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CASE REPORT

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A case of explosive community-acquired pneumonia and septic shock caused by Acinetobacter pittii

Xiaoying Zhan[1], Xin Tian[1*], Cangjian Zhang[2] and Jinqiang Ye[3]

Abstract

Background *Acinetobacter pittii*, belongs to the genus Acinetobacter, has a special pathogenesis and is commonly known as nosocomial pathogen; community infections are rare.

Objective To present a case study of community-acquired pneumonia and septic shock resulting from infection with Acinetobacter pittii and to investigate the diagnosis, clinical features and treatment of *Acinetobacter pittii* infection.

Methods The clinical features and prognosis of patients with *Acinetobacter pittii*, infection were analyzed retrospectively.

Results The sepsis caused by *Acinetobacter pittii,* was improved after treatment.

Discussion and conclusion Pneumonia caused by fully sensitive hypervirulent *Acinetobacter pittii* is rare, usually with acute course, severe illness and high mortality. It is necessary to identify the infectious agent as soon as possible, and early treatment can improve the success rate of treatment.

Keywords Acinetobacter pittii, Community-acquired pneumonia, Septic shock, Cavitary pneumonia, Metagenomic next-generation sequencing

Introduction

Acinetobacter species are commonly known as pathogens responsible for nosocomial infections, while community-acquired infections are relatively rare. *Acinetobacter pittii,* a member of the *Acinetobacter baumannii* genus, is typically found in aquatic, livestock, and environmental sources. Due to the limitations of current bacterial identification techniques in clinical laboratories, *Acinetobacter pittii* and hospital-acquired Acinetobacter are often misidentified as *Acinetobacter baumannii*. However, with the increasing use of next-generation sequencing (NGS), the detection rate of *Acinetobacter pittii* significantly improved. This article reports a case of community-acquired pneumonia caused by *Acinetobacter pittii*, leading to septic shock, multiple organ failure, and cavitary pneumonia.

Case report

The patient is a 50-year-old male, with a height of 172 cm and a weight of 80 kg. He has a long history of outdoor labor, with no prior health issues, no history of smoking or alcohol consumption, no underlying medical conditions, and no history of hospitalization.

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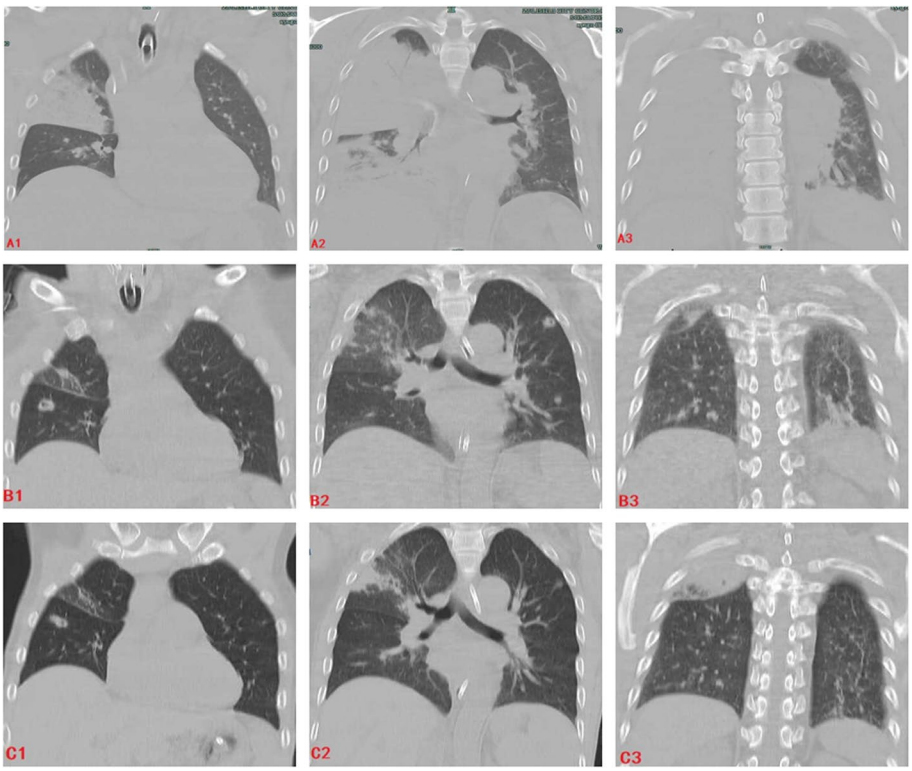

