

Case Report

A rare case of *Legionella bozemanai* leading to necrotizing pneumonia in an immunocompromised patient: a case study

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Received: 22 September 2025

Accepted: 18 October 2025

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ABSTRACT

Legionella bozemanai is a rare organism of *Legionella* species that can cause mild fever to severe pulmonary pneumonia known as Legionnaires' disease. It can also cause extra-pulmonary infections, particularly in immunocompromised patients although deadly outcome in immunocompetent has also been reported. Diagnosis is often delayed due to limitations of conventional methods such as culture, serology, and urinary antigen testing, making newer molecular methods more valuable for early detection. We report the case of a 68-year-old male with history of relapsed acute myeloid leukemia (AML) and Sweet's syndrome who presented with worsening dyspnea to emergency department. For evaluation, CT chest and the Karius test were performed. Which confirmed *L. bozemanai* infection and lobar consolidation. Despite multiple courses of broad-spectrum antimicrobials, including fluoroquinolones (Levofloxacin), macrolides (Azithromycin), and beta-lactams (Amoxi-clav), his condition deteriorated. Repeat CT scan showed progressive bilateral consolidation with cavitation, this suggested progressing to necrotizing pneumonia. Invasive procedures were deferred due to profound pancytopenia. Escalation of antibiotics were done but the patient eventually succumbed to his illness. This case presents the diagnostic and therapeutic challenges of *L. bozemanai* infection in immunocompromised patient. Routine diagnostic methods often fail to identify this organism, whereas next-generation sequencing plasma cfDNA, was valuable in confirming the pathogen. However, treatment response was poor, likely because of profound immunosuppression and possible antimicrobial resistance, consistent with previously reported cases in the literature. *L. bozemanai* infection, though rare, should be considered in immunocompromised patients with progressive pneumonia unresponsive to standard therapy. Early use of advanced molecular diagnostics may facilitate timely diagnosis and treatment. Management may require combination antimicrobial therapy, as monotherapy has been associated with resistance and relapse. Aggressive management is recommended, as the infection can lead to lung tissue necrosis, as seen in our case.

Keywords: Legionella, Karius, *L. Bozemanii*, Immunocompromised, Necrotizing pneumonia

INTRODUCTION

Legionella species are Gram-negative bacilli bacteria that cause diseases ranging from mild febrile illnesses to severe, life-threatening pneumonia, which is usually known as Legionnaires' disease. *Legionella* are found in freshwater environments and cause infections through

aspiration of water containing bacteria or inhalation of aerosolized water containing the bacteria.¹ Among *Legionella* species, *L. pneumophila* is the most common cause of pneumonia, while other non-pneumophila species, such as *L. bozemanai*, are rare but can also cause severe infections, especially in immunocompromised hosts. Although it is quite rare, it has been increasingly

identified in patients having comorbidities like chronic lung disease, malignancies, or patients on immunosuppressive therapy.² In an immunocompromised host, *L. bozemanæ* often presents more severely, including bilateral lung infiltrates, cavitation, lung abscess formation, etc. Poor response to therapy and relapse have been reported. In extrapulmonary manifestations, it also causes soft tissue infection or even septic arthritis, especially with immunosuppression and local trauma.³⁻⁶ Diagnosis of *L. bozemanæ* is challenging as this is insensitive to urinary antigen tests and serological tests for *L. pneumophila* serogroup 1 and misses *L. bozemanæ*. For diagnosis, culture, PCR, next-generation sequencing, or 16S rRNA sequencing have proven valuable in confirming infection, particularly when early detection is required, as culture might take up to 10 days.⁷ Based on reported case reports and series, fluoroquinolones (Levofloxacin) and macrolides (e.g., erythromycin or azithromycin) have been used for management. Notably, there is also a case of relapsing pneumonia involving *L. bozemanæ* that was resistant to erythromycin but responded well to ceftriaxone and aztreonam, indicating the potential for variable antibiotic resistance patterns.^{8,9}

Here we report a case of *L. Bozemanæ* occurred in an immunocompromised patient having a relapse of AML and Sweet's syndrome.

CASE REPORT

A 68-year-old male with a history of relapsed AML and Sweet's syndrome presented with worsening cough and shortness of breath. He denied recent fever, chills, chest pain, abdominal pain, nausea, vomiting, diarrhea, or neurological symptoms. He reported mild intermittent epistaxis but no other bleeding manifestations. On examination, the patient was hypoxic with SpO₂ in the 60s. He was stabilized with a non-rebreather mask, after which his oxygen saturation improved to 98%. He was then transferred to the acute cancer care unit for further management. On further history, he reported recurrent pneumonia over the last two months. During previous episodes, CT chest and bronchoalveolar lavage had been performed. CT chest showed right lower lobe consolidation with a small pleural effusion, and Karius confirmed *L. bozemanæ* (>210,000) infection. Based on these findings, a diagnosis of *L. bozemanæ* pneumonia was made, and he was started on an 8-week course of levofloxacin 750 mg daily. Despite therapy, he presented again with worsening cough and dyspnea. Repeat imaging showed worsening nodular opacities and consolidation in the right upper lobe (RUL) and right lower lobe (RLL). A follow-up CT after one week revealed consolidation of right lower lobe, small right pleural effusion, few ground glass and linear opacities also present in the right middle lobe. Which were suggestive of progression of pneumonia with development of a cavitary right lower lobe lesion for lung abscess and necrotic lung due to recurrent pneumonia.



