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Clinical Predictors of Mortality in *Achromobacter* spp. Infections: A 10-Year Retrospective Analysis

Achromobacter spp. Enfeksiyonlarında Mortalitenin Klinik Belirleyicileri: 10 Yıllık Retrospektif Bir Analiz

Dumlu and Mert. Mortality Predictors in *Achromobacter* spp. Infections

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Abstract
Introduction: *Achromobacter* infections are increasingly recognized among high-risk patient populations; however, data regarding factors associated with mortality remain limited. This study aimed to evaluate the clinical, microbiological, and treatment-related predictors of 30-day mortality in adult patients with *Achromobacter* spp. infections.
Materials and Methods: This retrospective, single-center study included adult patients (≥18 years) diagnosed with *Achromobacter* species infections between 2016 and 2025. Demographic characteristics, comorbidities, clinical severity parameters, microbiological findings, and treatment-related variables were analyzed. Univariable and multivariable logistic regression analyses were performed to identify independent predictors of 30-day all-cause mortality.
Results: A total of 218 patients were included in the analysis, and the 30-day mortality rate was 18.3%. In the multivariable analysis, a higher Charlson comorbidity index score, the presence of septic shock, and delayed initiation of targeted antimicrobial therapy (≥72 hours) were independently associated with increased mortality. Age and the use of immunosuppressive therapy were excluded from the multivariable model because of collinearity with comorbidity burden. Carbapenem resistance was not associated with mortality in either the univariable or multivariable analyses.
Conclusion: Mortality in *Achromobacter* spp. infections appears to be primarily driven by host vulnerability, disease severity, and the timing of targeted antimicrobial therapy rather than antimicrobial resistance alone. Early risk stratification and prompt initiation of appropriate targeted therapy are therefore critical for improving clinical outcomes in this high-risk population.
Keywords: *Achromobacter*, mortality, comorbidity, septic shock, delayed antimicrobial therapy

Özet
Amaç: *Achromobacter* enfeksiyonları yüksek riskli hasta popülasyonlarında giderek daha sık tanımlanmaktadır; ancak mortalite ile ilişkili faktörlere dair veriler sınırlıdır. Bu çalışmada, erişkin hastalarda *Achromobacter* spp. enfeksiyonlarında 30 günlük mortalitenin klinik, mikrobiyolojik ve tedaviye ilişkin belirleyicilerinin değerlendirilmesi amaçlanmıştır.
Gereç ve Yöntem: Bu retrospektif, tek merkezli çalışmaya 2016–2025 yılları arasında *Achromobacter* türleri (spp.) enfeksiyonu tanısı alan ≥18 yaş erişkin hastalar dahil edilmiştir. Demografik özellikler, komorbiditeler, klinik şiddet parametreleri, mikrobiyolojik bulgular ve tedaviye ilişkin değişkenler analiz edilmiştir. Otuz günlük tüm nedenlere bağlı mortalitenin bağımsız belirleyicilerini saptamak amacıyla tek değişkenli ve çok değişkenli lojistik regresyon analizleri yapılmıştır.
Bulgular: Toplam 218 hasta çalışmaya dahil edilmiş olup 30 günlük mortalite oranı %18,3 olarak saptanmıştır. Çok değişkenli analizde, daha yüksek Charlson Komorbidite İndeksi skoru, septik şok varlığı ve etkene yönelik antimikrobiyal tedavinin gecikmiş başlanması (≥72 saat) mortalite ile bağımsız olarak ilişkili bulunmuştur. Yaş ve immünsüpresif tedavi kullanımı, komorbidite yükü ile kollineerite göstermesi nedeniyle çok değişkenli modele dahil edilmemiştir. Karbapenem direnci, ne tek değişkenli ne de çok değişkenli analizlerde mortalite ile ilişkili bulunmamıştır.
Sonuç: Achromobacter spp. enfeksiyonlarında mortalite, antimikrobiyal dirençten ziyade konak kırılganlığı, enfeksiyonun klinik şiddeti ve etkene yönelik tedavinin zamanlaması ile belirlenmektedir. Yüksek riskli bu hasta grubunda erken risk sınıflaması ve uygun hedefe yönelik tedavinin zamanında başlanması klinik sonuçların iyileştirilmesinde kritik öneme sahiptir.
Anahtar Kelimeler: *Achromobacter*, mortalite, komorbidite, septik şok, gecikmiş antimikrobiyal tedavi

