

Received: 2026.03.17
Accepted: 2026.07.15
Available online: 2026.07.27
Published: 2026.XX.XX

A 16-Year-Old Male Patient With Severe Electrical Burns With Chronic and Recurrent Hospital-Acquired Opportunistic Infection With Carbapenem-Resistant *Acinetobacter baumannii*

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1,2
CE 2,3
AE 2
D 1
ADE 1
Jiawen Hong
Shengke Wang
Jimei Du
Qiongqian Pan
Jiao Qian

1 Department of Clinical Laboratory, Taizhou Hospital of Zhejiang Province, affiliated with Wenzhou Medical University, Linhai, Zhejiang, PR China
2 Department of Microbiology and Immunology, School of Laboratory Medicine and Life Science, Institute of One Health, Wenzhou Key Laboratory of Sanitary Microbiology, Wenzhou Medical University, Wenzhou, Zhejiang, PR China
3 Department of Transfusion Medicine, Ningbo Yinzhou No. 2 Hospital, Ningbo, Zhejiang, PR China

Corresponding Author: Jiao Qian, Department of Clinical Laboratory, Taizhou Hospital of Zhejiang Province, affiliated to Wenzhou Medical University, 150 Ximen Street, Linhai, 317000, China, Phone: +86 15215814110, e-mail: qianj@enzemed.com
Financial support: None declared
Conflict of interest: None declared

Patient: Male, 16-year-old
Final Diagnosis: Chronic recurrent hospital-acquired opportunistic infection caused by carbapenem-resistant *Acinetobacter baumannii* • severe electrical burns
Symptoms: Gastrointestinal bleeding • hypoxic encephalopathy • pneumonia • respiratory failure • skin lesions
Clinical Procedure: —
Specialty: Critical Care Medicine • Laboratory Diagnostics • Infectious Diseases
Objective: Unusual clinical course
Background: *Acinetobacter baumannii* is an opportunistic gram-negative bacterium found in health facilities, including intensive care units (ICUs), and for patients with burn injuries, it can cause severe wound infections and systemic infection. Electrical burn is a unique type of trauma. Burns and electrical injuries disrupt the integrity of skin and mucous membrane barriers, which results in impairment of the body's defense mechanisms, thus significantly increasing the possibility of infection. This report describes the case of a 16-year-old male patient with severe electrical burns and chronic and recurrent ICU-acquired opportunistic infection with carbapenem-resistant *A. baumannii* (CRAB).
Case Report: A 16-year-old male patient was admitted with 220-V electrical burns and was subsequently transferred to the ICU. On admission, he had impaired consciousness and recurrent fever, complicated by respiratory failure, gastrointestinal bleeding, nasal hemorrhage, hypoxic brain injury, and pulmonary infection. During hospitalization, cutaneous ulceration of the left lower limb, necrosis of the third to fifth toes of the left foot, and skin lesions involving the buttocks, sacrococcygeal region, and occiput developed sequentially. CRAB was successively isolated from sputum, blood, and multiple skin wound specimens. The patient underwent repeated debridement, wound expansion, and limb reconstructive surgery. He was discharged in stable condition after 156 days of hospitalization.
Conclusions: Early identification of CRAB colonization and invasive infection is essential. Strict infection control measures should be implemented in ICUs and burn wards. This case provides clinical reference for the management of recurrent CRAB infection in patients with severe electrical burns and for the prevention and control of nosocomial infection.
Keywords: *Acinetobacter baumannii* • Electric Injuries • Intensive Care Units • Bacterial Infections
Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/953461>

 3489

 2

 2

 17

Publisher's note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher



Introduction

Acinetobacter baumannii is a gram-negative, aerobic, nonmotile coccobacillus. The microorganism is prevalent in nature, occurring in soils and waters. Moreover, *A. baumannii* can be a persistent colonizer of human skin, lungs, and gut mucosa [1]. Desiccation tolerance, resistance to ultraviolet radiation, and resistance to antiseptics enable this microorganism to survive for long periods and spread across hospitals. Human colonization often interferes with the diagnosis of infection and the choice of antibiotics [2]. The pathogen's virulence depends on various virulence factors. Adhesion and invasion of host epithelial cells by *A. baumannii* is possible due to lipopolysaccharides, outer membrane proteins, and capsular polysaccharides [3,4]. In addition, the pathogen evades the immune response through biofilms, quorum-sensing systems, and immune evasion, thereby causing inflammation [5]. Given that *A. baumannii* is one of the most common opportunistic pathogens in healthcare settings, it is a primary cause of ventilator-associated pneumonia, bloodstream infections associated with central venous catheter insertion, and urinary tract infections in intensive care units (ICUs) [6]. An extensive use of carbapenems for clinical treatment is the cause of the growing prevalence of carbapenem-resistant *A. baumannii* (CRAB) worldwide [7]. Being highly drug resistant, CRAB poses a serious public health problem that requires the development of new antimicrobials to treat infections caused by it [8].

