

# Treatment strategies and mortality risk factors in patients with multidrug-resistant *Acinetobacter baumannii* pneumonia: A retrospective analysis.

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**Keywords:** *Acinetobacter baumannii*; Pneumonia; Drug Resistance; Multiple; Risk Factors; Tigecycline.

**Abstract.** This study aimed to investigate the determinants of drug resistance risk factors, 30-day all-cause mortality risk factors, and related clinical treatment strategies in patients with multidrug-resistant *Acinetobacter baumannii* (MDRAB) pneumonia. This retrospective study analyzed data from 168 patients with MDRAB pneumonia and 141 patients with non-MDRAB pneumonia between February 2022 and February 2025. On the second day of admission, the severity of illness and use of carbapenems, tigecycline, etc., were higher in MDRAB pneumonia patients than in non-MDRAB pneumonia patients ( $p < 0.05$ ). The risk factors significantly associated with MDRAB pneumonia included ICU stay prior to AB infection ( $p < 0.001$ ), APACHE II score  $\geq 18$  ( $p = 0.002$ ), invasive procedures ( $p < 0.001$ ), septic shock ( $p = 0.002$ ), and drug abuse ( $p < 0.001$ ). Length of ICU stay before culture, recent surgery, APACHE II score  $\geq 18$ , tigecycline-containing treatment, and the use of two or more antibiotic types (all  $p < 0.05$ ) were significantly linked to 30-day mortality. In a cohort of 168 MDRAB patients, the non-tigecycline treatment group ( $n = 85$ ) showed a significantly lower 30-day mortality rate compared to the tigecycline treatment group ( $n = 83$ ) ( $p = 0.003$ ). Among those receiving tigecycline, the incidence of gastrointestinal adverse reactions was significantly higher, while allergic reactions were less frequent (both  $p < 0.05$ ). In conclusion, prior ICU admission, invasive procedures, and drug abuse are risk factors for developing MDRAB. Severe pneumonia and tigecycline treatment are strongly associated with higher mortality in MDRAB patients, and tigecycline should be used cautiously.

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## **Estrategias de tratamiento y factores de riesgo de mortalidad en pacientes con neumonía por *Acinetobacter baumannii* multirresistente: Un análisis retrospectivo.**

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**Palabras clave:** *Acinetobacter baumannii*; Neumonía; Resistencia a Múltiples Medicamentos; Factores de riesgo; Tigeciclina.

**Resumen.** El objetivo del trabajo fue explorar los factores de riesgo de resistencia a múltiples medicamentos en pacientes con neumonía por *Acinetobacter baumannii* (MDRAB), los factores de riesgo de muerte por todas las causas en 30 días y las estrategias de tratamiento. Este estudio retrospectivo (febrero de 2022-febrero de 2025) analizó datos de 168 pacientes con neumonía por MDRAB y de 141 con neumonía por Non-MDRAB. Al segundo día, los pacientes con neumonía por MDRAB presentaron mayor gravedad y mayor uso de carbapenémicos y tigeciclina que los de Non-MDRAB ( $p < 0,05$ ). Los factores de riesgo significativamente asociados con neumonía por MDRAB incluyeron estancia en UCI previa a la infección por AB ( $p < 0,001$ ), puntuación APACHE II  $\geq 18$  ( $p = 0,002$ ), procedimientos invasivos ( $p < 0,001$ ), shock séptico ( $p = 0,002$ ) y abuso de drogas ( $p < 0,001$ ). La estancia en UCI previa al cultivo, cirugía reciente, puntaje APACHE II  $\geq 18$ , tratamiento con tigeciclina y uso de  $\geq 2$  antibióticos (todos  $p < 0,05$ ) se asociaron significativamente con la mortalidad a 30 días. La cohorte de 168 pacientes con MDRAB mostró una tasa de mortalidad a 30 días significativamente menor en el grupo sin tigeciclina ( $n = 85$ ) que en el grupo con tigeciclina ( $n = 83$ ) ( $p = 0,003$ ). En el tratamiento con tigeciclina, la incidencia de eventos adversos gastrointestinales fue mayor y la de reacciones alérgicas, menor (ambas  $p < 0,05$ ). En conclusión, la admisión previa en UCI, los procedimientos invasivos y el abuso de drogas son factores de riesgo para el desarrollo de MDRAB. La enfermedad grave y el tratamiento con tigeciclina se asocian significativamente con una alta mortalidad en pacientes con MDRAB, por lo que debe usarse con precaución.

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### **INTRODUCTION**

*Acinetobacter baumannii* (*A. baumannii* or AB) is an aerobic, Gram-negative opportunistic pathogen. As a significant pathogen in hospital-acquired infections worldwide, it accounts for approximately 20% of intensive care unit (ICU) infections<sup>1-3</sup>. The bacteria demonstrate strong environmental adaptability, can survive and reproduce across various pH levels and temperatures, and can ad-

here to surfaces of medical equipment such as surgical instruments, ventilators, catheters, and respiratory measuring devices to form biofilms and continue spreading<sup>4,5</sup>. Because *A. baumannii* closely resembles other strains, phenotypic and biochemical classification methods often misidentify it, resulting in an underestimation of its role in nosocomial infections. *A. baumannii* causes various hospital-associated infections, including ventilator-associated pneumonia and

bloodstream infections <sup>6,7</sup>. As a multidrug-resistant pathogen that has garnered global attention, infections caused by *A. baumannii* are associated with higher mortality rates, ranging from 7.8% to 43%. The risk of death is particularly high among ICU patients. It is the second most common Gram-negative pathogen in hospital-acquired pneumonia, responsible for about 3% to 5% of such cases, with associated mortality rates reaching 30% to 75%.

Recently, the overuse of antibiotics, frequent cross-infections among hospitalized patients, and the horizontal transfer of drug-resistant genes have led to the emergence of multidrug-resistant (MDR), extensively drug-resistant (XDR), and even pan-drug-resistant (PDR) strains of *A. baumannii*. This has severely limited options for clinical anti-infective therapy. Globally, the multidrug resistance rate of *A. baumannii* remains high, at approximately 45%. Currently, mainstream strains show extensive resistance to multiple antibiotics, especially carbapenems. In Asia, bacterial susceptibility to carbapenems is even lower than 27%<sup>8,9</sup>. Many guidelines at home and abroad recommend treating pneumonia caused by MDRAB with polymyxin, tigecycline, and sulbactam, combined with other antibiotics. However, regional differences in bacterial sensitivity to specific drugs necessitate the development of individualized drug combination strategies based on local resistance profiles and current guidelines <sup>10</sup>. Polymyxin and tigecycline remain the effective first-line treatments for infections caused by MDR strains. With MDRAB infections, treatment options are extremely limited, mainly relying on a few classes such as polymyxin and tigecycline. Unfortunately, strains resistant to polymyxin are emerging, and tigecycline resistance is also increasing in *A. baumannii*, further intensifying treatment difficulties.

