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Relato de caso

Septic shock of pulmonary origin due to multidrug-resistant *Acinetobacter baumannii* in an immunocompromised patient: Case report

Choque séptico de origem pulmonar por Acinetobacter baumannii multirresistente em paciente imunocomprometido: relato de caso

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ABSTRACT

Infections caused by *Acinetobacter baumannii* represent a growing challenge for healthcare services, especially in hospital settings, due to their resistance to antibiotics. This report addresses the therapeutic management of a severe infection caused by multidrug-resistant *A. baumannii* in a patient admitted to an intensive care unit (ICU) A 42-year-old man was admitted to the ICU with respiratory distress and fever. Examinations revealed a severe pulmonary infection, associated with other complications. The patient was empirically treated with polymyxin B and fluconazole, and subsequently with meropenem and linezolid. Following initial antimicrobial therapy, cultures were performed confirming the presence of multidrug-resistant *A. baumannii*. The antibiogram revealed resistance to several antibiotics, except for tigecycline. The conduct was adjusted based on culture results. Managing infections caused by multidrug-resistant *A. baumannii* is challenging due to the limited efficacy of available antibiotics. Tigecyclin has shown effectiveness against multidrug-resistant strains, however, closely monitoring treatment response and considering therapeutic changes when necessary is essential.

RESUMO

Infecções causadas por Acinetobacter baumannii representam um desafio crescente para os serviços de saúde, especialmente em ambientes hospitalares, devido à sua resistência aos antibióticos. Este relato aborda a gestão terapêutica de uma infecção grave por A. baumannii multirresistente em um paciente internado em unidade de terapia intensiva. Um homem de 42 anos foi admitido na UTI com desconforto respiratório e febre. Exames revelaram uma infecção pulmonar grave, associada a outras complicações. O paciente foi tratado empiricamente com polimixina B e fluconazol, e, posteriormente, com meropenem e linezolida. Após a terapia inicial, foram realizados cultivos que confirmaram a presença de A. baumannii multirresistente. O antibiograma revelou resistência a vários antibióticos, exceto à tigeciclina. A conduta foi ajustada com base nos resultados dos cultivos. A gestão de infecções por A. baumannii multirresistente é desafiadora devido à limitada eficácia dos antibióticos disponíveis. A tigeciclina demonstrou ser eficaz contra cepas resistentes; no entanto, é essencial monitorar de perto a resposta ao tratamento e considerar mudanças terapêuticas, quando necessário.

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INTRODUCTION

Infections caused by *Acinetobacter baumannii* are becoming an increasing concern for healthcare facilities worldwide and are frequently associated with hospital-acquired infections that result in unfavorable clinical outcomes for patients with prolonged hospital stays. Management of these infections requires rapid identification of the causative strain, isolation of the infection source, and appropriate selection of the antibiotic regimen[1].

A. baumannii is a non-motile, gram-negative coccobacillus, catalase-positive, oxidase-negative, and strict aerobe, classified as a multidrug-resistant bacterium, especially in intensive care units and burn units, making it a significant source of nosocomial infections[2]. Recognized as an important cause of hospital infections and currently considered one of the main contributors to the antibiotic resistance crisis, this bacterium is classified among the ESKAPE pathogens (*Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, A. baumannii, Pseudomonas aeruginosa*, and *Enterobacter spp.*), whose prevalence continues to rise globally[3].

Multidrug-resistant (MDR) *A. baumannii* infections are generally defined by resistance to three or more classes of antibiotics, including quinolones, cephalosporins, β-lactams, aminoglycosides, and carbapenems[2]. Therapeutic options for multidrug-resistant *A. baumannii* are limited, with polymyxins, either as monotherapy or combined with other agents (carbapenems, tigecycline, or rifampicin), being the most commonly used treatments[4].