Electrical burn is a unique type of trauma. Long-term exposure to low-voltage alternating electric current can not only lead to the formation of burn wounds on the skin but can also induce arrhythmias, and in extreme conditions, even respiratory and cardiac arrest [9]. Burns and electrical injuries disrupt the integrity of skin and mucous membrane barriers, which results in impairment of the body's defense mechanisms, thus significantly increasing the possibility of infection by pathogens. Studies have shown that the prevalence of *A. baumannii* among patients in burn departments is higher than 16% [10]. Another study highlights the high risk of early-onset nosocomial infections in burn patients [11]. In addition, strains isolated from burn units are characterized by multidrug resistance and high genotypic variability [12]. In comparison to traditional wounds, secondary infections that occur as a result of electrical burns pose more of a challenge both clinically and in terms of treatment. This report describes the case of a 16-year-old male patient with severe electrical burns and chronic and recurrent ICU-acquired opportunistic infection with CRAB. We aim to provide information from his experience.

Case Report

A 16-year-old male patient with no previous medical illnesses experienced an electric burn as a result of exposure to

alternating current of 220 V and then fell from an estimated height of 0.5 meters onto his back. About 40 minutes of cardiopulmonary resuscitation (CPR) were administered during the course of transportation to the nearby hospital, and circulation spontaneously returned 2 hours after admission. He was subsequently shifted to our ICU the next day.

Consent Declarations

This study involving a human participant was approved by the institutional ethics committee. The study was conducted in accordance with local legislation and institutional requirements (approval No. KL20250113). Given the retrospective nature of this study, the ethics committee waived the requirement for additional written informed consent. The patient had previously provided informed consent for their clinical imaging examinations as part of routine medical care, and all identifiable personal information was anonymized to protect patient privacy.

Upon admission, a thorough examination and diagnostic testing were done. Physical examination results showed that there was an arrhythmia, a soft abdomen with bowel sounds heard thrice per minute, and non-compliance in the muscle strength test because of the patient's altered mental status. Extracorporeal membrane oxygenation (ECMO) support was initiated. Additional clinical observations included muscle rigidity of the left lower limb, stiffness of the left ankle, mild active nasal bleeding, and aspiration of black fluid during gastrointestinal decompression.

Evaluation of the echocardiogram showed dysfunction of left ventricular systolic function in terms of the global reduction of amplitude of motion in the interventricular septum and the left ventricle's posterior wall. The findings included mild regurgitation of the mitral valve, tricuspid valve, and pulmonary valve with an ejection fraction of 25% and fractional shortening of 11%.

Biochemical analysis yielded the following results: albumin level was 25.5 g/L (reference range: 40.0-55.0 g/L); alanine aminotransferase level was 231 U/L (reference range: 9-50 U/L); aspartate aminotransferase level was 861 U/L (reference range: 15-40 U/L); C-reactive protein (CRP) level was 2.1 mg/L (reference range: <8.0 mg/L); and procalcitonin (PCT) level was 5.93 ng/mL (reference range: <0.05 ng/mL).

Hematological evaluation revealed a white blood cell count of $13.1 \times 10^9/L$ (reference range: $3.9-9.5 \times 10^9/L$); hemoglobin level of 95 g/L (reference range: 130-175 g/L); absolute neutrophil count of $11.79 \times 10^9/L$ (reference range: $1.8-6.3 \times 10^9/L$); prothrombin time of 18.5 seconds (reference range: 11.0-14.5 seconds); activated partial thromboplastin time of 52 seconds (reference range: 28.0-42.0 seconds); and thrombin time exceeding 100 seconds (reference range: 14.0-21.0 seconds).

Admission diagnoses included electrical burn, cardiac arrest requiring CPR, shock, hypoxic encephalopathy, respiratory failure, gastrointestinal bleeding, acute renal insufficiency, mediastinal emphysema, left lower limb ischemia, and cardiac insufficiency.

After admission, the patient was maintained on ECMO, endotracheal intubation, and mechanical ventilation. He was unconscious and had compromised immune function. Acute renal failure developed on the second day of hospitalization, and blood purification therapy was initiated. The patient also presented with gastrointestinal bleeding and mild nasal bleeding. Mottled skin of the left lower limb and partial stiffness of the calf muscles suggested possible tissue necrosis. A large amount of sputum was suctioned through the endotracheal tube. Chest computed tomography (CT) revealed increased and thickened lung markings, indicating pulmonary infection. Intravenous meropenem was administered at 1.0 g every 8 hours for anti-infective treatment.