Tigecycline is a broad-spectrum semi-synthetic glycopeptide antibiotic derived from minocycline. The drug has strong in vitro activity against a variety of MDR bac-

teria (e.g., *A. baumannii*), but is ineffective against *P. aeruginosa* and *Shigella* <sup>11</sup>. The structural design of tigecycline enables it to avoid the common tetracycline resistance mechanism, thus showing good application potential in dealing with multi-drug resistant bacteria infection <sup>12</sup>. In general, tigecycline alone is not recommended when other effective antibiotics are available. The combination of tigecycline and sulbactam is one of the common strategies for the treatment of hospital-acquired MDRAB infection. For pulmonary and systemic carbapenem-resistant *A. baumannii* infection, high-dose tigecycline regimen is often given priority because of its high drug concentration in plasma and lung tissue, which shows better efficacy than conventional doses in retrospective studies<sup>13</sup>. Despite demonstrating in vitro susceptibility against MDRAB, the clinical efficacy of tigecycline is controversial. Evidence suggests that its antibacterial activity fails to translate into significant clinical benefit, offering little improvement in patient prognosis <sup>14</sup>.

This study investigated the risk factors for drug resistance and the determinants of 30-day all-cause mortality in patients with MDRAB pneumonia, and related clinical treatment strategies. At the same time, the safety evaluation of the adverse reactions of Radical Antimicrobial Regimens was carried out to provide evidence-based basis for early identification of high-risk patients, optimization of treatment strategies and improvement of prognosis.

## MATERIAL AND METHODS

### Study design

This retrospective, single-center, observational study included 343 patients with AB pneumonia treated at the Third People's Hospital of Yichang City, China, between February 2022 and February 2025. After applying exclusion criteria, 168 patients had MDRAB pneumonia, and 141 had non-MDRAB pneumonia. Their basic characteristics, in vitro

antimicrobial susceptibility testing, treatment strategies on the second day of admission, and survival curves of Radical Antimicrobial Regimens were analyzed. Potential risk factors for drug resistance and 30-day all-cause mortality were evaluated using univariate and multivariate logistic regression analyses. The process flow is shown in Fig. 1.

### Inclusion criteria

(1) diagnosed as pneumonia; (2) The culture of blood and respiratory tract was AB positive; (3) Age greater than 18 years old; (4) Complete clinical data.

### Exclusion criteria

(1) transfer, death, or treatment abandonment within 24 hours of admission; (2) Incomplete clinical data; (3) Lactating women during pregnancy; (4) No clinical manifestations of infection.

### Sample size calculation

The mortality rate of *A. baumannii* in hospital-acquired pneumonia ranges from

30% to 75%<sup>15</sup>. This study assumed that the mortality rate could be reduced by 30% with tigecycline treatment. A bilateral test was used to set the significance level  $\alpha$  at 0.05, with a test power  $(1-\beta)$  of 0.8. After estimating and adjusting the sample size, the minimum total sample size was 142. Overall, 309 patients were included in this study, of whom 83 with MDRAB infection received a comprehensive treatment regimen containing tigecycline, meeting the preset sample size requirements and providing sufficient statistical power for the research conclusions.

### Ethical statement

The study protocol was approved by our hospital's research ethics committee and strictly adhered to the ethical guidelines and norms established by the 'Helsinki Declaration' (latest revision)<sup>16</sup>. It prioritizes the rights, safety, and well-being of the subjects and ensures all research activities follow international ethical standards. We have ef-

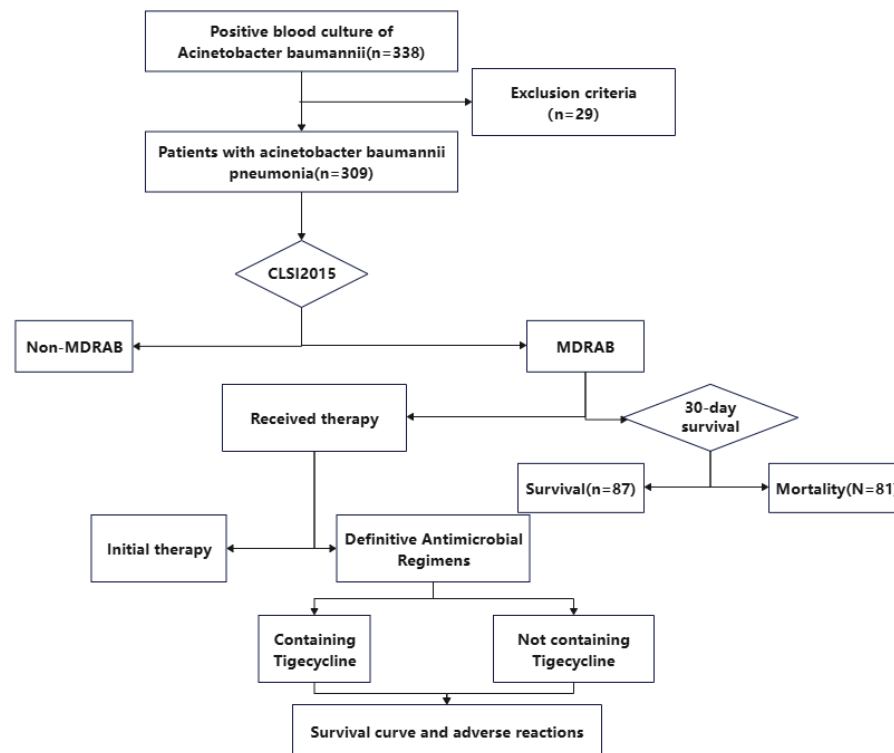


Fig. 1. Case identification flow chart.

fectively protected the legitimate rights and interests of all participants by implementing a thorough informed consent process, safeguarding the privacy and confidentiality of subjects, and adhering to principles of fair benefit and risk reduction.

### Basic information collection

Clinical data of patients with AB pneumonia were retrieved from the Hospital Information System (HIS) database. The collected data include age, gender, hospitalization history, past medical history, surgical history, disease diagnosis, clinical outcomes, antibiotic use history, and microbiological data. Disease severity was assessed at the onset of AB using the SOFA and APACHE II scores. All-cause 30-day mortality following the onset of AB pneumonia was the primary clinical outcome. All antibiotic regimens were administered in accordance with the instructions and clinical medication protocols.

### Antimicrobial susceptibilities

In this study, the VITEK 2 Compact system (bioMérieux, France) and its supporting MALDI-TOF MS mass spectrometry were used to identify the strains of *A.baumannii* isolates. The drug sensitivity test was performed by the VITEK-2 Compact system combined with the AST-GN16 drug sensitivity card (bioMérieux). Tigecycline susceptibility was determined per FDA breakpoints. For all other antibiotics, susceptibility was interpreted according to the latest CLSI guidelines<sup>17,18</sup>.

### Adverse reactions

During the treatment, adverse reactions such as nausea and vomiting, Diarrhea, ototoxicity, allergic reactions, liver injury, and kidney injury were observed and recorded.

### Definitions

(1) Blood and respiratory cultures should be collected within the first 48 hours of hospitalization. AB isolates were categorized as S (sensitive), I (intermediate), or R (resis-

tant). For this analysis, I and R were considered non-sensitive. MDRAB was defined as any AB resistant to at least one of three or more classes of antimicrobial agents. (2) Complications included cerebrovascular disease, liver disease, chronic lung disease, renal failure, etc. (3) The simultaneous administration of two or more different antibacterial agents is called combination antibacterial therapy. (4) Invasive procedures included tracheotomy, catheterization, abdominal puncture, ventilator use, etc.

