the clinical samples was evaluated using the Kirby–Bauer disk diffusion method, adhering to the norms set by the Clinical and Laboratory Standards Institute.[8] *Pseudomonas aeruginosa* ATCC 27853 was used as the reference strain. The Buyyon microdilution method was employed to assess the susceptibility of colistin and tigecycline, with minimum inhibitory concentration (MIC) ≤4 and >8 μg/mL for cholismetate sodium indicating susceptibility and resistance to colistin, respectively.[9] The MIC thresholds for tigecycline in the *Acinetobacter* strains were assessed in accordance with the Food and Drug Administration guidelines (≤2 μg/mL for susceptibility).[10] Commercially acquired antibiotic powders included colistin (cholismetate sodium) and tigecycline. The classification of multidrug-resistant strains was based on the categories provided by Falagas et al.[11]

Repetitive extragenic palindromic polymerase chain reaction (REP–PCR) using the DiversiLab system (bioMerieux, France) was employed to investigate the clonal relationship among *A. baumannii* strains. Pearson's correlation coefficient was used for the band analysis, whereas the unweighted pairwise grouping mathematical averaging was adopted for the clustering analysis.

Statistical Analysis: The Statistical Package for Social Sciences version 16.0 was used for data analysis. The Shapiro–Wilk test was employed to evaluate the normality of the groups' distributions. The unpaired t-test, chi-squared test, or Fisher's exact test was used for intragroup comparisons. Univariate logistic regression analysis revealed the risk factors for hospital-acquired *Acinetobacter* infections, including variations with P <0.25 in multivariate logistic regression models. Through retrospective elimination, the best predictive risk factor for *Acinetobacter* infection was identified by computing the odds ratios, 95% confidence intervals, and significance levels. A P-value of 0.05 was deemed statistically significant.

RESULTS

The predominant *Acinetobacter* infection identified was ventilator-associated pneumonia (VAP), which occurred in 68 (63%) patients. Invasive procedures, such as urinary catheterization, tracheotomy, mechanical breathing, central venous catheterization, and percutaneous endoscopic gastrostomy (PEG), were more prevalent in the case than in the control group ($P = 0.0001$ for each). The case group demonstrated significantly elevated risks for *Acinetobacter* infections, including cerebrovascular and pulmonary illnesses ($P = 0.024$ and $P = 0.033$, respectively). In the case group, it was determined that beta-lactam/beta-lactamase inhibitor-aminoglycoside combina-

tions and carbapenem group antibiotics were used more. Table 1 presents the comparison of the cases and controls.

The independent risk factors were identified as a younger average age, mechanical ventilator use, tracheotomy, PEG, carbapenem administration, and nonuse of third-generation cephalosporin (Table 2).

In the univariate statistical study, potential risk factors influencing mortality were incorporated into a multivariate logistic regression model. The study showed that enteral nutrition and the APACHE II score were independent risk factors for mortality (Table 3). The incidence of *Acinetobacter* infection did not substantially affect the mortality rates.

Table 1. Comparison of the case and control groups.