Introduction
Achromobacter species (spp.), particularly *Achromobacter xylosoxidans*, are environmental, nonfermenting Gram-negative bacilli that have been increasingly isolated in recent years, especially among high-risk populations such as patients with cystic fibrosis and immunocompromised hosts[1-3]. Although initially regarded as opportunistic pathogens, *Achromobacter* spp. are now recognized as clinically significant causes of both nosocomial and community-acquired infections. This shift in recognition has been facilitated by improved diagnostic capabilities and increasing clinical awareness[1,4,5]. These organisms are most frequently associated with respiratory tract infections, bloodstream infections, and catheter-related infections[6]. Their pathogenic potential is supported by biofilm formation and multiple antimicrobial resistance mechanisms, including metallo-β-lactamase production,

porin loss, and efflux pump activation[3,7,8]. Collectively, these resistance determinants substantially limit therapeutic options and complicate clinical management[8].

Despite their increasing clinical relevance, studies systematically evaluating prognostic determinants in *Achromobacter* infections remain limited. Most available publications consist of descriptive case series that focus primarily on clinical and microbiological characteristics rather than on the independent analysis of mortality predictors[4,6]. However, in Gram-negative bloodstream infections, identifying epidemiological patterns and prognostic determinants is essential for optimizing clinical management and improving patient outcomes[9]. In Türkiye, the epidemiological characteristics of *Achromobacter* infections and the factors independently associated with mortality have not yet been adequately characterized. Therefore, this study aimed to evaluate the clinical, microbiological, and treatment-related factors associated with 30-day all-cause mortality among adult patients (≥18 years) diagnosed with *Achromobacter* spp. infections at a tertiary care center. In particular, the independent effects of host vulnerability, infection severity, antimicrobial resistance profiles, and the timing of targeted antimicrobial therapy on mortality were systematically assessed. We hypothesized that these factors would contribute differentially and independently to mortality risk.

Materials and Methods

This study was designed as a retrospective, single-center observational investigation. Adult patients (≥18 years) who were followed at a tertiary care university hospital between January 1, 2016, and December 31, 2025, and who had *Achromobacter* spp. isolated from microbiological cultures in the presence of clinical findings consistent with infection were included. Study data were retrospectively retrieved from the hospital information management system and microbiology laboratory records.

Ethics committee approval for this study was obtained from the İstanbul Medipol University Clinical Research Ethics Committee (decision number: 82, date: 08.01.2026). The study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice guidelines. Because of the retrospective and observational design of the study, the requirement for informed consent from patients or their legal representatives was waived by the ethics committee. No intervention affecting patient safety was performed during the study period. All data were anonymized and analyzed in accordance with confidentiality principles.

Only the first episode of *Achromobacter* spp. infection was included for each patient, and recurrent infections in the same patient were excluded from the analysis. Cases classified as polymicrobial infections or colonization were also excluded. This approach was adopted to ensure a homogeneous study population and to better reflect the true clinical course of infection and its impact on mortality.

Collected data included demographic characteristics (age, sex, and body mass index) as well as underlying clinical conditions. Clinical variables included heart failure, chronic obstructive pulmonary disease, diabetes mellitus, chronic kidney disease, neurological and rheumatological disorders, solid and hematological malignancies, a history of solid organ or hematopoietic stem cell transplantation, use of immunosuppressive therapy, neutropenia, and the presence of cystic fibrosis.

Immunosuppressive therapy was defined as the use of immunosuppressive medications following solid organ or hematopoietic stem cell transplantation; receipt of chemotherapy, targeted therapy, or biological agents for active malignancy; or systemic corticosteroid therapy administered for ≥14 days at a dose of ≥20 mg/day of prednisone or its equivalent[10]. Neutropenia was defined as an absolute neutrophil count of <500 cells/mm³ or a count between 500 and 1,000 cells/mm³ with a declining trend[11]. Comorbidity burden was assessed using the Charlson comorbidity index (CCI) and was included in the analyses as a continuous variable[12].

The clinical severity of infection was determined by the patient's admission setting (intensive care unit or ward) and the presence of sepsis or septic shock, as defined by Sepsis-3 criteria[13]. To quantitatively assess infection severity, the previously described Pitt bacteremia score (PBS) was calculated[14]. The use of invasive devices, including central venous catheters, urinary catheters, and other devices, was also recorded.