### Statistical analysis

Continuous and categorical data were expressed as mean  $\pm$  standard deviation and number (percentage), respectively. For comparisons of mean values between groups, the independent t-test was used under the normality assumption, and categorical data were evaluated using the chi-square test. Statistical analysis was conducted using SPSS version 26. GraphPad Prism was used for plotting.  $p < 0.05$  indicated statistical significance of each test. Univariate and multivariate logistic regression models were used to examine associations between independent variables and dichotomous outcomes, including MDR-AB infection and 30-day mortality.

## RESULTS

### Antibiotic resistance characteristics of *Acinetobacter baumannii* pneumonia

The drug-sensitivity test results showed that the AB strain was most sensitive to polymyxin and tigecycline, followed by minocycline, cefoperazone/sulbactam, and levofloxacin. Resistance rates to other antibiotics were more than 45%, particularly carbapenems, and resistance to carbapenems was more than 75%. By comparing the antimicrobial susceptibility spectra, we found significant differences in the susceptibility of carbapenems, cephalosporins, quinolones, aminoglycosides, tigecycline, and combination drugs between the MDRAB and Non-MDRAB groups ( $p < 0.05$ ). In the MDRAB group, in addi-

tion to tigecycline, polymyxin, minocycline, cefoperazone/sulbactam, and levofloxacin (6.20%, 3.52%, 45.53%, 49.43%, and 63.40%, respectively), the resistance rates to other antibiotics exceeded 75% (Table 1).

### Treatment of *Acinetobacter baumannii* pneumonia

By day 2, MDRAB patients exhibited greater disease severity, with higher ICU ad-

mission rates (70.8% vs. 18.4%,  $p < 0.001$ ) and mechanical ventilation rates (54.2% vs. 36.2%,  $p = 0.002$ ). The use of carbapenems, extended-spectrum cephalosporins, aminoglycosides, and tigecycline was more frequent than in the Non-MDRAB group ( $p < 0.05$ ). This indicates that MDRAB pneumonia patients have fewer clinical options, which complicates treatment and increases the risk of side effects (Table 2).

**Table 1.** Antibiotic Resistance characteristics of *Acinetobacter baumannii* pneumonia.

Antimicrobial	Total (N=309)	MDRAB (N=168)	Non-MDRAB (N=141)	$\chi^2$	<i>p</i>
Carbapenem antibiotic					
Meropenem	77.35% (239/309)	91.07% (153/168)	60.99%(86/141)	37.885	<0.001
Imipenem	82.85% (256/309)	95.83% (161/168)	67.38% (95/141)	41.708	<0.001
Cephalosporins					
Cefepime	52.10%(161/309)	91.07%(153/168)	5.67%(8/141)	220.61	<0.001
Ceftriaxone	53.21%(141/265)	97.83%(135/138)	4.27%(6/127)	175.9	<0.001
Ceftazidime	54.50%(109/200)	90.27%(102/113)	8.05%(7/87)	101.93	<0.001
Aminoglycosides					
Gentamicin	53.36% (135/253)	94.20%(130/138)	4%(5/125)	168.992	<0.001
Amikacin	50.19% (133/265)	97.73%(129/132)	3.01% (4/133)	171.457	<0.001
Tobramycin	48.51%(129/268)	84.21%(128/152)	1.72%(2/116)	175.136	<0.001
Quinolones					
Ciprofloxacin	56.30%(152/270)	96.58%(141/156)	8.87%(11/124)	177.794	<0.001
Levofloxacin	35.03%(103/294)	63.40%(97/153)	4.26%(6/141)	99.579	<0.001
Tetracycline					
Tigecycline	3.29%(8/243)	6.20%(8/129)	0%(0/114)	5.133	0.024
Minocycline	25.38%(66/260)	45.53%(56/123)	7.30%(10/137)	31.447	0.011
Others					
Colistin	1.87%(5/268)	3.52%(5/142)	0%(0/126)	5.133	0.092
Combined medication					
Trimethoprim/ sulfamethoxazole	54.58%(149/273)	93.06%(134/144)	11.63%(15/129)	146.218	<0.001
Piperacillin/ tazobactam	54.64%(100/183)	97.96%(96/98)	4.71%(4/85)	103.26	<0.001
Cefoperazone/ sulbactam	26.47%(45/170)	49.43%(43/87)	2.41%(2/83)	35.978	<0.001

Data expressed as % (n). MDRAB: multidrug-resistant *Acinetobacter baumannii*; Non-MDRAB: no multidrug-resistant. Comparisons of mean values between groups, was performed by independent  $\chi^2$  test.

**Table 2.** Treatment of *Acinetobacter baumannii* pneumonia.

Treatment	MDRAB (N=168)	Non-MDRAB (N=141)	$\chi^2$	<i>p</i>
Illness severity measured by day 2				
ICU admission	119(70.83%)	26(18.44%)	84.399	< 0.001
Mechanical ventilation	91(54.17%)	51(36.17%)	9.909	0.002
Antibiotics administered by day 2				
Extended-spectrum cephalosporins	58(34.52%)	29(20.57%)	7.386	0.007
Fluoroquinolones	63(37.5%)	46(32.627%)	0.783	0.376
Carbapenems	97(57.74%)	31(22%)	40.247	< 0.001
Aminoglycosides	42(25%)	16(11.35%)	9.376	0.002
Combined medication	46(27.38%)	20(14.18%)	7.942	0.005
Tetracyclines	14(8.33%)	6(4.26%)	13.341	< 0.001
Polymyxins	7(4.17%)	0(0%)	6.024	0.014

Data expressed as n (%). MDRAB: multidrug-resistant *Acinetobacter baumannii*; Non-MDRAB: no multidrug-resistant. Comparisons of mean values between groups, was performed by independent  $\chi^2$  test.

### Baseline characteristics of *Acinetobacter baumannii* pneumonia

Data on 309 patients with AB pneumonia were collected, and their characteristics were analyzed. Patients with MDRAB pneumonia were older ( $64.72 \pm 10.19$  years vs  $60.72 \pm 10.71$  years,  $p = 0.001$ ), and the proportion of males in both groups exceeded 55%. The incidence of AB infection in hospitals was above 90%, and 82.14% of MDRAB pneumonia patients were diagnosed in the ICU. Complications were common in patients with MDRAB pneumonia, among which hypoproteinemia (76.19%) and septic shock (32.14%) were the most common. The APACHE II and SOFA scores of patients with MDRAB pneumonia were higher than those of patients with Non-MDRAB pneumonia. In addition, alcohol abuse and drug abuse also have a greater impact on patients with MDRAB pneumonia. Detailed data are shown in Table 3.