Tigecycline, a tetracycline-class antibiotic within the glycylcycline group, has demonstrated high in vitro antibacterial activity and is considered a last-resort treatment for multidrug-resistant *A. baumannii* infections[5]. However, clinical isolates of *A. baumannii* resistant to tigecycline have recently emerged[6], posing a significant challenge in clinical management.

Antibiotic resistance has increased due to the acquisition of mobile genetic elements such as transposons, plasmids, and integrons, resulting in the spread of multidrug-resistant strains. The rising resistance to carbapenems, which are used as last-resort antibiotics for treating infections caused by multidrug-resistant gram-negative bacteria, is particularly concerning[7].

Among these, carbapenem-resistant *Acinetobacter baumannii* (CR-Ab) stands out as a global problem since these strains frequently exhibit resistance to nearly all available antibiotics, significantly complicating the treatment of associated infections. Plasmid-mediated resistance has been responsible for outbreaks of extensively drug-resistant strains. This bacterium can be identified by 16S ribosomal RNA, in addition to a conserved region of seven housekeeping genes: *gltA, gyrB, gdhB, recA, cpn60, gpi, and rpoD*, using multilocus sequence typing (MLST) technique[8].

Hospitalized and vulnerable patients are at increased risk of acquiring *A. baumannii* infections due to its ability to penetrate through skin and airway breaches. Most infections caused by this bacterium occur in intensive care unit patients, with ventilator-associated pneumonia being the most common nosocomial infection. The bacterium is capable of causing a variety of infections including skin and soft tissue infections, wound infections, bacteremia, endocarditis, urinary tract infections, meningitis, and pneumonia.

A. baumannii strains can persist in the environment for prolonged periods, contributing to the spread of multidrug-resistant pathogenic strains[7, 9]. The most commonly affected sites include the respiratory tract, surgical wounds, blood, and urine. The majority of infections are associated with trauma or invasive procedures, which can rapidly progress to severe infections such as pneumonia and bacteremia.

Mortality rates associated with *A. baumannii* infections range from 40% to 60%[10],

being higher in cases of ventilator-associated pneumonia (40% to 70%) and bloodstream infections (28% to 43%)[7].

In this study, we describe a case of pulmonary septic shock caused by a multidrug-resistant *Acinetobacter baumannii* bacterial infection in an immunosuppressed patient admitted to an intensive care unit in the Northern Fluminense region. This case report was approved by the Ethics Committee of the Faculdade de Medicina de Campos, under CAAE 81425524.8.0000.5244. The approval number corresponding to this submission is 6975137.

CASE DESCRIPTION

A 42-year-old male patient with a diagnosis of trisomy 21, neuropathy, and a surgical history of hiatoplasty with gastric fundoplication—subsequently resulting in gastrostomy—was admitted to a specialized healthcare facility presenting with respiratory discomfort and reports of febrile episodes. On physical examination, the patient was awake and lucid but exhibited limited interaction with the examiner. Pupils were isocoric and reactive to light. He was hydrated, pale (++/4+), eupneic in room air but with effort, and febrile (38 °C). Cardiovascular examination revealed a regular two-beat cardiac rhythm, no murmurs, blood pressure of 110/80 mmHg, heart rate of 95 bpm, and symmetrical pulses. Respiratory examination showed universally audible vesicular breath sounds with transmitted rhonchi, presence of secretions, and oxygen saturation of 92% on room air. Extremities showed no edema and were well perfused. The abdomen was soft, depressible, non-tender on palpation, and without signs of peritoneal irritation. After admission, complementary exams were performed, including chest computed tomography (**Figure 1**), which demonstrated a small bilateral pleural effusion causing restrictive atelectasis of the adjacent parenchyma, with small consolidation foci in the lower lobes, as well as bronchial thickening with impacted areas in the lower lobes of both lungs. Additionally, a small esophageal hiatal hernia was observed. Given these findings, an upper digestive endoscopy was requested to rule out complications related to the patient's previous gastric surgery. The exam revealed pangastritis and recurrent hiatal hernia, raising the suspicion of a twisted or migrated fundoplication. Furthermore, findings compatible with a urinary tract infection (UTI) were identified through urinalysis, showing massive pyuria and bacteriuria.