On hospital day 3, nucleic acid testing for pathogenic bacteria in sputum yielded a weakly positive result for *A. baumannii*. Laboratory analyses revealed troponin I levels exceeding 80.00 ng/mL (reference range: < 0.10 ng/mL), and the PCT level was 887.00 ng/mL (reference range: < 0.05 ng/mL). Subsequent chest CT scans showed patchy shadows with indistinct margins in both lower lung fields, consistent with pulmonary infection. Intravenous meropenem (1.0 g every 8 hours) was continued, supplemented by intravenous vancomycin (initial loading dose 1000 mg, followed by 350 mg every 12 hours).

On hospital day 9, sputum pathogen nucleic acid testing was positive for *A. baumannii* and methicillin-resistant *Staphylococcus aureus*. Intravenous linezolid (600 mg every 12 hours) was therefore initiated.

On hospital day 11, CRAB was isolated from the sputum culture. Susceptibility testing revealed the organism to be sensitive to tigecycline and polymyxin. As a result, the treatment protocol was modified to include the use of a combination of drugs, namely tigecycline (100 mg every 12 hours) along with cefoperazone (2.0 g every 8 hours). Laboratory test results showed elevated inflammatory markers: high-sensitivity CRP was 178.6 mg/L (reference range: < 8.0 mg/L), serum amyloid A protein exceeding 320 mg/L (reference range: 0-10 mg/L), and PCT level was 87.93 ng/mL (reference range: < 0.05 ng/mL).

On hospital day 14, the presence of CRAB was detected in the blood culture. On chest CT, there was evidence of pulmonary infection. Redness, swelling, and significant local hyperthermia were observed on the left lower extremity with skin ulceration and significant exudation on the toes of the left foot. As a result, the treatment strategy was changed to include tigecycline (100 mg every 12 hours) combined with meropenem (0.5 g every 8 hours).

On hospital day 23, the patient was conscious but apathetic. Neuroimaging revealed hypoxic cerebral alterations. In terms of clinical picture, there were episodes of uncontrollable tremors and confusion with complications such as otomastoiditis. There were observed skin ulcerations of the left lower limb, necrosis of the third to fifth toes of the left leg, lesions of the occipital skin, and sacral pressure ulcers. A temperature increase was recorded. CRAB pathogens were found repeatedly in samples collected from lesions of the skin of the left leg, the tip of the left subclavian catheter, and the gluteal area. Due to difficult expectoration and dyspnea, endotracheal intubation was done on day 25, then tracheostomy and bronchoscopy with the aspiration of sputum on day 27. Antibiotic therapy was escalated to tigecycline (75 mg every 12 hours) combined with polymyxin B injection (500 000 units every 12 hours).

On hospital day 37, the skin of the patient's left lower extremity showed dryness with minimal tissue growth. Moist dressings helped remove eschars. The interdigital space of the left foot was cleaned and treated using iodophor gauze. Samples of secretions were taken for culturing. There was an improvement in the mental state of the patient, with stable hemodynamic parameters. No abnormalities in the lungs were found. Oxygen therapy replaced previous ventilation.

On hospital day 42, the patient had recurrent fever, and CRAB was detected in lower-limb secretions. However, at that point, the inflammatory biomarkers were still under control. Hemodynamics revealed stabilization of heart rate and blood pressure, and the levels of both brain natriuretic peptide and troponin were decreased. Magnetic resonance imaging (MRI) of the leg revealed bone infarction, significant edema, and hematoma formation in the muscles of the left calf. Because of a poor response to the previously used antibiotics, surgery became necessary. The patient had the revision of the left toe stump and debridement of the calf. Antibiotic therapy was adjusted to polymyxin B (500 000 units every 12 hours) combined with tigecycline (50 mg every 12 hours).

On hospital day 47, CRAB was detected in left foot secretions, and *Acinetobacter lwoffii* was isolated from aerobic blood cultures. The next day, slight fluid exudation was observed in the buttocks. The patient's temperature was normal, and infection markers were not elevated. Tigecycline was discontinued, replaced by cefoperazone-sulbactam sodium (2 g every 12 hours), which was continued for 2 weeks.

On hospital days 49 and 58, the patient underwent 2 debridement procedures. His temperature remained well controlled, consciousness was clear, his condition was stable, and oxygen was administered via nasal cannula.