Variate		Case group (n=108)	Control group (n=105)	p
Demographic and laboratory data	Age	59.1±18.3	66.8±16.8	0.002
	Gender	68 male+40 female	55 male+50 female	0.129
	White blood cell count ($x10^3/mm^3$)	13.3±10.2	12.4±10.2	0.52
	Platelet count ($x10^3/mm^3$)	241.6±132.9	210.6±119.3	0.076
	C reactive protein	13.7±10.8	10.0±9.7	0.009
	APACHE II score	23.7±7.3	19.6±7.1	0.0001
	Mortality	44 (%40.7)	32 (%30.5)	0.118
Risk factors	Immunosuppression	29 (%26.9)	30 (%28.6)	0.779
	Malignancy	15 (%13.9)	23 (%21.9)	0.127
	Diabetes mellitus	17 (%15.7)	21 (%20)	0.417
	Cerebrovascular disease	67 (%62)	49 (%46.7)	0.024
	Pulmonary disease	61 (%56.5)	44 (%41.9)	0.033
	Cardiovascular disease	41 (%38)	50 (%47.6)	0.154
	Kidney failure	13 (%12)	17 (%16.2)	0.384
	Nasogastric tube	6 (%5.6)	8 (%7.6)	0.543
	Urinary catheter	104 (%96.3)	79 (%75.2)	0.0001
	Tracheotomy	41 (%38)	5 (%4.8)	0.0001
	Mechanical ventilator use	67 (%62)	19 (%18.1)	0.0001
	Hemodialysis catheter	10 (%9.3)	10 (%9.5)	0.947
	Central venous catheter	70 (%64.8)	35 (%33.3)	0.0001
	PEG	29 (%26.9)	4 (%3.8)	0.0001
Antibiotic use	Carbapenem	49 (%45.4)	11 (%10.5)	0.0001
	Beta lactam/beta lactamase inhibitor	23 (%21.3)	27 (%25.7)	0.447
	Third-generation cephalosporin	3 (%2.8)	35 (%33.3)	0.0001
	Carbapenem and quinolone	5 (%4.6)	1 (%1)	0.212
	Beta-lactam/beta-lactamase inhibitor and aminoglycoside	6 (%5.6)	0 (%0)	0.029
	Beta-lactam/beta-lactamase inhibitor and quinolone	13 (%12)	15 (%14.3)	0.627
Nutrition type	Enteral	54 (%50)	36 (%34.3)	0.02
	Parenteral	54 (%50)	69 (%65.7)	0.02

APACHE II score: Acute Physiology and Chronic Health Evaluation II score; PEG: Percutaneous Endoscopic Gastrostomy.

Table 2. Results of the multivariate logistic regression analysis of the risk factors for hospital-acquired *Acinetobacter* infections.

Variate	Odd's rate	95% confidence interval	p
Age	1.03	1.00–1.05	0.008
Mechanical ventilator use	5.68	2.53–12.72	0.0001
Tracheotomy	5.29	1.64–17.08	0.005
PEG	9.49	2.41–37.27	0.001
Carbapenem administration	6.02	2.42–14.94	0.0001
Nonuse of third-generation cephalosporin	0.11	0.02–0.49	0.004

PEG: Percutaneous Endoscopic Gastrostomy.

Table 3. Results of the multivariate logistic regression analysis of the risk factors for mortality.

Variate	Odd's rate	95% confidence interval	p
Enteral nutrition	2.53	1.33–4.78	0.004
APACHE II score	1.08	1.03–1.13	0.0001

APACHE II score: Acute Physiology and Chronic Health Evaluation II score.

The sensitivity of the recovered *Acinetobacter* strains to different antibiotics was evaluated. According to the evaluation made based on the categories created by Falagas et al., the rate of multidrug resistance (MDR) in Acinetobacter isolates in this study was found to be 94%. The strains exhibited the highest susceptibility to colistin and the highest resistance to ciprofloxacin, ertapenem, piperacillin, and piperacillin-tazobactam. Table 4 presents the antibiotic susceptibility profiles for all the isolates included in the study.

In our study, sensitivity was found to be 100% for colistin and 99.1% for tigecycline in the evaluation made according to the reference MIC breakpoints. Among the aminoglycoside antibiotics (amikacin, netilmicin, tobramycin, gentamicin), the highest sensitivity was found in netilmicin, and the highest resistance was found in amikacin. Genotypic analysis was conducted on 96 isolates, of which 83 were categorized into 24 distinct clusters (Figure 1). The data showed a clustering rate of 86% among the isolates (Figure 1), indicating a high level of clonal similarity among the *A. baumannii* strains obtained in this study. By examining the isolation dates of 24 strains in the P5 cluster with the highest number of isolates in our study, it was determined that this clone had persisted in our hospital for approximately 14 months.

Table 4. Antibiotic susceptibility of the identified *Acinetobacter* strains.