The patient was already receiving empirical treatment with polymyxin B and fluconazole, a regimen that was maintained and completed over 14 days due to his recent hospitalization history and risk factors for multidrug-resistant infections, such as trisomy 21—a syndrome associated with immunological dysfunctions, including impaired humoral and cellular responses.

Following assessment, the patient's condition progressed, necessitating intervention and transfer to the intensive care unit, where orotracheal intubation was performed for seven days. Peripheral venous access devices were placed in the left femoral vein, and invasive arterial pressure monitoring was established via the left radial artery. Meropenem (1 g IV every 8 hours) and linezolid (600 mg IV every 12 hours) were added to the treatment after initiation of mechanical ventilation.

Blood cultures were collected (yielding negative results for bacteremia), urine cultures (also negative after antibiotic use), and tracheal secretion cultures (collected one day prior to extubation). The culture results are shown in **Table 1**. The tracheal secretion culture was obtained by aspiration shortly before extubation, based on clinical findings consistent with resolving pulmonary infection. Bronchoalveolar lavage via bronchoscopy was not performed. This limitation, along with the absence of more detailed radiological assess-

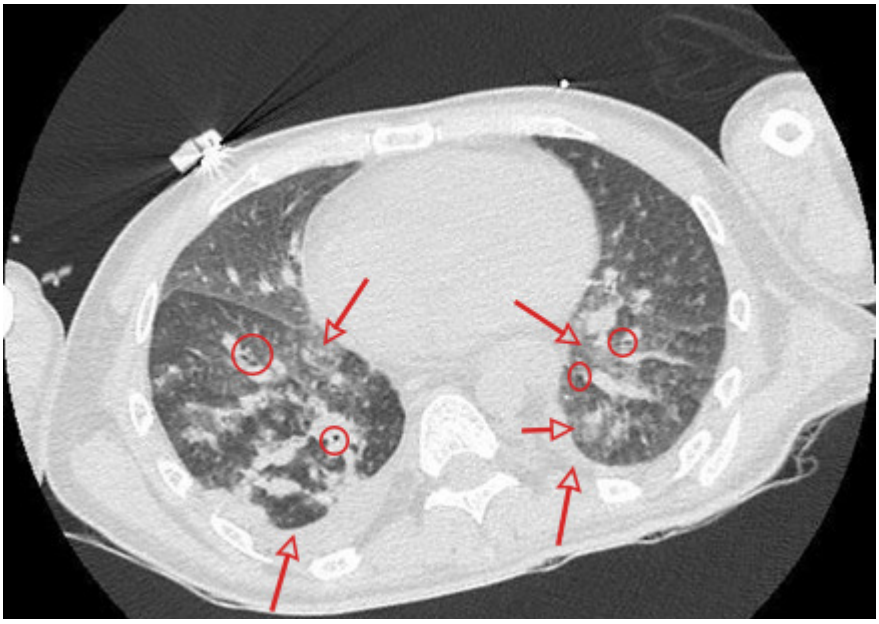

Figure 1. Chest computed tomography: Bilateral pleural effusion with diffuse alveolar infiltrates in the lower lobes, restrictive atelectasis of the adjacent parenchyma, findings suggestive of pneumonia.

ment at the time of sampling, hindered definitive differentiation between active infection and bacterial colonization.

Table 1. Antibiogram results for *A. baumannii* isolated from tracheal secretion culture.