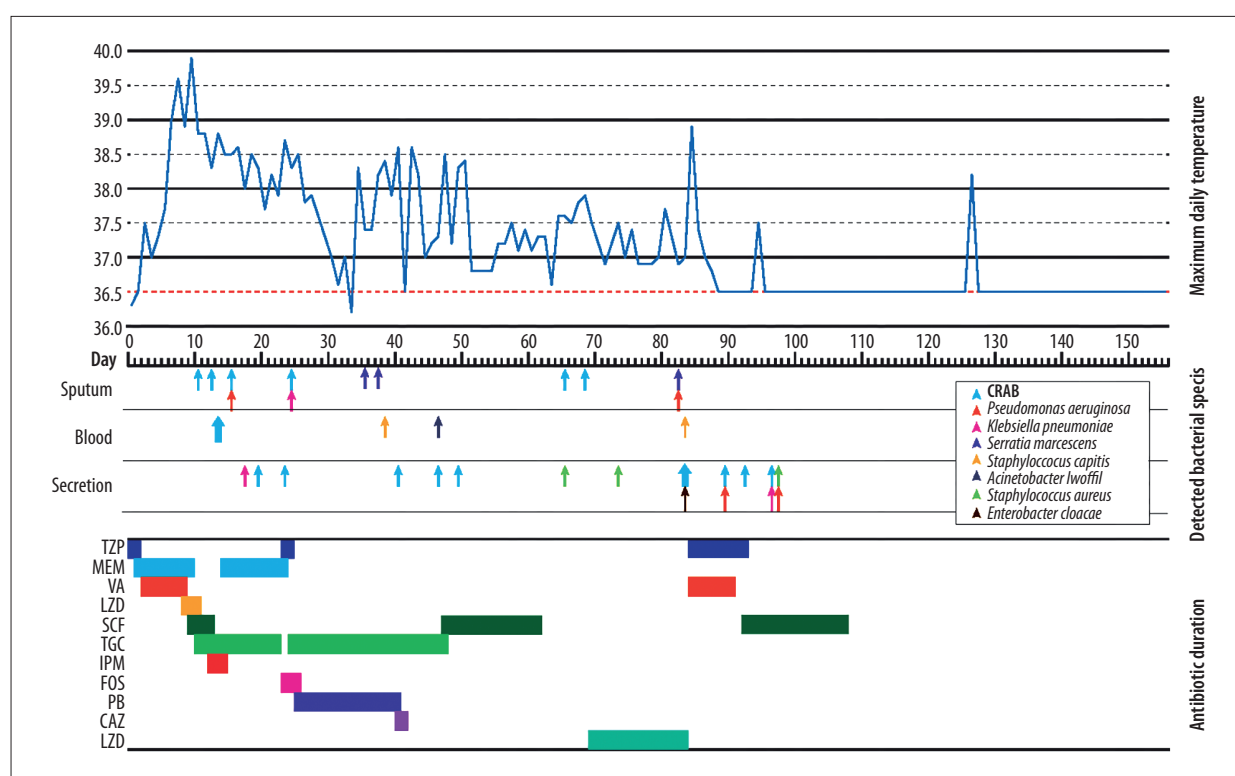


Figure 1. Antibiotic duration, detected bacterial species, and maximum daily temperature. This figure shows the patient's daily maximum body temperature during hospitalization, the sites, names, and timing of pathogen detection, as well as antibiotic usage. Abbreviations: CRAB, carbapenem-resistant *Acinetobacter baumannii*; TGP, piperacillin/tazobactam; MEM, meropenem; VA, vancomycin; LZD, linezolid; SCF, cefoperazone/sulbactam; TGC, tigecycline; IPM, imipenem; FOS, fosfomycin; PB, polymyxin B; CAZ, ceftazidime.

On hospital day 66, CRAB was detected in sputum culture, and *S. aureus* was isolated from buttock secretions. Symptomatic dressing changes were performed without antibiotics.

On hospital day 70, the patient experienced low-grade fever, a rapid heart rate (141 beats per minute), and rapid breathing (approximately 30 breaths per minute). Chest X-ray showed no pulmonary exudation. Due to the previous detection of *S. aureus* in buttock discharge, intravenous linezolid (0.6 grams every 12 hours) was administered for 2 weeks. Nine days later, sacrococcygeal debridement and wound repair surgery were performed.

On hospital day 84, *Klebsiella pneumoniae* was isolated from urine. CRAB and *Enterobacter cloacae* were identified in back secretions, and *Staphylococcus capitis* was detected in blood cultures. The patient's temperature remained normal; linezolid was discontinued. The sacrococcygeal wound decreased in size. Based on blood culture results, intravenous vancomycin (1 g every 12 hours) was administered.

On hospital day 93, partial necrosis of the left lower leg skin, necrosis of the third to fifth toes on the left foot, and a

sacrococcygeal pressure injury were noted. CRAB was isolated from sacrococcygeal secretions. Cefoperazone-sulbactam sodium 1.0 g IV for 2 hours was started, while piperacillin-tazobactam was stopped. After that, the patient was stable. With supportive treatment and repeated surgeries to the limbs, the patient improved and was discharged after 156 days in the hospital.

The antibiotic duration, detected bacterial species, and maximum daily temperature are summarized in a time-series format in **Figure 1**. Chest X-ray findings are presented in **Figure 2**.

All clinical specimens were processed under sterile conditions by professional microbiology personnel according to standardized procedures. After obtaining pure colonies, bacterial identification was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry, confirming the isolated strain as *A. baumannii*. Antimicrobial susceptibility testing was conducted using a fully automated microbial susceptibility analysis system and the Kirby-Bauer disk diffusion method. Minimum inhibitory concentration results were strictly interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines, and *A. baumannii* strains resistant to imipenem or meropenem were classified as CRAB.