Fig. 1 **a** Chest CT on admission revealed obvious consolidation of the right lung **b** Chest CT examination on 17 days after admission revealed multiple nodular high-density shadows in both lungs with cavity formation **c** Chest CT examination on 35 days after admission revealed multiple solid nodules in both lungs with cavitation

Eighteen hours before admission, he developed a high fever of 39 °C with no obvious cause, accompanied by chills. Self-medication with cold medicine did not alleviate his symptoms (specific medication unknown), and he developed shortness of breath. At the local hospital, due to hypoxemia, he underwent tracheal intubation and was transferred to our hospital. Upon admission, the patient was under drug-induced sedation, his face was pale, his hands and feet were cold, his body temperature was 38.2 °C, his heart rhythm was regular, heart rate was 115 beats per minute, respiratory rate was 20 breaths per minute, and blood pressure was 72/53 mmHg (maintained with norepinephrine at 80 μg/kg·h). Auscultation revealed wet rales in both lungs during inhalation. The ventilator settings were AC/PC (PC 15 cmH2O, PEEP 7.0 cmH2O, 80% oxygen concentration), and tidal volumes fluctuated around 500 ml. Laboratory tests revealed: CRP 198.15 mg/L, PLT 34 × 10^9/L, PCT > 100.00 ng/ml, metabolic acidosis (pH 7.306, PCO2 34.8 mmHg, bicarbonate 16.8 mmol/L, lactate 5.0 mmol/L), renal insufficiency (Cr 133 μmol/L), and cardiac insufficiency (BNP 3463.2 pg/ml, myoglobin 1111.0 ng/ml, troponin 0.080 ng/ml). There was also a high inflammatory response (IL-6 4856.48 pg/ml, IL-10 131.66 pg/ml, IL-8 1030.99 pg/ml). The electrocardiogram showed no abnormalities, and cardiac ultrasound indicated a left ventricular ejection fraction of 49%. A chest CT scan showed large areas of consolidation in both lungs, more pronounced in the right lung (Fig.1). A brain CT scan was normal, while bronchoscopy revealed congestion of the mucosa in both bronchi. Upon admission, his APACHE II score was 28, and SOFA score was 22. He was diagnosed with "severe pneumonia, septic shock, and multiple organ dysfunction." Treatment included Imipenem-cilastatin 1 g q8h combined with moxifloxacin 0.4 g qd for anti-infection (Fig. 2), methylprednisolone 80 mg q12h for anti-inflammation, restrictive fluid resuscitation, improvement of cardiac function, expectorant therapy, and collection of bronchoalveolar lavage fluid for high-throughput sequencing and microbial culture. On the 4th day of hospitalization, metagenomic next-generation sequencing (mNGS) detected *Acinetobacter pittii* (sequence count: 257,029, relative abundance: 52.92%, gene coverage: 58.36%) (Fig. 3). After 7 days of hospitalization, Imipenem-cilastatin and moxifloxacin were discontinued, and piperacillin-tazobactam 4.5 g q8h was started for anti-infection therapy. On the 15th day