### Risk Factors for patients with *Acinetobacter baumannii* pneumonia

Univariate analysis showed that age, ICU stay prior to AB infection, hospital stay over 30 days before AB infection, hemodialysis,

immunosuppressive status, APACHE II score of 18 or higher, SOFA score of 10 or higher, invasive procedures, hypoproteinemia, septic shock, alcohol abuse, and drug abuse were associated with MDRAB. After adjusting for confounders, multivariate logistic regression revealed that ICU stay prior to AB infection [ $p < 0.001$ ; OR(95% CI): 17.855 (9.764-32.650)], APACHE II score  $\geq 18$  [ $p = 0.002$ ; OR(95% CI): 4.002 (1.658-9.662)], invasive procedures [ $p < 0.001$ ; OR(95% CI): 5.707 (2.933-11.104)], septic shock [ $p = 0.002$ ; OR(95% CI): 5.059 (1.834-13.956)], and drug abuse [ $p < 0.001$ ; OR(95% CI): 5.092 (2.351-11.024)] were independent risk factors for MDRAB resistance (Table 4).

### Risk factors for death within 30 days in patients with *Acinetobacter baumannii* pneumonia

The 30-day all-cause mortality of 168 MDRAB patients was 48.21% (81). Univariate analysis showed that the length of ICU stay prior to culture, recent surgery, immunocompromised status, endotracheal tube, fiberoptic bronchoscopy, and a SOFA score  $\geq 10$  were associated with culture positivity.

**Table 3.** Baseline characteristics of *Acinetobacter baumannii* pneumonia.

Baseline characteristics	MDRAB (N=168)	Non-MDRAB (N=141)	$\chi^2/t$	<i>p</i>
<b>Age(years)</b>	63.64±10.55	60.92±8.17	2.492	0.013
<b>Gender</b>				
Male	94(56.95%)	82(58.16%)	0.145	0.703
Female	74(44.05%)	59(41.84%)		
<b>Infection</b>				
Nosocomial infection	157(93.45%)	129(91.49%)	0.406	0.524
Community infections	11(6.55%)	12(8.51%)		
<b>Hospital exposure</b>				
ICU stay prior to AB infection	138(82.14%)	31(21.99%)	111.956	<0.001
Hospital stay >30 days prior to AB infection	15(8.93%)	4(2.84%)	4.922	0.027
Operation	64(38.10%)	52(36.88%)	0.048	0.826
Hemodialysis	26(15.48%)	8(5.67%)	7.521	0.006
<b>Illness severity at time of AB</b>				
APACHE II	24.36±8.39	20.31±7.36	4.479	<0.001
SOFA	8.03±5.16	4.11±2.89	7.998	<0.001
Invasive procedures	82(48.81%)	25(17.73%)	32.7	<0.001
<b>Comorbid conditions</b>				
Cerebrovascular diseases	36(21.43%)	27(19.15%)	0.245	0.621
Liver disease	40(23.81%)	31(21.99%)	0.144	0.704
Chronic pulmonary disease	16(9.52%)	14(9.93%)	0.015	0.903
Renal failure	27(16.07%)	16(11.35%)	1.428	0.232
Malignant tumor	32(19.05%)	28(19.86%)	0.032	0.858
Diabetes	28(16.67%)	21(14.89%)	0.181	0.67
Hypoproteinemia	128(76.19%)	89(63.12%)	6.214	0.013
Septic shock	54(32.14%)	11(7.80%)	27.34	<0.001
Immunocompromised status	30(17.86%)	6(4.26%)	13.788	<0.001
Coagulopathy	31(18.45%)	15(10.64%)	3.694	0.055
Paralysis	29(17.26%)	22(15.60%)	0.165	0.684
Gastrointestinal bleeding	20(11.90%)	14(9.93%)	0.305	0.581
<b>Other factors</b>				
Obesity	32(19.05%)	26(18.44%)	0.019	0.891
Weight loss	34(20.24%)	39(27.66%)	2.33	0.127
Fluid and electrolyte disorders	92(54.76%)	79(56.03%)	0.044	0.834
Anemia	64(38.1%)	61(43.26%)	0.85	0.357
Alcohol abuse	28(16.67%)	9(6.38%)	7.723	0.005
Drug abuse	66(39.29%)	18(12.77%)	27.257	<0.001
Mental disorders diseases	31(18.45%)	27(19.15%)	0.024	0.877
Hypertension	102(60.71%)	95(67.38%)	1.451	0.228
<b>Outcome</b>				
30-day mortality	81(48.21%)	10(7.09%)	62.393	<0.001

Data expressed as n (%) or mean ± SD. MDRAB: multidrug-resistant *Acinetobacter baumannii*; Non-MDRAB: no multidrug-resistant. Comparisons of mean values between groups, was performed by independent t-test or  $\chi^2$ .

APACHE II  $\geq 18$  and invasive interventions ( $n > 3$  types) were significantly associated with 30-day mortality. Multivariate logistic regression showed that independent risk factors included length of ICU stay prior to culture [ $p = 0.012$ , OR (95% CI): 0.327 (0.137-0.778)], recent surgery [ $p = 0.001$ , OR (95% CI): 0.063 (0.012-0.338)], APACHE II  $\geq 18$  [ $p < 0.001$ , OR (95% CI): 0.104 (0.034-0.321)], which were significantly associated with 30-day mortality (Table 5).

### The effect of radical antimicrobial regimens on the 30-day mortality of patients with *Acinetobacter baumannii* pneumonia

In this study, 83 MDRAB pneumonia patients received radical treatment containing tigecycline, of which 57 patients (68.67%) died within 30 days. Univariate analysis showed that radical treatment, including tigecycline, polymyxin, and  $\geq 2$  an-

tibiotic agents, was significantly associated with 30-day mortality. Multivariate logistic regression analysis showed that Tigecycline + Other drugs,  $\geq 2$  types of antibiotics, were independent risk factors for 30-day mortality. 168 patients with MDRAB were divided into Non-tigecycline treatment group ( $n = 85$ ), and tigecycline treatment group ( $n = 83$ ). The 30-day mortality of the Non-tigecycline group was lower than that of the tigecycline group ( $p = 0.003$ ) (Table 6 and Fig. 2). This suggests that physicians should select a regimen containing tigecycline to treat MDRAB infection with discretion and prioritize other treatment strategies that may be more effective or safer.

### Adverse reaction of radical antimicrobial regimens

The incidence of gastrointestinal adverse reactions, including nausea and vomiting ( $p = 0.044$ ) and diarrhea ( $p = 0.035$ ),

**Table 4.** Risk factors for death within 30 days in patients with MDRAB.

Baseline characteristics	Univariable Analysis OR (95% CI)	<i>p</i>	Multivariable Analysis OR (95% CI)	<i>p</i>
Age(years)	0.970(0.947-0.994)	0.015		
Hospital exposure				
ICU stay prior to AB infection	16.323(9.314-28.604)	<0.001	17.855(9.764-32.650)	<0.001
Hospital stay >30 days prior to AB infection	3.358(1.088-10.361)	0.035	1.364(0.278-6.683)	0.702
Hemodialysis	3.044(1.331-6.960)	0.008	0.565(0.165-1.931)	0.362
Illness severity at time of AB				
APACHE II $\geq 18$	2.327(1.460-3.708)	<0.001	4.002(1.658-9.662)	0.002
SOFA $\geq 10$	1.619(1.031-2.541)	0.036	0.527(0.2221-1.254)	0.148
Invasive procedures	4.424(2.611-7.498)	<0.001	5.707(2.933-11.104)	<0.001
Comorbid conditions				
Hypoproteinemia	1.870(1.142-3.061)	0.013	1.233(0.733-2.076)	0.43
Septic shock	5.598(2.793-11.222)	<0.001	5.059(1.834-13.956)	0.002
Immunocompromised status	4.891(1.973-12.128)	0.001	1.042(0.238-3.832)	0.951
Other factors				
Alcohol abuse	2.933(1.334-6.449)	0.007	0.737(0.259-2.059)	0.567
Drug abuse	4.422(2.467-7.925)	<0.001	5.092(2.351-11.024)	<0.001

MDRAB: multidrug-resistant *Acinetobacter baumannii*. Data expressed as n (%). OR(95% CI): Odds Ratio (OR) with a 95% Confidence Interval.