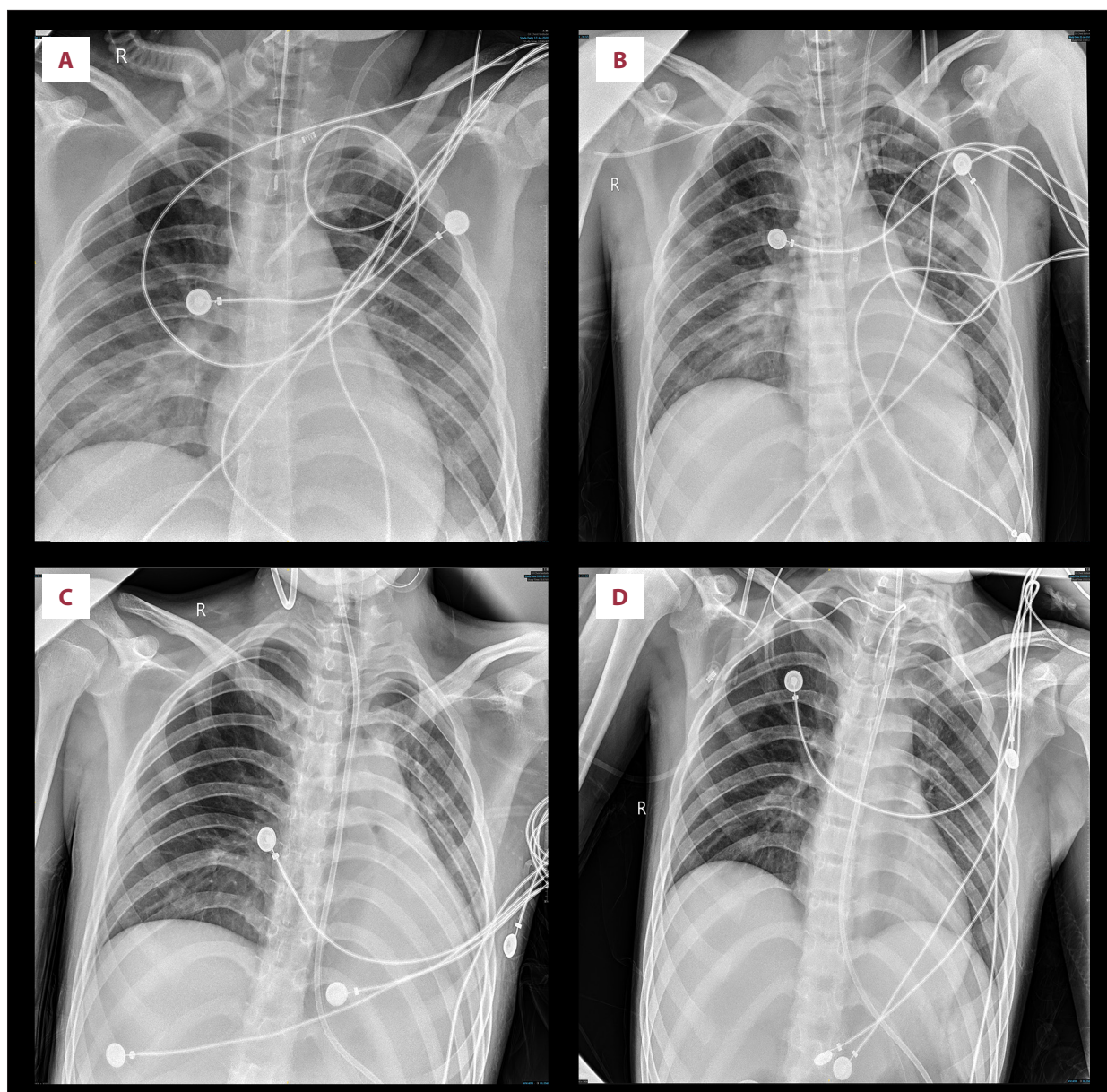


Figure 2. Chest X-ray findings of the patient. Chest radiographs obtained at different time points during hospitalization. On hospital day 3 (A), slightly increased and thickened pulmonary markings with patchy opacities and blurred margins were observed in both lower lung fields. On hospital day 7 (B), a patchy area of increased density with blurred margins was observed in the middle field of the left lung. On hospital day 24 (C), compared with previous findings, patchy opacities in the middle and lower lung fields were partially absorbed. A patchy area of increased density with relatively clear margins was observed in the upper field of the right lung, with local pleural adhesion at the right lung apex. On hospital day 34 (D), pulmonary markings were distinct, with no obvious abnormalities detected.