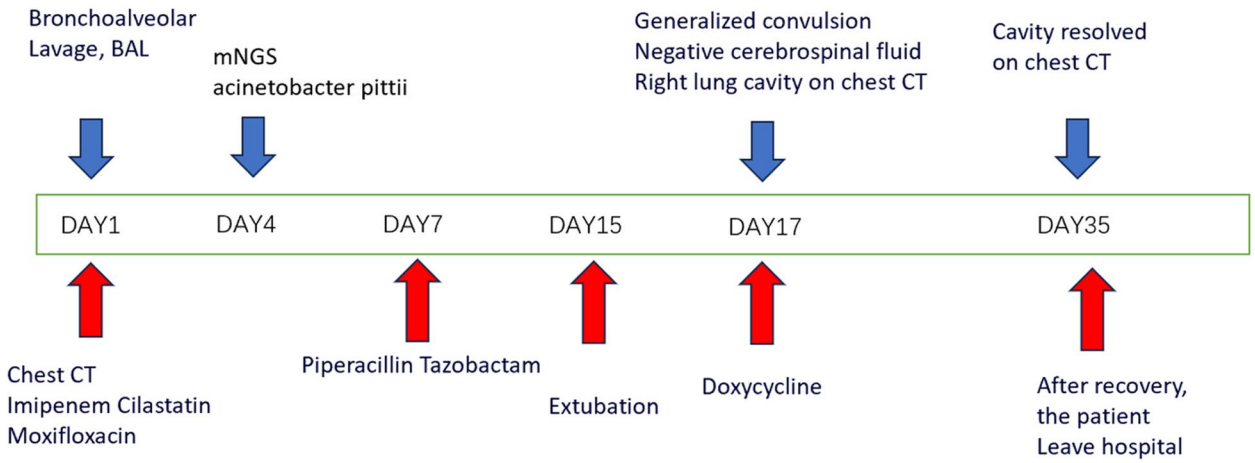

Fig. 2 Patient's treatment course

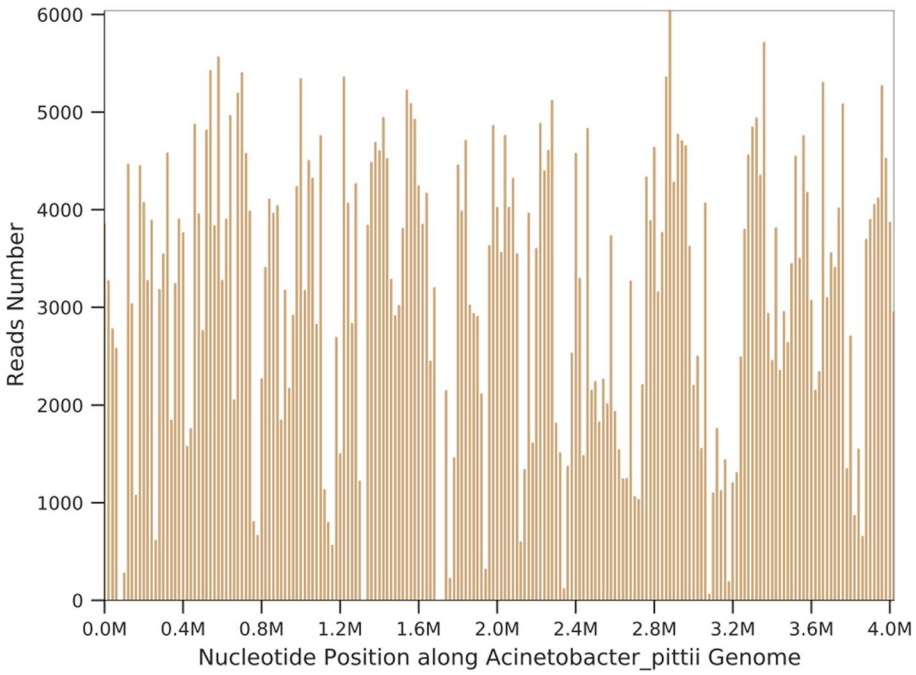

Fig. 3 bronchoalveolar lavage fluid macrogene sequencing results: 257,029 reads of Acinetobacter pittii, with total coverage of 58.36% and relative abundance of 52.92%

of hospitalization, the patient was successfully extubated, but 2 days post-extubation, the patient experienced recurrent paroxysmal convulsions, most notably in the jaw, lasting for about 30 min. Cerebrospinal fluid analysis revealed clear fluid, with 0 white blood cells and no abnormalities in bacterial culture. Electroencephalography, electromyography, brain MRI, and NGS for autoimmune encephalitis were all negative. A repeat chest CT scan showed a cavity-like lesion in the lower right lung, raising suspicion of fungal infection. Multiple tests for fungal galactomannan, β-D-glucan, and fungal cultures from bronchoalveolar lavage fluid were negative. Multiple sputum cultures revealed *Acinetobacter baumannii* susceptible to all antibiotics, while NGS of the sputum detected *Acinetobacter pittii* with high sequence counts. Multiple blood cultures were negative. Laboratory tests suggested highly virulent *Acinetobacter pittii*. The patient was diagnosed with "fulminant community-acquired pneumonia (*Acinetobacter pittii*)." On the 17th day of hospitalization,

based on drug sensitivity, treatment was switched to doxycycline. After 20 days of treatment, a follow-up chest CT showed improvement in the lung cavities.

Discussion

Acinetobacter pittii belongs to the Acinetobacter calcoaceticus-baumannii complex and is commonly found in moist environments such as wet soil, farms, wastewater, and even daily household items and animals [1]. It has a certain degree of pathogenicity, frequently causing infections during the summer and rainy seasons [2]. In this case, the patient had long-term outdoor labor in high-temperature and high-humidity conditions, with no underlying diseases or prior hospitalization, which made him susceptible to infection. However, the route of infection remains unclear. Multiple sputum cultures consistently showed *Acinetobacter baumannii*, and bronchoalveolar lavage fluid (BALF) NGS reported *Acinetobacter pittii*. A review of laboratory results revealed that these two bacteria have similar phenotypes. Due to the frequent use of technologies like matrix-assisted laser desorption/ionization (MALDI) or automated bacterial identification systems, *Acinetobacter pittii* is often misidentified as *Acinetobacter baumannii*, leading to limited understanding and underreporting of *Acinetobacter pittii* infections [1, 2]. Through next-generation metagenomic sequencing (NGS), we can gain a deeper understanding of *Acinetobacter pittii*.