**Table 5.** Risk factors for mortality of patients with *Acinetobacter baumannii* pneumonia.

Risk factors	Survival (N=87)	Mortality (N=81)	Univariable Analysis OR (95% CI)	<i>p</i>	Multivariable Analysis OR (95% CI)	<i>p</i>
Age >60 years	51(58.62%)	49(60.49%)	0.833 (0.448-1.550)	0.565		
Male	43(49.43%)	51(62.96%)	0.575 (0.310-1.065)	0.078		
ICU length of stay before AB culture (d)	21(24.14%)	38(46.91%)	0.360 (0.187-0.694)	0.002	0.327 (0.137-0.778)	0.012
Recent surgery (within 1 month)	12(13.79%)	36(44.44%)	0.200 (0.094-0.424)	<0.001	0.063 (0.012-0.338)	0.001
Immunocompromised status	8(9.20%)	22(27.16%)	0.272 (0.113-0.653)	0.004	1.273 (0.322-5.032)	0.731
Endotracheal tube	34(39.08%)	47(58.02%)	0.464 (0.250-0.860)	0.015	0.740 (0.269-2.037)	0.56
Fiberoptic bronchoscopy	25(28.74%)	38(46.91%)	0.448 (0.239-0.839)	0.012	3.872 (0.857-17.507)	0.079
SOFA≥10	39(44.83%)	55(67.90%)	0.367 (0.195-0.688)	0.002	1.579 (0.593-4.206)	0.361
APACHE II≥18	41(51.72%)	39(87.65%)	0.151 (0.069-0.331)	<0.001	0.104 (0.034-0.321)	<0.001
Invasive interventions (n > = 3 types)	11(12.64%)	21(25.93%)	0.414(0.185-0.924)	0.031	0.786(0.280-2.206)	0.647

Data expressed as n (%). OR(95% CI): Odds Ratio (OR) with a 95% Confidence Interval.

**Table 6.** The Effect of Radical Antimicrobial Regimens on the 30-Day mortality of patients with *Acinetobacter baumannii* pneumonia.

Antimicrobial Regimen	Survival (N=87)	Mortality (N=81)	Univariable Analysis OR (95% CI)	<i>p</i>	Multivariable Analysis OR (95% CI)	<i>p</i>
Containing Tigecycline	<b>26(29.89%)</b>	<b>57(70.37%)</b>				
Tigecycline	2(2.30%)	5(6.17%)	0.294 (0.058-1.501)	0.012	0.697 (0.130-3.736)	0.673
Tigecycline+Other drugs	24(27.59%)	52(64.20%)	0.212 (0.110-0.408)	<0.001	0.220 (0.112-0.431)	<0.001
Not containing Tigecycline	<b>61(70.11%)</b>	<b>24(29.63%)</b>				
Polymyxins	2(2.30%)	8(9.88%)	0.215 (0.044-0.215)	0.056	0.225 (0.044-1.153)	0.074
Cefoperazone/sulbactam	32(36.78%)	35(43.21%)	0.765 (0.412-1.420)	0.396	1.579 (0.721-3.459)	0.254
≥2 types of antibiotic	51(58.62%)	62(76.54%)	0.434 (0.223-0.847)	0.014	0.371 (0.166-0.833)	0.016

Data expressed as n (%). OR(95% CI): Odds Ratio (OR) with a 95% Confidence Interval.

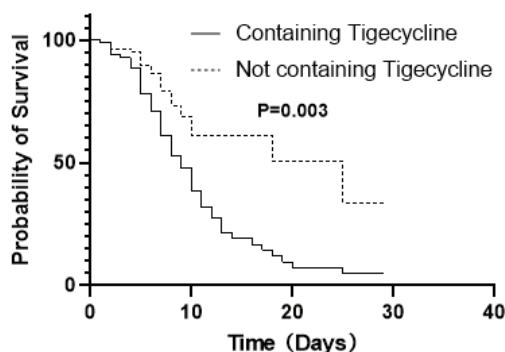


Fig. 2. Kaplan–Meier survival estimates among patients with MDRAB.

MDRAB: multidrug-resistant *Acinetobacter baumannii*.

was significantly higher in regimens containing tigecycline than in regimens without it. In contrast, the occurrence of allergic reactions was lower with the tigecycline treatment ( $p = 0.04$ ). Additionally, there was no significant difference in adverse reactions such as ototoxicity, liver injury, and kidney injury between the two groups. This indicates that clinicians and patients should pay close attention to, and proactively manage, gastrointestinal adverse reactions when using tigecycline in clinical practice. The risk of allergy caused by tigecycline is relatively low, making it a potentially favorable option for patients allergic to other antibiotics (Table 7).

## DISCUSSION

Pneumonia poses a major burden on global health, and its high morbidity and mortality make it one of the main causes of

in-hospital death in hospitalized patients<sup>19</sup>. *A. baumannii* is a notable opportunistic pathogen responsible for severe nosocomial infections, especially in critically ill patients, which often leads to serious complications such as ventilator-associated pneumonia. Due to its significant drug resistance and susceptibility to clonal transmission, the prevention and control of this bacterium have become a major challenge for global health-care systems<sup>20</sup>. Recently, the resistance rate of *A. baumannii* to a variety of commonly used antibiotics, including carbapenems, has continued to rise. Therefore, *A. baumannii* has been listed as a ‘first-class’ key pathogen by the WHO. It is urgent to develop new or more effective antibiotic treatment programs<sup>21</sup>. The emergence of MDRAB has not only increased the morbidity and mortality of patients, but also become one of the core objectives of hospital infection prevention and control, which further highlights the urgent need for effective treatment strategies for MDRAB<sup>22</sup>. Currently, sulbactam-containing combinations, polymyxins, and tigecycline are recommended as standard combination therapies for the treatment of *A. baumannii* infections. However, global reports of drug resistance to these drugs are gradually increasing<sup>23</sup>. Through retrospective analysis, this study summarized the clinical characteristics of patients with AB pneumonia, identified risk factors for MDRAB pneumonia, further evaluated the predictive factors for 30-day mortality in patients with MDRAB

Table 7. Adverse Reaction of Radical Antimicrobial Regimens.