Sources and foci of infection were classified according to standard diagnostic criteria established by the Centers for Disease Control and Prevention[15]. Analyses also included the presence of bacteremia and the need for source control.

Identification of isolates from clinical specimens was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS; Bruker Daltonics, Bremen, Germany). Antimicrobial susceptibility testing was conducted using the BD Phoenix™ automated microbiology system (BD Diagnostics, Sparks, MD, USA), and results were interpreted according to the European Committee on Antimicrobial Susceptibility Testing (EUCAST) criteria applicable during the study period[16]. Based on EUCAST-recommended clinically interpretable breakpoints for *Achromobacter* spp., susceptibility analyses were limited to trimethoprim–sulfamethoxazole, piperacillin–tazobactam, and meropenem. Carbapenem resistance was defined according to meropenem resistance per EUCAST interpretive criteria valid during the study period. Multidrug resistance was defined as resistance to at least three antimicrobial classes, following the criteria of Magiorakos et al.[17].

Empirical therapy was defined as antibiotic treatment initiated before microbiological results were available, and its appropriateness was assessed based on the *in vitro* susceptibility of the isolate. Targeted therapy was defined as treatment adjusted according to antimicrobial susceptibility results. Time to initiation of targeted therapy was calculated from the collection of the first positive culture to the start of an appropriate antibiotic. A duration of ≥72 hours was classified as delayed targeted therapy. The ≥72-h cut-off was determined in accordance with routine microbiological processing times, reflecting the expected interval required to obtain definitive susceptibility results and allowing differentiation between early and delayed therapy initiation.

The primary outcome was defined as 30-day all-cause mortality following *Achromobacter* spp. isolation. For patients who died, the day of death was recorded; survivors were followed for 30 days.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 29.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Continuous variables with a nonnormal distribution were expressed as median and interquartile range, whereas categorical variables were presented as counts and percentages (%).

Comparisons between groups were performed using the Mann–Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical variables. To identify factors associated with 30-day mortality, univariable logistic regression analyses were initially conducted.

The multivariable logistic regression model was constructed based on clinical relevance and assessment of collinearity rather than solely on univariable p-value thresholds. Variables considered clinically meaningful and not exhibiting significant collinearity were included in the multivariable analysis. Collinearity was evaluated using variance inflation factor analysis and correlation matrix assessment. Variables demonstrating clinically meaningful overlap and statistical collinearity were not entered simultaneously into the final model. Specifically, age and use of immunosuppressive therapy were excluded due to collinearity with the CCI. The final multivariable model comprised four variables. The events-per-variable ratio was 10 (40 events for 4 covariates), consistent with conventional recommendations for logistic regression model stability. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test.

Results are reported as odds ratios (ORs), adjusted ORs, 95% confidence intervals (CIs), and p values. A two-sided p-value <0.05 was considered statistically significant.

Results

A total of 218 adult patients diagnosed with *Achromobacter* spp. infection were included in the study. Thirty-day mortality occurred in 18.3% of patients (n=40), whereas 81.7% (n=178) survived during the follow-up period. Patients with and without 30-day mortality were compared with respect to demographic characteristics, comorbidity burden, clinical severity of infection, microbiological findings, and treatment-related variables.

In descriptive analyses, patients who died within 30 days were older and had a significantly higher median CCI score than survivors. Hematological malignancy, a history of hematopoietic stem cell transplantation, use of immunosuppressive therapy, and neutropenia were more frequently observed among patients who died (Table 1).

Admission to the intensive care unit, presence of sepsis or septic shock, requirement for mechanical ventilation, and higher PBS were significantly more frequent in patients who died. Similarly, bacteremia was more common in the mortality group. The distribution of infection sources and foci was comparable between patients with fatal and nonfatal outcomes. The majority of isolates were *A. xylosoxidans*, and antimicrobial resistance patterns were similar across groups.

Empirical antibiotic regimens included fourth-generation cephalosporins (cefepime) in 23.4% (n=51), third-generation cephalosporins in 21.6% (n=47), carbapenems in 21.1% (n=46), piperacillin–tazobactam plus an aminoglycoside in 20.6% (n=45), and piperacillin–tazobactam monotherapy in 13.3% (n=29). After antimicrobial susceptibility results became available, all patients were switched to targeted therapy. The most frequently used agents for targeted therapy were piperacillin–tazobactam (35.3%, n=77), trimethoprim–sulfamethoxazole (33.0%, n=72), and meropenem (31.7%, n=69). Mortality rates did not differ significantly across empirical or targeted therapy subgroups.