Since the bacteria, CRAB, were found at a high frequency in the hospitalization period, 2 CRAB isolates (WMTZ1 and WMTZ2) were selected for whole-genome sequencing (WGS) to evaluate their genetic relatedness. Isolate WMTZ1 was found in the blood sample collected on the 14th day after admission, while isolate WMTZ2 was identified in the bronchial fluid sample on the 84th day after admission.

Two purified colonies of *A. baumannii* strains were sent to Suzhou Weiciyuan Biotechnology Co, Ltd for WGS sequencing using the Illumina MiSeq platform, and genome assembly was conducted using SPAdes software. All assembled genomic sequences have been deposited in the China National Center for Bioinformation database under accession number PRJCA055264.

Table 1. Resistance genes and insertion sequences detected in WMTZ1 and WMTZ2.

Functional category	Gene name	WMTZ 1	WMTZ 2
β-lactamases	<i>blaADC - 73</i>	1	1
	<i>blaOXA - 66</i>	1	1
	<i>blaOXA - 23</i>	1	1
	<i>blaTEM - 1</i>	1	0
Efflux pumps	<i>adeC</i>	1	1
	<i>abaF</i>	1	1
Tetracycline resistance	<i>tet(B)</i>	1	1
Aminoglycoside modifying enzymes	<i>aph(6) - Id</i>	1	1
	<i>aph(3'') - Ib</i>	1	1
	<i>ant(3'') - IIa</i>	1	1
	<i>aph(3') - Ia</i>	1	0
	<i>armA</i>	1	1
Macrolide resistance	<i>mph(E)</i>	1	1
	<i>msr(E)</i>	1	1
Sulfonamide resistance	<i>sul2</i>	1	1
Other functions	<i>amvA</i>	1	1
	<i>ISAba22</i>	1	1
	<i>ISVsa3</i>	1	1
	<i>ISAba24</i>	1	1
	<i>ISEc29</i>	1	1
	<i>ISAba26</i>	1	1
	<i>IS26</i>	1	0

Footnote: The number 1 indicates the presence of the gene/insertion sequence, and 0 indicates absence.

The sequencing results identified 135 single-nucleotide polymorphisms (SNPs) between the 2 CRAB isolates, suggesting they were not derived from the same clonal strain. WMTZ1 harbored 2 additional resistance genes, *blaTEM-1* and *aph(3')-Ia*, as well as the resistance-related insertion sequence *IS26*. WMTZ2 lacked these genes, consistent with its antibiotic-susceptibility phenotypes to cefoperazone/sulbactam and aminoglycosides. Resistance genes and insertion sequences detected in WMTZ1 and WMTZ2 are presented in **Table 1**. Drug susceptibility test results of WMTZ1 and WMTZ2 are shown in **Table 2**.

Discussion

This case illustrates that patients with severe electrical burns are susceptible to multisite invasive CRAB infections due to multiple organ damage and impaired immune function. Given the difficulty in distinguishing CRAB colonization from true

infection, continuous monitoring of clinical manifestations and inflammatory biomarkers is essential; meanwhile, we should place greater emphasis on infection control in burn units.

Electrical burns often lead to multi-system trauma and are characterized by high morbidity and mortality rates [13]. Epidemiologic data suggest that electrical burns are the fourth most common cause of death at workplaces, where the number of yearly deaths is 500 to 1000 cases [3]. Electrical burns can lead to considerable tissue destruction and, consequently, to multi-system infection [13]. CRAB is one of the most common opportunistic pathogens in burn ICUs and has a considerable resistance to drying and disinfection techniques [1]. There is limited literature on the clinical features and pathogenesis of CRAB infections developing after electrical trauma.

The patient in this case was a 16-year-old male with no prior underlying disease. He developed cardiac arrest after a 220-V

Table 2. Drug susceptibility test results of WMTZ1 and WMTZ2.

Antibiotic	WMTZ1		WMTZ2	
	MIC	Susceptibility	MIC	Susceptibility
Imipenem	≥16	R	≥16	R
Meropenem	18*	S	≥16	R
Trimethoprim-sulfamethoxazole	≥320	R	≥320	R
Tigecycline	2	I	2	I
Piperacillin/tazobactam	≥128	R	≥128	R
Levofloxacin	≥8	R	≥8	R
Ceftazidime	≥64	R	≥64	R
Cefoperazone/sulbactam	≥64	R	16	S
Cefepime	≥32	R	≥32	R

Abbreviations: MIC, minimum inhibitory concentration; S, susceptible; R, resistant; I, intermediate. The asterisk indicates the result was determined by the Kirby-Bauer method.