Variables	Containing Tigecycline(n=83)	Not containing Tigecycline(n=85)	$\chi^2$	$p$
Nausea and vomiting	22(26.51%)	11(12.94%)	4.074	0.044
Diarrhea	20(24.10%)	9(10.59%)	4.461	0.035
Allergic reactions	5(6.02%)	14(16.47%)	4.24	0.04
Ototoxicity	2(2.41%)	4(4.71%)	0.149	0.699
Liver injury	7(8.43%)	9(10.59%)	0.045	0.832
renal injury	6(7.23%)	8(9.41%)	0.054	0.816
Other	9(10.84%)	13(15.29%)	0.392	0.531

Data expressed as n (%). Comparison of mean values between groups was performed by the  $\chi^2$  test.

pneumonia, and assessed the efficacy and safety of different eradication regimens. It is expected to provide infection control and clinical care for MDRAB.

The emergence of MDRAB has become a major global healthcare challenge, severely compromising treatment options. Numerous studies have demonstrated this pathogen's resistance to a wide range of antimicrobial agents, including carbapenems, cephalosporins, tigecycline, quinolones, aminoglycosides, and sulbactam-containing compounds<sup>24,25</sup>. Several studies have shown that AB strains are highly sensitive to polymyxins and tigecycline and exhibit high resistance to other antimicrobial agents, particularly carbapenems. In the MDRAB group, except for tigecycline, polymyxin, minocycline, cefoperazone/sulbactam, and levofloxacin, the other antibacterial drugs had higher resistance rates, consistent with the trend observed in this study<sup>15,18,26</sup>. However, it has also been reported that there is a high resistance to myxobacterial polyantibodies<sup>27</sup>, this difference may be related to factors such as the source and type of the selected sample and the underlying disease of the patient. In addition, the severity of disease in patients with MDRAB pneumonia is significantly higher than that in patients with non-MDRAB pneumonia. This further complicates treatment and increases the risk of adverse drug events. Compared with the Non-MDRAB group, the frequency of carbapenem, extended-spectrum cephalosporin, aminoglycoside, and tigecycline use in patients with MDRAB pneumonia was higher, indicating that, under the pressure of multidrug-resistant infections, the clinic has to rely more on these drugs, which have certain toxicities and side effects.

In terms of clinical impact, drug-resistant strains were associated with previous admission to ICU, APACHE II score  $\geq 18$ , invasive procedures, septic shock, and drug abuse. The most common risk factors for obtaining MDRAB included age, previous admission to ICU, length of hospital stay before AB infec-

tion more than 30 days, hemodialysis, immunosuppressive status, APACHE II score  $\geq 18$ , SOFA score  $\geq 10$ , invasive procedures, hypo-proteinemia, septic shock, alcohol abuse, and drug abuse<sup>28-30</sup>, which were basically consistent with the conclusions of this study. This result may be attributed to AB biofilm formation, which enables bacteria to survive in the hospital environment (especially on the surfaces of medical equipment) for extended periods<sup>31</sup>. The occurrence of MDRAB pneumonia is often due to invasive procedures or certain surgeries that disrupt the patient's skin and mucosal barriers, thereby creating a pathway for bacterial invasion<sup>32</sup>. In addition, prolonged hospitalization, ICU admission, and hemodialysis showed that the patients were in a serious condition and their immune function was impaired. These patients further deteriorated after infection with MDRAB, and they had higher APACHE II and SOFA scores. Septic shock and drug abuse are the most common risk factors for MDRAB infection. ICU patients are mostly in a critical state and generally have immunosuppression. If they are combined with septic shock and have received multiple drug treatments, their susceptibility to MDRAB will be further increased. It is worth noting that interactions among these factors may attenuate their independent effects. In addition, other studies have reported additional relevant factors, but are limited by specific conditions; this study does not cover all of them.

The mortality rate of infection caused by MDRAB can be as high as 30% -75%, posing a serious threat to human health<sup>33</sup>. Previous studies have explored a variety of factors as potential predictors of mortality risk in patients with MDRAB pneumonia: such as length of ICU stay before AB culture, recent surgical history, immunocompromised status, tracheal intubation, fiberoptic bronchoscopy, SOFA score, APACHE II score  $\geq 18$  points, and the number of invasive interventions ( $\geq 3$  types)<sup>34-37</sup>, which is consistent with the results of this study. Although tigecycline can reach a high concentration in

multiple tissues (such as the concentration in lung tissue can reach twice that in serum)<sup>38</sup>, this study divided 168 patients with MDRAB into a non-tigecycline group and a tigecycline group according to the treatment method, and found that the 30-day mortality of the former was lower than that of the latter. This result is consistent with many reports that tigecycline-containing treatment regimens are associated with higher AB infection mortality<sup>39-41</sup>.

The limited choice of therapeutic drugs for patients with MDRAB pneumonia not only increases the difficulty of clinical treatment but also increases the risk of drug-related side effects. In the treatment regimen containing tigecycline, the incidence of gastrointestinal adverse events such as nausea, vomiting, and diarrhea was significantly higher than that of the non-tigecycline regimen, which was consistent with the existing research results<sup>42</sup>. In contrast, the incidence of allergic reactions in the tigecycline treatment group was lower. In addition, there was no significant difference in adverse reactions such as ototoxicity, liver injury, and kidney injury between the two groups. The lower allergenicity of tigecycline may make it a better choice for patients allergic to other antibiotics. These findings suggest that tigecycline should be used with caution, and that alternative regimens may yield safer and more effective outcomes.

This study explored risk factors for drug resistance, 30-day all-cause mortality, and associated clinical treatment strategies in patients with MDRAB pneumonia. It provides a new clinical basis for treatment strategies and mortality risk factors in patients with MDRAB pneumonia and has important implications for optimizing clinical prevention and treatment. It is suggested that clinicians should be cautious when prescribing tigecycline for MDRAB pneumonia, as its use may be associated with increased mortality. However, this study also has some limitations. First, because the cases in this study are all from a single center, there are

limitations to the representativeness of the sample. Therefore, the extrapolation of the research conclusions may be limited and should not be directly extended to other regions or populations. Furthermore, the study was a retrospective design. Although it can provide clinical reference to a certain extent, there are still unavoidable information biases and confounding factors, which may affect the accuracy of the results. Finally, a limitation of this study is its small sample size, which may limit statistical power and increase uncertainty in some results. Therefore, the current conclusions should be treated as a preliminary reference only. It is recommended that more prospective, multicenter, large-sample studies be conducted in the future, particularly covering patient groups across different institutions and geographical regions, to further verify the risk factors for MDRAB pneumonia and the effectiveness and safety of treatment strategies. Provide a higher level of evidence-based medical evidence for MDRAB pneumonia, so as to optimize clinical practice and improve the prognosis of patients. At the same time, it is necessary to strengthen research on the molecular epidemiology and mechanisms of drug resistance in MDRAB infection, thereby laying a scientific foundation for the development of new antibacterial drugs and precise treatment strategies. Through various efforts, a systematic and efficient comprehensive prevention and control system was ultimately established to address this serious public health challenge.