Antibiotic	Susceptible n (%)	Moderately susceptible n (%)	Resistant n (%)
Amikacin	22 (20.4)	7 (6.5)	79 (73.1)
Netilmicin	62 (93.9)	1 (1.5)	3 (4.5)
Tobramycin	44 (86.3)	1 (2)	6 (11.8)
Gentamicin	40 (40.8)	5 (5.1)	53 (54.1)
Ampicillin–sulbactam	36 (33.6)	6 (5.6)	65 (60.7)
Cefepime	6 (5.8)	4 (3.9)	93 (90.3)
Cefoperazone-sulbactam	40 (37.7)	12 (11.3)	54 (50.9)
Cefotaxime	5 (4.8)	-	99 (95.2)
Ceftazidime	7 (6.5)	5 (4.6)	96 (88.9)
Ceftriaxone	3 (3.3)	-	87 (96.7
Ciprofloxacin	8 (7.4)	-	100 (92.6)
Imipenem	12 (11.1)	7 (6.5)	89 (82.4)
Meropenem	10 (9.3)	3 (2.8)	95 (88)
Piperacillin	2 (1,9)	1 (1)	102 (97,1)
Piperacillin–tazobactam	4 (3.7)	3 (2.8)	101 (93.5)
Trimethoprim-sulphamethoxazole	6 (6)	-	94 (94)
Mezlocilline	4 (4.3)	-	96 (95.7)
Tetracycline	8 (8.2)	-	89 (91.8)

DISCUSSION AND CONCLUSION

Genotypic analysis is imperative for understanding the dissemination and evolution of *A. baumannii* in healthcare environments. A study that employed enterobacterial repetitive intergenic consensus-polymerase chain reaction for fingerprint genotypic analysis reported that among 59 *A. baumannii* strains, 51 were categorized into 7 clusters. In contrast, the remaining 8 were identified as distinct strains. The significant genetic similarity among these strains was taken as evidence of cross-contamination among hospitalized patients.[12] One study showed that the analyzed strains showed more than 90% similarity and were grouped into 11 distinct genotypes.[13] In our study, we employed REP–PCR using the DiversiLab system (bioMerieux, France) to examine the clonal relationships among *A. baumannii* isolated from the ICU of our hospital. Using this approach, 24 unique clusters were found among the 96 *A. baumannii* isolates. An 86% clustering rate indicated a high level of clonal proximity among isolates, implying a considerable incidence of cross-contamination among patients. This finding is corroborated by a study conducted in our country in which 66 *A. baumannii* isolates collected over a 14-month period were typed via pulsed-field gel electrophoresis, yielding a clustering rate of 80.3%, aligning with our findings.[14] A particularly significant finding in our investigation was the enduring presence of a particular clone (designated as P5) for almost 14 months in our hospital. In this respect, the findings obtained in our study are similar to domestic and international studies.[13,14]

Acinetobacter species, although capable of inducing hospital infections in any area, mainly cause respiratory tract, urinary tract, and wound infections. A notable increase in the number of hospital-acquired pneumonia cases attributed to *A. baumannii* has been documented at numerous sites.[15,16] In accordance with existing research, our study identified VAP as the predominant type of hospital-acquired infection, with tracheal aspirate cultures representing