This bacterium commonly affects immunocompromised individuals or those with underlying diseases, particularly the elderly, burn victims, trauma patients, and ICU patients [3]. Community-acquired fulminant pneumonia caused by Acinetobacter pittii is characterized by an acute and severe clinical course, with symptoms such as high fever, dyspnea, septic shock, and multi-organ failure affecting the heart, lungs, liver, kidneys, and even the nervous system. The mortality rate is high. This patient presented with an acute onset and severe condition, marked by severe metabolic acidosis, abnormal infection markers, progressive thrombocytopenia, septic shock, and significantly elevated inflammatory cytokines. Lung imaging revealed extensive consolidation, followed by cavitary pneumonia. Laboratory tests confirmed that the bacterium was highly virulent, leading us to hypothesize that *Acinetobacter pittii* may possess currently unknown virulence factors. However, the underlying mechanism remains unclear and requires further research [4, 5].

The pathogenic mechanism of multidrug-resistant *Acinetobacter baumannii* is often attributed to virulence factors, high adhesiveness, and biofilm formation [1]. In this patient, multiple sputum cultures during the first month of hospitalization consistently showed fully sensitive *Acinetobacter pittii*, with occasional multidrug-resistant strains detected (resistant to cefoperazone-sulbactam, ciprofloxacin, levofloxacin, imipenem, ceftazidime, piperacillin-tazobactam, etc.). This is markedly different from multidrug-resistant *Acinetobacter baumannii* typically found in ICUs. We hypothesize that the resistance rate of *Acinetobacter pittii* is lower than that of *Acinetobacter baumannii*, but further laboratory evidence is needed to clarify the resistance mechanisms. Additionally, when multiple cultures from community-acquired patients with no history of hospitalization consistently show fully sensitive *Acinetobacter baumannii*, we should highly suspect Acinetobacter pittii as the actual pathogen.

Reviewing past literature, cases of *Acinetobacter pittii*-induced fulminant community-acquired pneumonia have mostly been reported in regions like Taiwan, South Korea, and Australia. These cases are rare, and survival after treatment with follow-up is even rarer [6–8]. Effective antibiotic selection has been shown to reduce mortality [9]. According to the guidelines for severe community-acquired pneumonia [10], treatment options include carbapenems combined with quinolones for anti-infective therapy. Given the community onset in this patient, along with progressive thrombocytopenia and suspected Gram-negative bacterial infection, empirical treatment with imipenem-cilastatin and piperacillin-tazobactam, combined with moxifloxacin, was initiated. With adequate fluid resuscitation, the patient's condition improved significantly. The subsequent seizures and cavitary changes in the lungs lacked definitive laboratory evidence for a clear etiology, but we suspect an association with *Acinetobacter pittii* and *Acinetobacter baumannii* [11–13].

Conclusion

This report describes a case of fulminant community-acquired pneumonia caused by Acinetobacter pittii, which progressed rapidly. Acinetobacter pittii is extremely rare and prone to misdiagnosis, characterized by rapid onset, severe illness, strong virulence, high mortality, multi-organ failure, and even seizures and cavitary pneumonia. Early recognition and treatment can reduce mortality to some extent.

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Authors' contributions
Author contributions: Zhan XY designed the report and wrote the paper; Zhang CJ collected and analyzed the patient's clinical data;TianX revised the paper; all authors have read and approved the final version of this manuscript.

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Data availability
The datasets generated and/or analysed during the current study are not publicly available due [REASON WHY DATA ARE NOT PUBLIC] but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Written informed consent for publication has been obtained from the participants in this study.

Competing interests
The authors declare no competing interests.