Antibiotic	Susceptibility	MIC (µg/mL)
Amikacin	R	16
Ciprofloxacin	R	>=4
Gentamicin	R	>=16
Meropenem	R	>=16
Piperacillin/ tazobactam	I	>=128
Tigecycline	I	<=0,5

Note: Interpretative criteria—S: susceptible, standard dose; I: susceptible, increased exposure; R: resistant, even with increased exposure; EI: indicates insufficient evidence that the species in question is a suitable target; (–): no breakpoint established.

After extubation, the patient was transferred to the ward, and antibiotic therapy was continued until the scheduled completion date without changes following antibiogram results, as the patient showed progressive clinical improvement, remained afebrile, regained respiratory function, and exhibited increased oxygen saturation.

Supportive care included enteral feeding, dipyrone (2 mL as needed for fever or pain), omeprazole (40 mg IV once daily), quetiapine (50 mg continuously every 12 hours), enoxaparin 40 mg (subcutaneous once daily), motor and respiratory physiotherapy with regular airway suctioning, and contact precautions (red sign).

DISCUSSION

Treatment of multidrug-resistant (MDR) *A. baumannii* strains is challenging due to the limited options available. Generally, the use of antibiotic combinations is considered the most effective approach. However, an analysis of studies has shown that although combination therapy may result in better microorganism eradication, it does not appear to significantly impact mortality rates or length of hospital stay[11]. Nonetheless, it is important to high-

light that the use of multiple antibiotics may increase the risk of antimicrobial resistance development. This creates a clinical dilemma that must be carefully evaluated by each physician on a case-by-case basis.

Sub-minimum inhibitory concentration (sub-MIC) refers to antibiotic levels below the minimum inhibitory concentration (MIC) but still capable of affecting bacteria in some way. Under sub-MIC conditions, bacteria are more likely to develop mutations in response to drug treatment[12]. Additionally, exposure to sub-MIC concentrations of certain antibiotics has been observed to increase bacterial mutation rates, potentially accelerating the development of drug resistance[13].

Due to the rising number of multidrug-resistant superbugs, the need to avoid empirical treatment of infections should be widely disseminated. Although it requires some time, a precise decision-making method involves requesting cultures with antibiotic susceptibility testing and, after analysis of the report, selecting the best drug with appropriate dose and exposure time adjustments. In the case described, the microorganism isolated from the tracheal secretion showed resistance to amikacin, ciprofloxacin, gentamicin, meropenem, and two drugs with low evidence of efficacy (EI), but which, upon evaluation of the epidemiological cutoff, could be considered for use: piperacillin/tazobactam and tigecycline.

According to a technical note produced by the Brazilian Committee on Antimicrobial Susceptibility Testing (BrCAST), the evaluation of the epidemiological cutoff value (ECOFF) for *Acinetobacter* spp. is based on the following criteria: MIC ≤ 0.5 mg/L — the antibiotic may be used with caution depending on the infection site and severity; MIC ≥ 0.5 mg/L — high-dose use may present therapeutic activity but should be applied cautiously in cases of sepsis and pulmonary infections; MIC > 1.0 mg/L — use of the drug is not recommended[14].

Thus, evaluating the patient's antibiogram, piperacillin/tazobactam showed an MIC ≥ 128 and should be excluded as a treatment option. On the other hand, tigecycline presented an MIC ≤ 0.5, indicating its use based on the technical note interpretation.

Tigecycline, a semi-synthetic glycylcycline derivative, functions by inhibiting bacterial protein synthesis through high-affinity binding to the bacterial 30S ribosome[10]. Its efficacy has been demonstrated both in laboratory tests and clinical settings against certain multidrug-resistant *A. baumannii* strains, leading to its approval for patients older than eight years by the European Medicines Agency (EMA). Consequently, tigecycline is widely used to treat infections caused by this microorganism[13]. Similar cases to the one presented have been reported with therapeutic success following bacterial isolation and detailed antibiogram analysis.