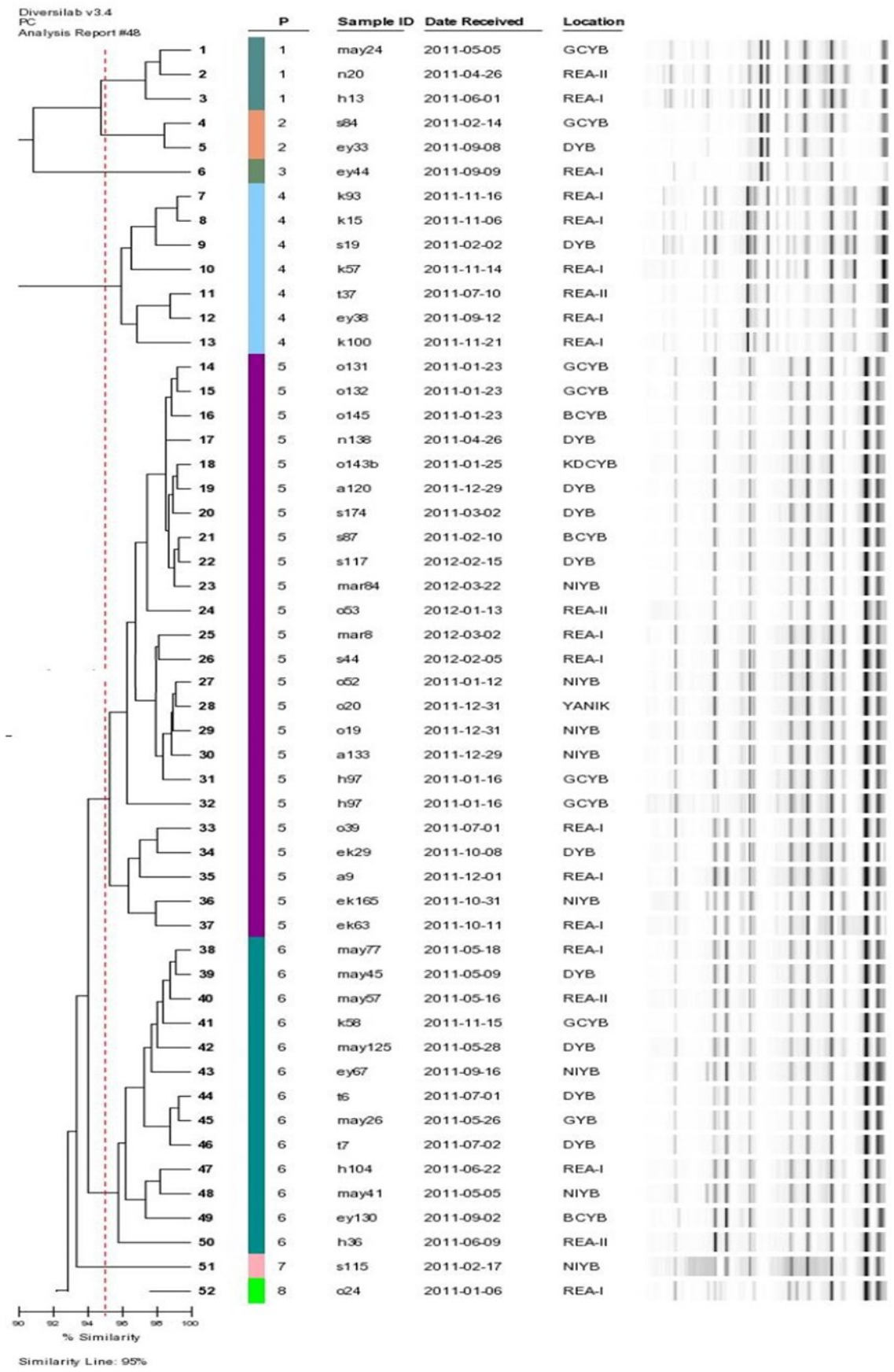

Figure 1. Clustering analysis using a 95% similarity coefficient.

the most prevalent site of *Acinetobacter* proliferation.

Previous research on the risk factors for hospital-acquired *Acinetobacter* infections has identified independent risk factors through multivariate analysis. These factors included male sex, advanced age,[1,16,17] high APACHE II score,[1,18] extended hospital and ICU stay,[18] previous ICU and hospital admissions, enteral nutrition,[1] administration of broad-spectrum antibiotics (particularly third-generation cephalosporins),[18,19] invasive procedures (central venous catheterization, mechanical ventilation, urinary catheterization, surgical interventions),[1] and immunosuppression in patients with critical illness.[20]

Through multivariate regression analysis, this study found age, mechanical ventilator use, tracheotomy, PEG, carbapenem administration, and nonuse of cephalosporins as independent risk factors for hospital-acquired *Acinetobacter* infections. Notably, enteral feeding via PEG was found to increase the incidence of hospital-acquired *Acinetobacter* infections. This is consistent with the finding of a previous study indicating that enteral nutrition is a risk factor for *Acinetobacter* colonization or infection.[1]

Similar to other research,[1,16,17] our study found that age is an independent risk factor for hospital-acquired *Acinetobacter* infections.