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References

1. Vasconcellos L, Silva SV, da Costa LV, de Miranda RVDSL, Dos Reis CMF, Braga LMPDS, Silva C, Conceição G, Mattoso J, Silva IB, Forsythe SJ, Midlej V, Boas MHSV, Brandão MLL. Phenotypical and molecular characterization of Acinetobacter spp. isolated from a pharmaceutical facility. Lett Appl Microbiol. 2023;76(9):ovad101. https://doi.org/10.1093/lambio/ovad101. PMID: 37660241.
2. Wong D, Nielsen TB, Bonomo RA, Pantapalangkoor P, Luna B, Spellberg B. Clinical and pathophysiological overview of acinetobacter infections: a century of challenges. Clin Microbiol Rev. 2017;30(1):409–47. https://doi.org/10.1128/CMR.00058-16. PMID:27974412;PMCID:PMC5217799.
3. Morris FC, Dexter C, Kostoulias X, Uddin MI, Peleg AY. The mechanisms of disease caused by *Acinetobacter baumannii*. Front Microbiol. 2019;17(10):1601. https://doi.org/10.3389/fmicb.2019.01601. PMID:31379771;PMCID:PMC6650576.
4. Morris FC, Dexter C, Kostoulias X, Uddin MI, Peleg AY. The mechanisms of disease caused by *Acinetobacter baumannii*. Front Microbiol. 2019;17(10):1601. https://doi.org/10.3389/fmicb.2019.01601. PMID:31379771;PMCID:PMC6650576.
5. 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. (Epub 2021 Feb 4 PMID: 33618061).
6. Chusri S, Chongsuvivatwong V, Rivera JI, Silpapojakul K, Singkhamanan K, McNeil E, Doi Y. Clinical outcomes of hospital-acquired infection with Acinetobacter nosocomialis and Acinetobacter pittii. Antimicrob Agents Chemother. 2014;58(7):4172–9. https://doi.org/10.1128/AAC.02992-14. Epub 2014 May 12. PMID: 24820079; PMCID: PMC4068534.
7. Nishimizu S, Shiota S, Oshiumi T, Takakura T, Miyazaki E. A case of community-onset *Acinetobacter* pneumonia in a healthy person. IDCases. 2021;26(24):e01133. https://doi.org/10.1016/j.idcr.2021.e01133. (PMID:34007787;PMCID:PMC8111266).
8. Riddles T, Judge D. Community-acquired, bacteraemic acinetobacter baumannii pneumonia: a retrospective review of cases in tropical Queensland, Australia. Trop Med Infect Dis. 2023;8(8):419. https://doi.org/10.3390/tropicalmed8080419. (PMID:37624357;PMCID:PMC10458713).
9. Davis JS, McMillan M, Swaminathan A, Kelly JA, Piera KE, Baird RW, Currie BJ, Anstey NM. A 16-year prospective study of community-onset bacteremic Acinetobacter pneumonia: low mortality with appropriate initial empirical antibiotic protocols. Chest. 2014;146(4):1038–45. https://doi.org/10.1378/chest.13-3065. (PMID: 24901292).
10. Martin-Loeches I, Torres A, Nagavci B, Aliberti S, Antonelli M, Bassetti M, Bos L, Chalmers JD, Derde L, de Waele J, Garnacho-Montero J, Kollef M, Luna C, Menendez R, Niederman M, Ponomarev D, Restrepo M, Rigau D, Schultz MJ, Weiss E, Welte T, Wunderink R. ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia. Eur Respir J. 2023;61(4):2200735. https://doi.org/10.1183/13993003.00735-2022. (PMID: 37012080).
11. Feng W, Jiang X, Zhang R, Guo Z, Gao D. Diagnosis of an Acinetobacter pittii from a patient in China with a multiplex PCR-based targeted gene sequencing platform of the cerebrospinal fluid: A case report with literature review. Medicine (Baltimore). 2022;101(42):e31130. https://doi.org/10.1097/MD.0000000000031130. (PMID:36281177;PMCID:PMC9592278).
12. Lázaro-Díez M, Navascués-Lejarza T, Remuzgo-Martínez S, Navas J, Icardo JM, Acosta F, Martínez-Martínez L, Ramos-Vivas J. Acinetobacter baumannii and A. pittii clinical isolates lack adherence and cytotoxicity to lung epithelial cells in vitro. Microbes Infect. 2016;18(9):559-64. https://doi.org/10.1016/j.micinf.2016.05.002. Epub 2016 May 24. PMID: 27235198.
13. Larcher R, Pantel A, Arnaud E, Sotto A, Lavigne JP. First report of cavitary pneumonia due to community-acquired Acinetobacter pittii, study of virulence and overview of pathogenesis and treatment. BMC Infect Dis. 2017;17(1):477. https://doi.org/10.1186/s12879-017-2589-0. PMID: 28683724; PMCID: PMC5500973.

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