electrical injury and was revived by CPR. On admission, cardiac insufficiency, organ damage, shock, and hypoxic brain dysfunction increased the risk of CRAB colonization, invasion, and infection. An immune system disorder caused by the electrical injury further increased the likelihood of CRAB infection. *A. baumannii* can colonize the respiratory tract and skin [14]. In a clinical setting, distinguishing CRAB infection from colonization with a single culture test alone may be difficult. Therefore, early detection of CRAB infection is imperative to provide appropriate antimicrobial therapy in patients with severe electrical burns. The diagnosis of CRAB infection depends on clinical presentation, imaging characteristics, sequential organism isolation, and inflammatory markers. The patient in the present case had recurrent fever after hospital admission. Chest CT showed bilateral pulmonary exudation and increased lung markings, suggesting nosocomial pneumonia. In addition, a significant wound infection was indicated by erythema, edema, increased skin temperature, skin rupture, and massive exudation in the left lower extremity. To identify the causative agents, sputum, wound discharge, the tips of central venous catheters, and blood were collected for culture. Weak positivity for nucleic acid of *A. baumannii* was observed in sputum samples collected on hospital days 3 and 9. CRAB was isolated from sputum cultures on hospital days 11 and 13, confirming CRAB pulmonary infection.

CRAB was isolated from a blood culture collected from the patient on day 13 of admission, indicating hematogenous spread and a CRAB bloodstream infection. Later studies repeatedly isolated CRAB from samples collected from the lower extremities, buttocks, sacrococcygeal area, and sputum. Thus, it was confirmed that the patient had sustained a severe electrical

injury with a multisite invasion of CRAB infection. Continuous monitoring of inflammatory markers revealed persistently and markedly elevated levels of high-sensitivity CRP, serum amyloid A, and PCT, further supporting the diagnosis of severe, drug-resistant bacterial infection.

In this case, prompt culture of the bacteria was key to treating a patient with secondary infections after electrical burn. CRAB and other microbes were repeatedly isolated from the patient's sputum, blood, and wound discharge. According to a 10-year surveillance study of microbial complications in high-voltage electrical burns by Váňa et al, infection of the burn wound was the most common infectious complication, followed by bloodstream infection, lower respiratory tract infection, and urinary tract infection [15]. Data show that the rate of resistant infections in burns is 56.82% [10]. The severity of damage, whether caused by low- or high-voltage electricity, depends on the length of time the individual is exposed to the current and the extent of contact with the electrical power supply [16]. In the present case, the patient developed shock and cardiac arrest immediately after electric shock and presented in critical condition, with recurrent fever, persistent pulmonary infection, and secondary wound infection. Serial bacterial cultures and antimicrobial susceptibility testing supported rational antibiotic administration throughout treatment.

Bacterial strains isolated from burn wards generally exhibit multidrug resistance and high genetic diversity, which facilitates cross-transmission within wards [12]. Shenoy et al noted that traditional epidemiological criteria for distinguishing community-acquired from nosocomial infections, based solely on onset setting, ward distribution, and time window, have clear

limitations and are insufficient for tracing pathogen transmission. They performed WGS on CRAB strains isolated from patient specimens and environmental samples, highlighting the risk of burn patients for early-onset nosocomial infections [11]. WGS is now widely used for molecular tracing of nosocomial infection outbreaks and provides accurate molecular evidence to distinguish recurrent CRAB infection from new nosocomial CRAB reinfection [17]. In the present case, 2 representative CRAB strains, WMTZ1 and WMTZ2, isolated at different time points and from different infectious sites during hospitalization, were subjected to WGS. A total of 135 SNPs were detected between the 2 strains, along with differences in drug resistance genes and insertion sequences. These findings indicate that the 2 isolates likely belonged to distinct clones, suggesting that subsequent positive CRAB results were most likely due to the acquisition of a new strain rather than the recurrence of the original strain. This also indirectly reflects the high genetic diversity of CRAB strains in burn ICUs, where alternating colonization and infection by multiple clones can occur.

Therefore, in addition to anti-infective therapy, strict adherence to nosocomial isolation protocols, environmental disinfection, and aseptic procedures may help reduce the risk of cross-transmission and recurrent infections caused by drug-resistant bacteria. There is still the possibility of overestimating the actual SNP diversity, even with the small sample size, because information about the relevant historical strains was not available for further comparison. The WGS method can accurately describe genetic differences between strains, identify potential links to infections resistant to medications, and serve as a basis for infection tracing, but this technique is rarely used in nosocomial outbreak surveillance. WGS requires storing previous strains of the pathogen, and it is difficult to fully reconstruct the chain of transmission of drug-resistant bacteria without them [11].

Conclusions

Early identification of CRAB colonization and invasive infection is central to the clinical management of such cases. This case highlights the importance of standardized antimicrobial stewardship and rigorous infection prevention and control strategies in ICUs and burn wards. This case report has several limitations. Large-scale clinical studies focusing on individualized antimicrobial regimens for patients with electrical

injuries complicated by CRAB infection remain scarce. Further large-scale clinical investigations are needed to optimize comprehensive diagnostic and treatment strategies for severe electrical injury complicated by recurrent CRAB infection.