Consistent with previous literature,[1] the present study also indicated that interventional procedures are critical risk factors for the aforementioned infections. This study specifically demonstrated that patients who underwent tracheotomy exhibited a greater risk of acquiring *Acinetobacter* infection in the hospital.

Retrospective stepwise elimination logistic regression analysis revealed that enteral nutrition and high APACHE II scores were independent risk factors for mortality. This corresponds to previous studies demonstrating a relationship between high APACHE II scores and an increased risk of acquiring ICU-associated infections.[19]

The use of broad-spectrum antibiotics is an important risk factor for the development of antibiotic resistance and colonization/infection by *Acinetobacter* species.[1,21] Our study demonstrated a markedly increased utilization of beta-lactam/beta-lactamase inhibitor–aminoglycoside combinations and carbapenems in the case group compared with the control group. Multivariate analysis revealed that carbapenem administration and nonuse of cephalosporin are independent risk factors, with the former increasing the probability of *Acinetobacter* infection.

The capacity of *Acinetobacter* to develop antimicrobial resistance and persist on many surfaces enhances its importance as a causal agent of hospital infections. A study found the multidrug resistance to be 63% for the *A. baumannii* isolates and has indicated that the prevalence of carbapenem-resistant *A. baumannii* exceeds 40%.[16] A study reported that a carbapenem-resistant *A. baumannii* strain showed resistance to the majority of the evaluated antimicrobial drugs.[17] In a study involving 2,636 *A. baumannii* isolates, MDR was detected in almost 65% of *A. baumannii* isolates.[22] A study reported resistance rates of 52.2% and 47.8% to meropenem and imipenem, respectively, among *Acinetobacter* strains.[13] Some countries in the South and Southeast Asian regions have resistance rates above 40%, representing the highest frequency among important hospital-acquired Gram-negative infections.[23]

Our analysis revealed that the isolates demonstrated a very high rate of MDR. This finding is consistent with the elevated rates of carbapenem resistance (82.4% for imipenem and 88% for meropenem) identified in our study. Within the aminoglycoside antibiotic category, netilmicin showed the greatest susceptibility, whereas amikacin exhibited significant resistance. In a recent study, the resistance rates for tobramycin, gentamicin, and amikacin were found to be 65.2%, 73.9%, and 52.2%, respectively.[13]

The importance of colistin and tigecycline in the treatment of *Acinetobacter* infections has substantially increased. Numerous studies have reported the development of *in vivo* resistance to colistin or tigecycline during the treatment of *A. baumannii* infections, frequently resulting in chronic or recurring infections.[24] In contrast, our study demonstrated a tigecycline susceptibility rate of 99.1%.

A study examining tigecycline heteroresistance in *A. baumannii* revealed the presence of a significant proportion of heteroresistant isolates.[25] Resistance to colistin, which is essential in combination therapy for managing MDR *A. baumannii* infections, has become increasingly prevalent. Asia has reported the highest resistance rates to colistin, followed by Europe.[26] Conversely, our study revealed complete susceptibility to colistin. A separate investigation showed that the polypeptides polymyxin B and colistin had the highest efficacy against *A. baumannii*, with susceptibility rates of 82.6% and 73.9%, respectively.[13]

Our study on Acinetobacter infections, which is an important health problem today, will make a significant contribution to the literature as it simultaneously includes research on risk factors, antibiotic resistance profile and genotypic analysis.

The limitation of our study is that it is a single-center study. This may limit the generalizability of our findings. Multicenter studies may yield even more valuable results.

In conclusion, multiple critical risk factors were discovered for *A. baumannii* infections in our study, including advanced age, particular interventional

procedures, and the administration of specific antibiotics, such as carbapenems. Genotypic analysis revealed significant clonal relatedness among the *A. baumannii* isolates, indicating considerable cross-contamination hazards within the hospital setting. Identification of molecular and epidemiological diagnostic markers that will help identify resistant clones and monitor their spread will guide the management of nosocomial multidrug-resistant Acinetobacter infections.