A patient with severe pneumonia and multiple risk factors for *Acinetobacter baumannii* infection, such as prolonged use without clinical improvement of multiple broad or ultra-broad spectrum antibiotics (amoxicillin, azithromycin, aztreonam, cefotaxime, and meropenem), invasive procedures (mechanical ventilation support for 14 days), and prolonged ICU stay (up to 20 days), achieved therapeutic success with clinical improvement and culture negativity after extended tigecycline use for five weeks, suggesting that even an antibiotic with intermediate susceptibility, as monotherapy, reached the expected outcomes when dose adjustment and exposure time were optimized[15].

It is important to emphasize that patients with Down syndrome exhibit reduced humoral response, placing them at higher risk for developing autoimmunity, malignancies, and infections that are difficult to manage and resolve. In adults, an aberrant lymphocyte phenotype is commonly observed, characterized by a decrease in naïve/memory T cell ratios and a reduction in switched memory B cells[16].

Analyzing the successful management and antibiogram results, tigecycline emerges as the most promising option for the patient's treatment, especially when combined with meropenem and linezolid already in use, or even considering the possibility of monotherapy and de-escalation of the prescribed antibiotics. However, due to the patient's sensitivity and the complexity of his clinical condition, it was decided to monitor the response to the previously administered medications. While receiving multidisciplinary care in the ward, the patient showed progressive improvement in oxygenation parameters, with an average increase of approximately 2% in oxygen saturation on room air per day during daytime, reaching 96% at the last assessment. He remained afebrile, with reduced respiratory effort and without the use of opioid analgesics (nalbuphine 10 mg prescribed at the physician's discretion).

Although the antibiogram indicated a potentially more effective therapy, the objective was achieved without altering the initial approach. It is important to emphasize, however, the significance of bacterial isolation and therapeutic reassessment in cases of clinical deterioration.

AUTHOR CONTRIBUTION

SOM was responsible for the conception and design of the study, data analysis, and manuscript writing. VHSAR and JBB carried out data collection, statistical analysis, and critical revision of the manuscript. . LAEP and LQH provided technical support, conducted the literature review, and performed the final revision of the text. All authors read and approved the final manuscript version and agree to take responsibility for its content.

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CONFLICT OF INTEREST

We wish to confirm that there are no known conflicts of interest associated with this publication and that no significant financial support has influenced its results.

DECLARATION REGARDING THE USE OF GENERATIVE AI

The authors declare that generative artificial intelligence tools (such as ChatGPT, Grammarly, Deepseek, etc.) were not used in the manuscript. However, the editorial board made the decision to utilize ChatGPT, an AI language model developed by OpenAI, for the translation of this manuscript from the original language, Portuguese, to English.

REFERENCES

1. Nasr P. Genetics, epidemiology, and clinical manifestations of multidrug-resistant Acinetobacter baumannii. J Hosp Infect. janeiro de 2020;104(1):4–11.
2. Lee CR, Lee JH, Park M, Park KS, Bae IK, Kim YB, et al. Biology of Acinetobacter baumannii: Pathogenesis, Antibiotic Resistance Mechanisms, and Prospective Treatment Options. Front Cell Infect Microbiol. 2017;7:55.
3. Patil A, Banerji R, Kanojiya P, Koratkar S, Saroj S. Bacteriophages for ESKAPE: role in pathogenicity and measures of control. Expert Review of Anti-infective Therapy [Internet]. 3 de julho de 2021 [citado 11 de maio de 2025];19(7):845–65. Disponível em: https://www.tandfonline.com/doi/full/10.1080/14787210.2021.1858800
4. Garnacho-Montero J, Timsit JF. Managing Acinetobacter baumannii infections. Current Opinion in Infectious Diseases [Internet]. fevereiro de 2019 [citado 11 de maio de 2025];32(1):69–76. Disponível em: https://journals.lww.com/00001432-201902000-00012
5. Li J, Yang X, Chen L, Duan X, Jiang Z. In Vitro Activity of Various Antibiotics in Combination with Tigecycline Against Acinetobacter baumannii: A Systematic Review and Meta-Analysis. Microb Drug Resist. dezembro de 2017;23(8):982–93.