Acknowledgements

We sincerely thank the medical and nursing staff of the ICU and the clinical laboratory, as well as all colleagues who assisted with clinical data collection and analysis for this case report.

During the preparation of this manuscript, an appropriate artificial intelligence language model was used to assist with English polishing, sentence structure optimization, and refinement of academic expression, ensuring the fluency, accuracy, and standardization of medical academic writing. All core research content, clinical data analysis, case sorting, view conclusion, and manuscript framework were independently completed by the authors. The use of artificial intelligence tools does not affect the authenticity, originality, or academic integrity of this study.

Department and Institution Where Work Was Done

Department of Clinical Laboratory, Taizhou Hospital of Zhejiang Province, affiliated to Wenzhou Medical University, Linhai, Zhejiang, PR China.

Patient Consent

The study involving human participant was approved by the Institutional Ethics Committee of Zhejiang Taizhou Hospital. The study was conducted in accordance with local legislation and institutional requirements (approval No. KL20250113). Given the retrospective nature of this study, the ethics committee waived the requirement for additional written informed consent. The patient had previously provided informed consent for their clinical imaging examinations as part of routine medical care, and all identifiable personal information was anonymized to protect patient privacy.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Bento D, Tobin EH. *Acinetobacter*. [Updated 2026 Mar 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK430784/>
2. Bartal C, Rolston KVI, Neshor L. Carbapenem-resistant *Acinetobacter baumannii*: Colonization, infection and current treatment options. *Infect Dis Ther*. 2022;11(2):683-94

3. Lee CR, Lee JH, Park M, et al. Biology of *Acinetobacter baumannii*: Pathogenesis, antibiotic resistance mechanisms, and prospective treatment options. *Front Cell Infect Microbiol.* 2017;7:55
4. Lees-Miller RG, Iwashkiw JA, Scott NE, et al. A common pathway for O-linked protein-glycosylation and synthesis of capsule in *Acinetobacter baumannii*. *Mol Microbiol.* 2013;89(5):816-30
5. Harding CM, Hennon SW, Feldman MF. Uncovering the mechanisms of *Acinetobacter baumannii* virulence. *Nat Rev Microbiol.* 2018;16(2):91-102
6. Jiang Y, Ding Y, Wei Y, Jian C, et al. Carbapenem-resistant *Acinetobacter baumannii*: A challenge in the intensive care unit. *Front Microbiol.* 2022;13:1045206
7. Adeyemi FM, Akinlade EA, Yusuf-Omoloye NA, et al. Carbapenem-resistance in *Acinetobacter baumannii*: Prevalence, antibiotic resistance profile and carbapenemase genes in clinical and hospital environmental strains. *BMC Infect Dis.* 2025;25(1):786
8. Sati H, Carrara E, Savoldi A, et al. The WHO Bacterial Priority Pathogens List 2024: A prioritisation study to guide research, development, and public health strategies against antimicrobial resistance. *Lancet Infect Dis.* 2025;25(9):1033-43
9. Zemaitis MR, Guirguis M, Cindass R. Electrical injuries. In: *StatPearls. Treasure Island (FL): StatPearls Publishing; July 6, 2025*
10. Maharjan S, Shrestha S, Acharya U, et al. Prevalence and determinants of multi-drug resistance bacterial infection among burn patients in a tertiary care center in Nepal. *Infect Drug Resist.* 2026;19:559803
11. Shenoy ES, Pierce VM, Sater MRA, et al. Community-acquired in name only: A cluster of carbapenem-resistant *Acinetobacter baumannii* in a burn intensive care unit and beyond. *Infect Control Hosp Epidemiol.* 2020;41(5):531-38
12. Gao J, Zhao X, Bao Y, et al. Antibiotic resistance and OXA-type carbapenemases-encoding genes in airborne *Acinetobacter baumannii* isolated from burn wards. *Burns.* 2014;40(2):295-99
13. Koumbourlis AC. Electrical injuries. *Crit Care Med.* 2002;30(11 Suppl.):S424-S30
14. Harding CM, Hennon SW, Feldman MF. Uncovering the mechanisms of *Acinetobacter baumannii* virulence. *Nat Rev Microbiol.* 2018;16(2):91-102
15. Váňa V, Lipový B, Cvanová M, et al. Results of microbiological surveillance in patients with high-voltage electrical injuries: A 10-year single center experience. *Epidemiol Mikrobiol Imunol.* 2025;74(2):97-106
16. Moukawam E, Aoun CB, Sfeir J, et al. Electrical burns: A retrospective study at the lebanese burn center in Geitaoui Hospital, Lebanon (2011-2024). *Int Wound J.* 2025;22(8):e70732
17. Jia H, Chen Y, Wang J, et al. Emerging challenges of whole-genome-sequencing-powered epidemiological surveillance of globally distributed clonal groups of bacterial infections, giving *Acinetobacter baumannii* ST195 as an example. *Int J Med Microbiol.* 2019;309(7):151339