Ethics Committee Approval: Our study was approved by the XX University Ethics Committee (Date: 01.03.2011, decision no: 2011/14). The study was carried out following the principles of the Declaration of Helsinki.

Conflict of Interest: No conflict of interest was declared by the authors.

Author Contributions: Concept–SED, FM; Supervision–SED; Materials–SED, CK, YE; Data Collection and/or Processing–SED; Analysis and/or Interpretation–SED, BO; Writing–SED.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Fournier PE, Richet H. The epidemiology and control of Acinetobacter baumannii in health care facilities. Clinical infectious diseases: an official publication of the Infectious Diseases Society of America. 2006;42(5):692-699. doi:10.1086/500202
2. Centers for Disease Control and Prevention. Antibiotic Resistance Threats in the United States 2019. https://ndc.services.cdc.gov/wp-content/uploads/Antibiotic-Resistance-Threats-in-the-United-States-2019.pdf. Accessed August 7, 2024.
3. Freire MP, de Oliveira Garcia D, Garcia CP, et al. Bloodstream infection caused by extensively drug-resistant Acinetobacter baumannii in cancer patients: high mortality associated with delayed treatment rather than with the degree of neutropenia. Clin Microbiol Infect. 2016;22(4):352-358. doi: 10.1016/j.cmi.2015.12.010
4. Ren J, Li X, Wang L, Liu M, Zheng K, Wang Y. Risk Factors and Drug Resistance of the MDR Acinetobacter Baumannii in Pneumonia Patients in ICU. Open Med (Wars). 2019;14:772-777. doi:10.1515/med-2019-0090
5. Falah F, Shokoohizadeh L, Adabi M. Molecular identification and genotyping of Acinetobacter baumannii isolated from burn patients by PCR and ERIC-PCR. Scars Burn Heal. 2019;5:2059513119831369. doi:10.1177/2059513119831369
6. Akavipat P, Thinkhamrop J, Thinkhamrop B, Sriraj W. Acute physiology and chronic health evaluation (APACHE) II score-the clinical predictor in neurosurgical intensive care unit. Acta Clin Croat. 2019;58(1):50-56. doi10.20471/acc.2019.58.01.07
7. Djuric O, Markovic-Denic L, Jovanovic B, Bumbasirevic V. Agreement between CDC/NHSN surveillance definitions and ECDC criteria in diagnosis of healthcare-associated infections in Serbian trauma patients. PLoS One. 2018;13(10):e0204893. doi:10.1371/journal.pone.0204893
8. Humphries R, Bobenchik AM, Hindler JA, Schuetz AN. Overview of changes to the clinical and laboratory standards institute performance standards for antimicrobial susceptibility testing, M100, 31st edition. J Clin Microbiol. 2021;59(12):e0021321. doi:10.1128/JCM.00213-21
9. Falagas ME, Kasiakou SK. Colistin: the revival of polymyxins for the management of multidrug-resistant gram-negative bacterial infections. Clin Infect Dis. 2005;40(9):1333-1341. doi:10.1086/429323
10. Accessdata.fda.gov. Tygacil (tigecycline) i.v. İnjection Label 2010. https://www.accessdata.fda.gov/drugsatfda_docs/label/2010/021821s021lbl.pdf. Accessed June 26, 2024.
11. Falagas ME, Koletsi PK, Bliziotis IA. The diversity of definitions of multidrug-resistant (MDR) and pandrug-resistant (PDR) Acinetobacter baumannii and Pseudomonas aeruginosa. J Med Microbiol. 2006;55(12):1619-1629. doi:10.1099/jmm.0.46747-0
12. Aljindan R, Alsamman K, Elhadi N. ERIC-PCR genotyping of Acinetobacter baumannii isolated from different clinical specimens. Saudi J Med Med Sci. 2018;6(1):13-17. doi:10.4103/sjmms.sjmms_138_16
13. El-Kazzaz W, Metwally L, Yahia R, Al-Harbi N, El-Taher A, Hetta HF. Antibiogram, prevalence of OXA carbapenemase encoding genes, and rapd-genotyping of multidrug-resistant Acinetobacter baumannii incriminated in hidden community-acquired infections. Antibiotics (Basel). 2020;9(9):603. doi:10.3390/antibiotics9090603
14. Cetin ES, Durmaz R, Tetik T, et al. Epidemiologic characterization of nosocomial Acinetobacter baumannii infections in a Turkish university hospital by pulsed-field gel electrophoresis. Am J Infect Control. 2009;37(1):56-64. doi:10.1016/j.ajic.2008.01.010
15. Zarrilli R, Bagattini M, Migliaccio A, Esposito EP, Triassi M. Molecular epidemiology of carbapenem-resistant Acinetobacter baumannii in Italy. Ann Ig. 2021;33(5):401-409. doi:10.7416/ai.2020.2395.
16. Chilam J, Argimón S, Limas MT, et al. Philippi-