In summary, the occurrence of MDRAB pneumonia was closely related to the history of ICU hospitalization, APACHE II score  $\geq 18$ , invasive operation, septic shock, and drug abuse. After the diagnosis of MDRAB pneumonia, Severe and tigecycline treatment were significantly associated with patient mortality. Patients with MDRAB pneumonia should be cautious about using tigecycline when receiving treatment. Although there are some limitations, the results of this study still provide an important reference

for the clinical prevention and treatment of MDRAB pneumonia. In the future, more rigorous methodological research is needed to systematically explore treatment strategies and associated mortality risk factors in MDRAB pneumonia, to further improve clinical prevention and treatment.

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#### Consent to participate

We have effectively protected the legal rights and interests of all participants by carrying out a thorough informed consent process.

#### Ethic approval

The study protocol was approved by the research ethics committee of The Third People's Hospital of Yichang City and strictly adhered to the ethical guidelines and norms established by the 'Helsinki Declaration' (latest revision).

#### Data availability statement

The data supporting the findings of this study are available from the corresponding author upon request.

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LL, WL: Conceived and designed the research, and analyzed data. Drafted and critically revised the manuscript for important intellectual content. GF, WZ: Contributed to data acquisition, analysis, and interpretation. Provided substantial intellectual input during manuscript drafting and revision. GF, WZ: Contributed to the study's conception and design. Played a vital role in data interpretation and manuscript writing. All authors have reviewed and approved the final version of the manuscript.

#### Conflicts of interest

The authors state that they have no financial conflicts of interest.

#### REFERENCES

1. Gauba A, Rahman KM. Evaluation of Antibiotic Resistance Mechanisms in Gram-Negative Bacteria. *Antibiotics* (Basel). 2023;12(11):1590. <https://doi.org/10.3390/antibiotics12111590>.
2. Ibrahim S, Al-Saryi N, Al-Kadmy IMS, Aziz SN. Multidrug-resistant *Acinetobacter baumannii* as an emerging concern in hospitals. *Mol Biol Rep*. 2021;48(10):6987-6998. <https://doi.org/10.1007/s11033-021-06690-6>.
3. Li Y, Xiao S, Huang G. *Acinetobacter baumannii* Bacteriophage: Progress in Isolation, Genome Sequencing, Preclinical Research, and Clinical Application. *Curr Microbiol*. 2023;80(6):199. <https://doi.org/10.1007/s00284-023-03295-x>.
4. Tikku V. *Acinetobacter baumannii*: Virulence Strategies and Host Defense Mechanisms. *DNA Cell Biol*. 2022;41(1):43-48. <https://doi.org/10.1089/dna.2021.0588>.

5. Singh S, Singh S, Trivedi M, Dwivedi M. An insight into MDR *Acinetobacter baumannii* infection and its pathogenesis: Potential therapeutic targets and challenges. *Microb Pathog*. 2024;192:106674. <https://doi.org/10.1016/j.micpath.2024.106674>.
6. Mea HJ, Yong PVC, Wong EH. An overview of *Acinetobacter baumannii* pathogenesis: Motility, adherence and biofilm formation. *Microbiol Res*. 2021;247:126722. <https://doi.org/10.1016/j.micres.2021.126722>.
7. Shi J, Cheng J, Liu S, Zhu Y, Zhu M. *Acinetobacter baumannii*: an evolving and cunning opponent. *Front Microbiol*. 2024;15:1332108. <https://doi.org/10.3389/fmicb.2024.1332108>.
8. Luo Q, Chang M, Lu P, Guo Q, Jiang X, Xiao T, et al. Genomic epidemiology and phylodynamics of *Acinetobacter baumannii* bloodstream isolates in China. *Nat Commun*. 2025, 16: 3536. <https://doi.org/10.1038/s41467-025-58772-9>.
9. Gautam D, Dolma KG, Khandelwal B, Mitsuwan W, Mahboob T, Pereira ML, et al. *Acinetobacter baumannii*: An overview of emerging multidrug-resistant pathogen. *Med J Malaysia*. 2022;77(3):357-370. PMID: 35638493.
10. Marino A, Augello E, Stracquadanio S, Bellanca CM, Cosentino F, Spampinato S, et al. Unveiling the Secrets of *Acinetobacter baumannii*: Resistance, Current Treatments, and Future Innovations. *Int J Mol Sci*. 2024;25(13):6814. <https://doi.org/10.3390/ijms25136814>.
11. Jo J, Ko KS. Tigecycline Heteroresistance and Resistance Mechanism in Clinical Isolates of *Acinetobacter baumannii*. *Microbiol Spectr*. 2021;9(2):e0101021. <https://doi.org/10.1128/Spectrum.01010-21>.
12. Sun C, Yu Y, Hua X. Resistance mechanisms of tigecycline in *Acinetobacter baumannii*. *Front Cell Infect Microbiol*. 2023;13:1141490. <https://doi.org/10.3389/fcimb.2023.1141490>.
13. Bartal C, Rolston KVI, Neshet L. Carbapenem-resistant *Acinetobacter baumannii*: Colonization, Infection and Current Treatment Options. *Infect Dis Ther*. 2022;11(2):683-694. <https://doi.org/10.1007/s40121-022-00597-z>.
14. Deng Y, Chen L, Yue M, Huang X, Yang Y, Yu H. Sulbactam combined with tigecycline improves outcomes in patients with severe multidrug-resistant *Acinetobacter baumannii* pneumonia. *BMC Infect Dis*. 2022;22(1):795. <https://doi.org/10.1186/s12879-022-07778-5>.
15. Al-Tamimi M, Albalawi H, Alkhalalwah M, Alazzam A, Ramadan H, Altalalwah M, et al. Multidrug-Resistant *Acinetobacter baumannii* in Jordan. *Microorganisms*. 2022;10(5):849. <https://doi.org/10.3390/microorganisms10050849>.
16. Wen B, Zhang G, Zhan C, Chen C, Yi H. The 2024 revision of the Declaration of Helsinki: a modern ethical framework for medical research. *Postgrad Med J*. 2025;101(1194):371-382. <https://doi.org/10.1093/postmj/qgae181>.
17. 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. <https://doi.org/10.1128/JCM.00213-21>.
18. Huang H, Chen B, Liu G, Ran J, Lian X, Huang X, et al. A multi-center study on the risk factors of infection caused by multi-drug resistant *Acinetobacter baumannii*. *BMC Infect Dis*. 2018;18(1):11. <https://doi.org/10.1186/s12879-017-2932-5>.
19. Huang SS, Qiu JY, Li SP, Ma YQ, He J, Han LN, et al. Microbial signatures predictive of short-term prognosis in severe pneumonia. *Front Cell Infect Microbiol*. 2024;14:1397717. <https://doi.org/10.3389/fcimb.2024.1397717>.
20. Müller C, Reuter S, Wille J, Xanthopoulou K, Stefanik D, Grundmann H, et al. A global view on carbapenem-resistant *Acinetobacter baumannii*. *mBio*. 2023;14(6):e0226023. <https://doi.org/10.1128/mbio.02260-23>.
21. Jo J, Kwon KT, Ko KS. Multiple heteroresistance to tigecycline and colistin in *Acinetobacter baumannii* isolates and its implica-