In contrast, time to initiation of targeted therapy was significantly longer in patients who died, and delayed targeted therapy (≥72 hours) was more frequently observed in this group. Furthermore, the proportion of patients receiving empirical therapy concordant with in vitro susceptibility results was significantly lower among those who died (Table 2).

In univariable logistic regression analysis, age, CCI, presence of septic shock, use of immunosuppressive therapy, and delayed initiation of targeted therapy (≥72 hours) were associated with 30-day mortality. Carbapenem resistance was not significantly associated with mortality in univariable analysis but was further evaluated in the multivariable model based on clinical relevance.

Following collinearity assessment, age and immunosuppressive therapy were excluded from the multivariable model due to collinearity with CCI. In the final multivariable model, higher CCI score, presence of septic shock, and delayed initiation of targeted therapy (≥72 hours) remained independently associated with 30-day mortality, whereas carbapenem resistance was not (Table 3). The Hosmer–Lemeshow goodness-of-fit test demonstrated satisfactory model calibration ($\chi^2=3.57$, df=8, p=0.894).

Discussion

In this 10-year single-center cohort, 30-day mortality among adult patients with *Achromobacter* spp. infections was independently associated with high comorbidity burden, presence of septic shock, and delayed initiation of targeted antimicrobial therapy. In contrast, carbapenem resistance was not independently associated with mortality. These findings extend beyond the predominantly descriptive nature of the existing literature on *Achromobacter* and address an important gap regarding prognostic determinants.

Recent studies have characterized the clinical and microbiological spectrum of *Achromobacter* infections, demonstrating that these infections predominantly occur in patients with significant underlying comorbidities[18-20]. Although mortality rates have been reported, independent predictors of mortality have not been systematically evaluated using multivariable models. Consequently, prognostic determinants in *Achromobacter* infections remain insufficiently defined. Our study addresses this limitation by identifying independent variables associated with mortality.

The independent association between higher CCI and mortality is consistent with previous observations that *Achromobacter* infections are more frequent in patients with substantial comorbid conditions[18-20]. However, prior reports have not systematically quantified the independent impact of cumulative comorbidity burden on mortality. In the broader literature on Gram-negative bloodstream infections, elevated CCI scores have been shown to independently increase mortality risk[21]. Our findings suggest that, in *Achromobacter* infections, prognosis is influenced less by isolated risk factors and more by cumulative host vulnerability and reduced physiological reserve. These data support the concept that mortality is shaped by overall host frailty rather than a single underlying disease.

Septic shock also emerged as a strong independent predictor of mortality. Large multicenter intensive care studies have consistently reported significantly higher mortality among patients with septic shock compared to those without shock[22]. Septic shock represents the clinical manifestation of severe systemic inflammatory dysregulation and organ dysfunction and is a universal driver of adverse outcomes, irrespective of the infecting pathogen. Our results indicate that, once *Achromobacter* infection progresses to hemodynamic instability and organ failure, its mortality dynamics parallel those observed in severe sepsis in general populations.

Delay in targeted antimicrobial therapy of ≥72 hours was independently associated with mortality, representing the most clinically actionable finding of this study. The importance of early and appropriate antimicrobial therapy in sepsis is well established. Delayed initiation of effective therapy in septic shock significantly increases mortality[23], prolonged time to first antimicrobial administration is linked to clinical deterioration and progression to shock[24], and early antibiotic administration reduces hospital mortality in large sepsis cohorts[25]. Our findings indicate that *Achromobacter* infections follow this broader Gram-negative sepsis paradigm, in which timely administration of effective antimicrobial therapy critically influences outcomes.

The absence of an independent association between carbapenem resistance and mortality is noteworthy. Although resistance patterns complicate antimicrobial selection and therapeutic strategy, the resistance phenotype alone does not appear to determine prognosis. This observation suggests that mortality in *Achromobacter* infections is primarily driven by host vulnerability and infection severity rather than resistance classification per se. When effective alternative therapeutic options are available and administered promptly, carbapenem resistance does not independently translate into increased mortality. These findings highlight the importance of clinical risk stratification and timely targeted therapy over reliance on isolated resistance metrics.