nes antimicrobial resistance surveillance program. genomic surveillance of Acinetobacter baumannii in the Philippines, 2013-2014. Western Pac Surveill Response J. 2021;12(4):1-15. doi:10.5365/wpsar.2021.12.4.863
17. Alnimr A, Alamri A, Alsultan A. Genetic diversity of imipenem-resistant Acinetobacter baumannii infections at an intensive care unit. Crit Care Res Pract. 2020;2020(1):3290316. doi:10.1155/2020/3290316
18. Alrahmany D, Omar AF, Alreesi A, Harb G, Ghazi IM. Acinetobacter baumannii infection-related mortality in hospitalized patients: risk factors and potential targets for clinical and antimicrobial stewardship interventions. Antibiotics (Basel). 2022;11(8):1086. doi:10.3390/antibiotics11081086
19. Kumar A, Chaudhry D, Goel N, Tanwar S. Epidemiology of intensive care unit-acquired infections in a tertiary care hospital of North India. Indian J Crit Care Med. 2021;25(12):1427-1433. doi:10.5005/jp-journals-10071-24058
20. Bisaro F, Shuman HA, Feldman MF, Gebhardt MJ, Pukatzki S. Acinetobacter baumannii ATCC 17978 encodes a microcin system with antimicrobial properties for contact-independent competition. Microbiology (Reading). 2023;169(6):001346. doi:10.1099/mic.0.001346
21. Kurihara MNL, Sales RO, Silva KED, Maciel WG, Simionatto S. Multidrug-resistant Acinetobacter baumannii outbreaks: a global problem in healthcare settings. Rev Soc Bras Med Trop. 2020;53:e20200248. doi:10.1590/0037-8682-0248-2020
22. Liu L, Liu B, Li W. Successful incidences of controlling multidrug-resistant, extensively drug-resistant, and nosocomial infection Acinetobacter baumannii using antibiotic stewardship, infection control programs, and environmental cleaning at a Chinese university hospital. infect drug resist. 2020;13:2557-2570. doi:10.2147/IDR.S260525
23. Hsu LY, Apisarnthanarak A, Khan E, Suwantarat N, Ghafur A, Tambyah PA. Carbapenem-resistant Acinetobacter baumannii and enterobacteriaceae in south and Southeast Asia. Clin Microbiol Rev. 2017;30(1):1-22. doi:10.1128/CMR.masthead.30-1
24. Karakonstantis S. A systematic review of implications, mechanisms, and stability of in vivo emergent resistance to colistin and tigecycline in Acinetobacter baumannii. J Chemother. 2021;33(1):1-11. doi:10.1080/1120009X.2020.1794393
25. Jo J, Ko KS. Tigecycline heteroresistance and resistance mechanism in clinical isolates of Acinetobacter baumannii. Microbiol Spectr. 2021;9(2):e0101021. doi:10.1128/Spectrum.01010-21
26. Cai Y, Chai D, Wang R, Liang B, Bai N. Colistin resistance of Acinetobacter baumannii: clinical reports, mechanisms and antimicrobial strategies. J Antimicrob Chemother. 2012;67(7):1607-1615. doi:10.1093/jac/dks084