6. Sun Y, Cai Y, Liu X, Bai N, Liang B, Wang R. The emergence of clinical resistance to tigecycline. Int J Antimicrob Agents. fevereiro de 2013;41(2):110–6.
7. Ibrahim S, Al-Saryi N, Al-Kadmy IMS, Aziz SN. Multidrug-resistant Acinetobacter baumannii as an emerging concern in hospitals. Mol Biol Rep. outubro de 2021;48(10):6987–98.
8. Mulani MS, Kamble EE, Kumkar SN, Tawre MS, Pardesi KR. Emerging Strategies to Combat ESKAPE Pathogens in the Era of Antimicrobial Resistance: A Review. Front Microbiol. 2019;10:539.
9. Al-Kadmy IMS, Ali ANM, Salman IMA, Khazaal SS. Molecular characterization of Acinetobacter baumannii isolated from Iraqi hospital environment. New Microbes New Infect. janeiro de 2018;21:51–7.
10. Rawson TM, Ming D, Ahmad R, Moore LSP, Holmes AH. Antimicrobial use, drug-resistant infections and COVID-19. Nat Rev Microbiol [Internet]. agosto de 2020 [citado 11 de maio de 2025];18(8):409–10. Disponível em: https://www.nature.com/articles/s41579-020-0395-y
11. Mohammadi M, Khayat H, Sayehmiri K, Soroush S, Sayehmiri F, Delfani S, et al. Synergistic Effect of Colistin and Rifampin Against Multidrug Resistant Acinetobacter baumannii: A Systematic Review and Meta-Analysis. TOMICROJ [Internet]. 28 de abril de 2017 [citado 11 de maio de 2025];11(1):63–71. Disponível em: https://openmicrobiologyjournal.com/VOLUME/11/PAGE/63/
12. Andersson DI, Hughes D. Evolution of antibiotic resistance at non-lethal drug concentrations. Drug Resistance Updates [Internet]. junho de 2012 [citado 11 de maio de 2025];15(3):162–72. Disponível em: https://linkinghub.elsevier.com/retrieve/pii/S1368764612000179
13. Liu C, Liu J, Lu Q, Wang P, Zou Q. The Mechanism of Tigecycline Resistance in Acinetobacter baumannii under Sub-Minimal Inhibitory Concentrations of Tigecycline. IJMS [Internet]. 2 de fevereiro de 2024 [citado 11 de maio de 2025];25(3):1819. Disponível em: https://www.mdpi.com/1422-0067/25/3/1819
14. Ministério da Saúde (Brasil). Notas sobre NDM e coprodução de carbapenemase [Internet]. Ministério da Saúde; 2022 [citado 11 de maio de 2025]. Disponível em: https://www.gov.br/anvisa/pt-br/centraisdeconteudo/publicacoes/servicosdesaude/notas-tecnicas/notas-tecnicas-vigentes/nota-tecnica-no-74-2022-cglab-daevs-svs-ms
15. Liu X, Zhou H, Shu S, Li G, Fang F. Successful treatment with tigecycline of severe pandrug-resistant Acinetobacter baumannii pneumonia complicated with a diaphragmatic hernia: a case report. Ann Palliat Med [Internet]. janeiro de 2023 [citado 11 de maio de 2025];12(1):193–9. Disponível em: https://apm.amegroups.com/article/view/104570/html
16. Dieudonné Y, Uring-Lambert B, Jeljeli MM, Gies V, Alembik Y, Korganow AS, et al. Immune Defect in Adults With Down Syndrome: Insights Into a Complex Issue. Front Immunol [Internet]. 8 de maio de 2020 [citado 11 de maio de 2025];11:840. Disponível em: https://www.frontiersin.org/article/10.3389/fimmu.2020.00840/full