- tions for combined antibiotic treatment. *J Biomed Sci.* 2023;30(1):37. <https://doi.org/10.1186/s12929-023-00914-6>.
22. Shein AMS, Hongsing P, Smith OK, Phatharapornjaroen P, Miyanaga K, Cui L, et al. Current and novel therapies for management of *Acinetobacter baumannii*-associated pneumonia. *Crit Rev Microbiol.* 2025;51(3):441-462. <https://doi.org/10.1080/1040841X.2024.2369948>.
  23. Sato Y, Hatayama N, Ubagai T, Tansho-Nagakawa S, Ono Y, Yoshino Y. Tigecycline Suppresses the Virulence Factors of Multi-drug-Resistant *Acinetobacter baumannii* Allowing Human Neutrophils to Act. *Infect Drug Resist.* 2022;15:3357-3368. <https://doi.org/10.2147/IDR.S368890>.
  24. Kyriakidis I, Vasileiou E, Pana ZD, Tragianidis A. *Acinetobacter baumannii* Antibiotic Resistance Mechanisms. *Pathogens.* 2021;10(3):373. <https://doi.org/10.3390/pathogens10030373>.
  25. Tu Q, Pu M, Li Y, Wang Y, Li M, Song L, et al. *Acinetobacter Baumannii* Phages: Past, Present and Future. *Viruses.* 2023;15(3):673. <https://doi.org/10.3390/v15030673>.
  26. AlAmri AM, AlQurayan AM, Sebastian T, AlNimr AM. Molecular Surveillance of Multidrug-Resistant *Acinetobacter baumannii*. *Curr Microbiol.* 2020;77(3):335-342. <https://doi.org/10.1007/s00284-019-01836-z>.
  27. Vrancianu CO, Gheorghe I, Czobor IB, Chifiriuc MC. Antibiotic Resistance Profiles, Molecular Mechanisms and Innovative Treatment Strategies of *Acinetobacter baumannii*. *Microorganisms.* 2020;8(6):935. <https://doi.org/10.3390/microorganisms8060935>.
  28. Reina R, León-Moya C, Garnacho-Montero J. Treatment of *Acinetobacter baumannii* severe infections. *Med Intensiva (Engl Ed).* 2022;46(12):700-710. <https://doi.org/10.1016/j.medine.2022.08.007>.
  29. Diao H, Lu G, Zhang Y, Wang Z, Liu X, Ma Q, et al. Risk factors for multidrug-resistant and extensively drug-resistant *Acinetobacter baumannii* infection of patients admitted in intensive care unit: a systematic review and meta-analysis. *J Hosp Infect.* 2024;149:77-87. <https://doi.org/10.1016/j.jhin.2024.04.013>.
  30. Gharaibeh MH, Abandeh YM, Elnasser ZA, Lafi SQ, Obeidat HM, Khanfar MA. Multi-drug Resistant *Acinetobacter baumannii*: Phenotypic and Genotypic Resistance Profiles and the Associated Risk Factors in Teaching Hospital in Jordan. *J Infect Public Health.* 2024;17(4):543-550. <https://doi.org/10.1016/j.jiph.2024.01.018>.
  31. Roy S, Chowdhury G, Mukhopadhyay AK, Dutta S, Basu S. Convergence of Biofilm Formation and Antibiotic Resistance in *Acinetobacter baumannii* Infection. *Front Med (Lausanne).* 2022;9:793615. <https://doi.org/10.3389/fmed.2022.793615>.
  32. Lan M, Dongmei K, Guodong S, Haifeng Y, Guofeng C, Mengting C, et al. Risk factors for bacteremic pneumonia and mortality (28-day mortality) in patients with *Acinetobacter baumannii* bacteremia. *BMC Infect Dis.* 2024;24(1):448. <https://doi.org/10.1186/s12879-024-09335-8>.
  33. Sharma R, Lakhanpal D. *Acinetobacter baumannii*: A comprehensive review of global epidemiology, clinical implications, host interactions, mechanisms of antimicrobial resistance and mitigation strategies. *Microb Pathog.* 2025;204:107605. <https://doi.org/10.1016/j.micpath.2025.107605>.
  34. Zhou H, Yao Y, Zhu B, Ren D, Yang Q, Fu Y, et al. Risk factors for acquisition and mortality of multidrug-resistant *Acinetobacter baumannii* bacteremia: A retrospective study from a Chinese hospital. *Medicine (Baltimore).* 2019;98(13):e14937. <https://doi.org/10.1097/MD.00000000000014937>.
  35. Černiauskiėnė K, Vitkauskienė A. Multi-drug-Resistant *Acinetobacter baumannii*: Risk Factors for Mortality in a Tertiary Care Teaching Hospital. *Trop Med Infect Dis.* 2025;10(1):15. <https://doi.org/10.3390/tropicalmed10010015>.
  36. Huang C, Gao Y, Lin H, Fan Q, Chen L, Feng Y. Prognostic Factors That Affect Mortality Patients with *Acinetobacter baumannii* Bloodstream Infection. *Infect Drug Resist.* 2024;17:3825-3837. <https://doi.org/10.2147/IDR.S475073>.

37. Yu K, Zeng W, Xu Y, Liao W, Xu W, Zhou T, et al. Bloodstream infections caused by ST2 *Acinetobacter baumannii*: risk factors, antibiotic regimens, and virulence over 6 years period in China. *Antimicrob Resist Infect Control*. 2021;10(1):16. <https://doi.org/10.1186/s13756-020-00876-6>.
38. Zhang S, Di L, Qi Y, Qian X, Wang S. Treatment of infections caused by carbapenem-resistant *Acinetobacter baumannii*. *Front Cell Infect Microbiol*. 2024; 14:1395260. <https://doi.org/10.3389/fcimb.2024.1395260>.
39. Niu T, Xiao T, Guo L, Yu W, Chen Y, Zheng B, et al. Retrospective comparative analysis of risk factors and outcomes in patients with carbapenem-resistant *Acinetobacter baumannii* bloodstream infections: cefoperazone-sulbactam associated with resistance and tigecycline increased the mortality. *Infect Drug Resist*. 2018;11:2021-2030. <https://doi.org/10.2147/IDR.S169432>.
40. Gu S, Xiong J, Peng S, Hu L, Zhu H, Xiao Y, et al. Assessment of Effective Antimicrobial Regimens and Mortality-Related Risk Factors for Bloodstream Infections Caused by Carbapenem-Resistant *Acinetobacter baumannii*. *Infect Drug Resist*. 2023;16:2589-2600. <https://doi.org/10.2147/IDR.S408927>.
41. Mei H, Yang T, Wang J, Wang R, Cai Y. Efficacy and safety of tigecycline in treatment of pneumonia caused by MDR *Acinetobacter baumannii*: a systematic review and meta-analysis. *J Antimicrob Chemother*. 2019;74(12):3423-3431. <https://doi.org/10.1093/jac/dkz337>.
42. Yaghoubi S, Zekiy AO, Krutova M, Gholami M, Kouhsari E, Sholeh M, et al. Tigecycline antibacterial activity, clinical effectiveness, and mechanisms and epidemiology of resistance: narrative review. *Eur J Clin Microbiol Infect Dis*. 2022;41(7):1003-1022. <https://doi.org/10.1007/s10096-020-04121-1>.