This study represents one of the largest single-center cohorts to evaluate independent mortality predictors in *Achromobacter* spp. infections using multivariable analysis. While prior reports have largely been descriptive, our analysis integrates host-related variables, clinical severity parameters, and treatment timing within a structured prognostic framework. The 10-year study period reflects sustained clinical experience and enhances the consistency of findings. The use of validated instruments, such as the CCI and PBS, strengthens methodological rigor. Inclusion of both bloodstream and nonbloodstream infections improves clinical representativeness.

Study Limitations

Several limitations of this study should be acknowledged. The retrospective, single-center design restricts causal inference and limits generalizability. Molecular characterization of resistance mechanisms was not performed, preventing genotype–phenotype correlation analyses. Antimicrobial management was not protocolized and may have been influenced by clinician-dependent variability. Despite multivariable adjustment, residual confounding cannot be entirely excluded. Because delayed targeted therapy is inherently a time-dependent variable, the potential for immortal-time bias or indication bias cannot be fully ruled out. Although time to targeted therapy was calculated from culture collection to initiation of appropriate therapy, early deaths occurring before susceptibility results were available may have affected the observed association. Therefore, residual time-related bias should be considered when interpreting this finding. Further multicenter studies with larger independent cohorts are needed to better define prognostic determinants in *Achromobacter* infections.

Conclusion

This study demonstrates that 30-day mortality in *Achromobacter* spp. infections is primarily influenced by host vulnerability, infection severity, and the timing of targeted antimicrobial therapy, rather than by antimicrobial resistance. The CCI, presence of septic shock, and a ≥72-h delay in targeted therapy emerged as independent predictors of mortality, whereas carbapenem resistance was not associated with mortality. These findings

underscore the critical importance of early risk stratification and timely initiation of appropriate targeted therapy in the management of *Achromobacter* spp. infections.

Ethics

Ethics Committee Approval: Ethics committee approval for this study was obtained from the İstanbul Medipol University Clinical Research Ethics Committee (decision number: 82, date: 08.01.2026). The study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice guidelines.

Informed Consent: Because of the retrospective and observational design of the study, the requirement for informed consent from patients or their legal representatives was waived by the ethics committee.

Footnotes

Authorship Contributions

Surgical and Medical Practices: R.D., A.M., Concept: A.M., Design: R.D., A.M., Data Collection or Processing: R.D., Analysis or Interpretation: R.D., Literature Search: R.D., Writing: R.D., A.M.

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

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Table 1. Demographic characteristics and comorbidities according to 30-day mortality.

Variable	Overall, n=218 (%)[1]	Death n=40 (18.3%)[1]	Survived n=178 (81.7%)[1]	p-value[2]
Patient-related factors				
Age (years) (median, IQR)	61 (58–63)	74 (71–76)	60 (57–62)	<0.001
Male sex	130 (59.6)	23 (57.5)	107 (60.1)	0.90
BMI (kg/m²) (median, IQR)	25.3 (23.9–26.3)	25.3 (23.8–26.3)	25.2 (24.0–26.3)	0.65
Comorbidities				
CCI (median, IQR)	4.5 (3.8–5.1)	4.9 (4.2–5.4)	4.4 (3.6–4.9)	0.003
Heart failure	36 (16.5)	8 (20.0)	28 (15.7)	0.67
COPD	42 (19.3)	4 (10.0)	38 (21.3)	0.15
DM	65 (29.8)	12 (30.0)	53 (29.8)	0.90
CKD	48 (22.0)	8 (20.0)	40 (22.5)	0.90
Neurological disease	31 (14.2)	7 (17.5)	24 (13.5)	0.57
Rheumatologic disease	18 (8.3)	4 (10.0)	14 (7.9)	0.74
Solid organ malignancy	54 (24.8)	8 (20.0)	46 (25.8)	0.57
SOT	22 (10.1)	4 (10.0)	18 (10.1)	0.90
Hematologic malignancy	58 (26.6)	26 (65.0)	32 (18.0)	<0.001
HSCT	26 (11.9)	16 (40.0)	10 (5.6)	<0.001
Immunosuppressive therapy	72 (33.0)	26 (65.0)	46 (25.8)	<0.001
Neutropenia	57 (26.1)	18 (45.0)	39 (21.9)	0.005
Cystic fibrosis	9 (4.1)	1 (2.5)	8 (4.5)	0.69

[1]n (%) and median (interquartile range [IQR] 25–75) values. [2]Pearson's chi-squared test, Wilcoxon rank-sum test, or Fisher's exact test. BMI, body mass index; CCI, Charlson comorbidity index; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; HSCT, hematopoietic stem cell transplant.

Table 2. Clinical severity and microbiological characteristics according to 30-day mortality.

Variable	Overall n=218 (%)[1]	Death n=40 (18.3%)[1]	Survived n=178 (81.7%)[1]	p-value[2]
Clinical characteristics and severity				
ICU admission	101 (46.3)	32 (80.0)	69 (38.8)	<0.001
Presence of sepsis	98 (45.0)	26 (65.0)	72 (40.4)	0.01
Presence of septic shock	52 (23.9)	18 (45.0)	34 (19.1)	<0.001
MV	66 (30.3)	25 (62.5)	41 (23.0)	<0.001
PBS (median, IQR)	3 (2–4)	6 (5–7)	2 (2–3)	<0.001
Invasive device use*	186 (85.3)	36 (90.0)	150 (84.3)	0.42
Presence of Bacteremia	117 (53.7)	30 (75.0)	87 (48.9)	0.005
Origin of infection				
Community-acquired infection	62 (28.4)	9 (22.5)	53 (29.8)	0.41
Healthcare-associated infection	156 (71.6)	31 (77.5)	125 (70.2)	0.28
Source of infection				
Primary BSI	52 (23.9)	14 (46.7)	38 (43.7)	0.78
Pneumonia	26 (11.9)	6 (20.0)	20 (23.0)	0.74
CR-BSI	20 (9.2)	5 (16.7)	15 (17.2)	0.95
Intra-abdominal infection	12 (5.5)	3 (10.0)	9 (10.3)	0.96
SSTI	4 (1.8)	1 (3.3)	3 (3.4)	1.00
UTI	3 (1.4)	1 (3.3)	2 (2.3)	0.75
Microbiological characteristics				
Achromobacter xylosoxidans	168 (77.1)	31 (77.5)	137 (77.0)	0.95
Non-*xylosoxidans* spp.	50 (22.9)	9 (22.5)	41 (23.0)	0.95
Meropenem resistance	69 (31.7)	14 (35.0)	55 (30.9)	0.42
Piperacillin–tazobactam resistance	61 (28.0)	13 (32.5)	48 (27.0)	0.49
Trimethoprim–sulfamethoxazole resistance	58 (26.6)	11 (27.5)	47 (26.4)	0.88
Treatment-related variables				
Source control was performed.	25 (11.5)	4 (10.0)	21 (11.8)	0.89
Appropriate empirical therapy	62 (28.4)	5 (12.5)	57 (32.0)	0.01
Targeted therapy delay (hours) (median, IQR)	44 (36–52)	96 (84–110)	38 (32–44)	<0.001
Targeted therapy delay ≥72 hours	62 (28.4)	21 (52.5)	41 (23.0)	<0.001

[1]n (%) and median (IQR 25–75) values. [2]Pearson's chi-squared test, Wilcoxon rank-sum test, or Fisher's exact test. *Central venous catheter, urinary catheter, and/or percutaneous endoscopic gastrostomy. ICU, intensive care unit; MV, mechanical ventilation; PBS, Pitt bacteremia score (range 0–14); CR-BSI, catheter-related bloodstream infection; SSTI, skin and soft tissue infection; UTI, urinary tract infection; IQR, interquartile range.

Table 3. Univariable and multivariable logistic regression analyses of factors associated with 30-day mortality.

Variable	Univariable OR (95% CI)[1]	p-value[2]	Multivariable aOR (95% CI)[1]	p-value[2]
Age (per year)	1.08 (1.04–1.12)	<0.001	—	—
CCI (per point)	1.74 (1.21–2.51)	0.003	1.75 (1.16–2.66)	0.008

Septic shock	5.01 (2.43–10.32)	<0.001	5.10 (2.32–11.24)	<0.001
Immunosuppressive therapy	5.10 (2.40–10.80)	<0.001	—	—
≥72 h delay in targeted therapy	3.69 (1.81–7.53)	<0.001	4.63 (2.07–10.32)	<0.001
Carbapenem resistance	1.21 (0.59–2.46)	0.32	1.18 (0.51–2.71)	0.46

[1]ORs and aORs with 95% CIs. [2]Univariable and multivariable logistic regression analyses. Variables with clinical relevance in univariable analysis were included in the multivariable model. Collinearity was assessed prior to model construction.
aOR, adjusted odds ratio; CI, confidence interval; CCI, Charlson comorbidity index; OR, odds ratio.