Figure 1: CT chest showing right lower lobe consolidation with pleural effusion.

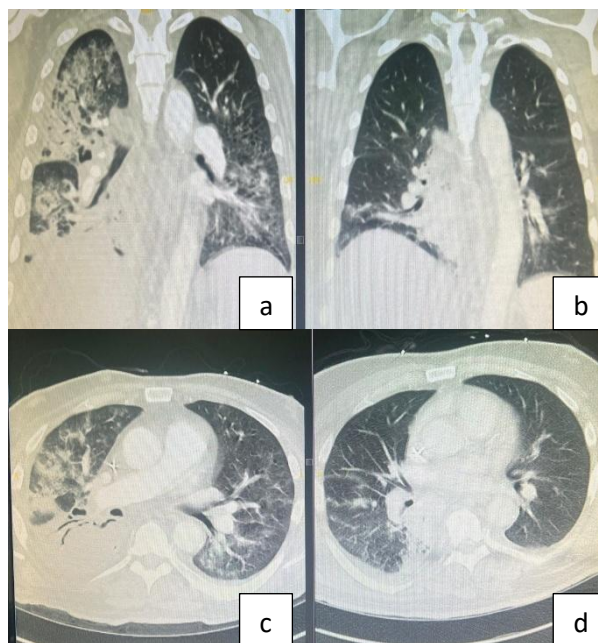


Figure 2 (a-d): CT chest showing ground glass and linear opacities.

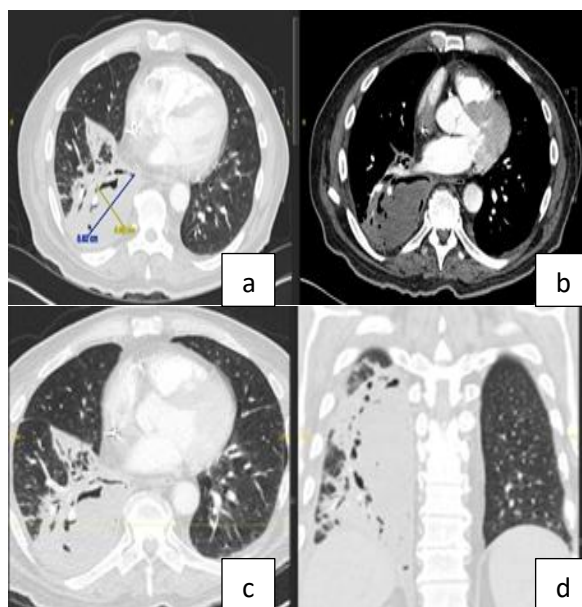


Figure 3 (a-d): CT pulmonary angiography showing cavitary lesion in right lung.

Based on this, it was came in to notice that pneumonia has progressed to necrotizing pneumonia. For further evaluation, a bronchoalveolar lavage was performed, which revealed *C. tropicalis*, but was negative for *L. Bozemanae*. Performed Karius testing again detected *L. bozemanae* (39,846).

Due to borderline neutropenia (ANC 990), lymphopenia (ALC 30), and thrombocytopenia, surgical drainage was deferred. He was initially treated with cefepime and vancomycin, later escalated to piperacillin-tazobactam. Infectious disease consultation recommended antifungal therapy. Voriconazole 300 mg twice daily was started but later switched to posaconazole 300 mg twice daily due to visual hallucinations. On discharge, he was prescribed azithromycin 500 mg daily for 3 months and amoxicillin-clavulanate 875 mg twice daily for 6 weeks, with instructions for follow-up CT chest imaging. After a week, his condition worsened and he again presented to the emergency department with dyspnea, dizziness on standing/walking, SpO₂ of 58%, and hypotension (80/40 mmHg).

After stabilization, he was admitted for further evaluation and management of sepsis and worsening pneumonia. Laboratory studies suggested pancytopenia secondary to malignancy and antineoplastic therapy. A CT chest with IV contrast revealed extensive consolidation in both lungs with multiple foci of air in the medial and posterior right lung, likely representing cavitation. These findings suggested worsening pneumonia compared to the prior examination. While continuing ongoing antibiotics, cefepime was added for 7 days with the option to extend if needed. After a week of admission, the patient's condition deteriorated, and a chest X-ray was ordered due to worsening hypoxia. It showed increased interstitial and airspace pulmonary opacities with a moderate right pleural effusion and a small left-sided pleural effusion. Despite being on multiple antimicrobials for over a week, no significant improvement was observed. Afterwards, levofloxacin was discontinued and treatment was shifted to IV vancomycin and cefepime. Despite aggressive management, the patient's condition worsened, and he passed away a few days later due to necrotizing pneumonia.

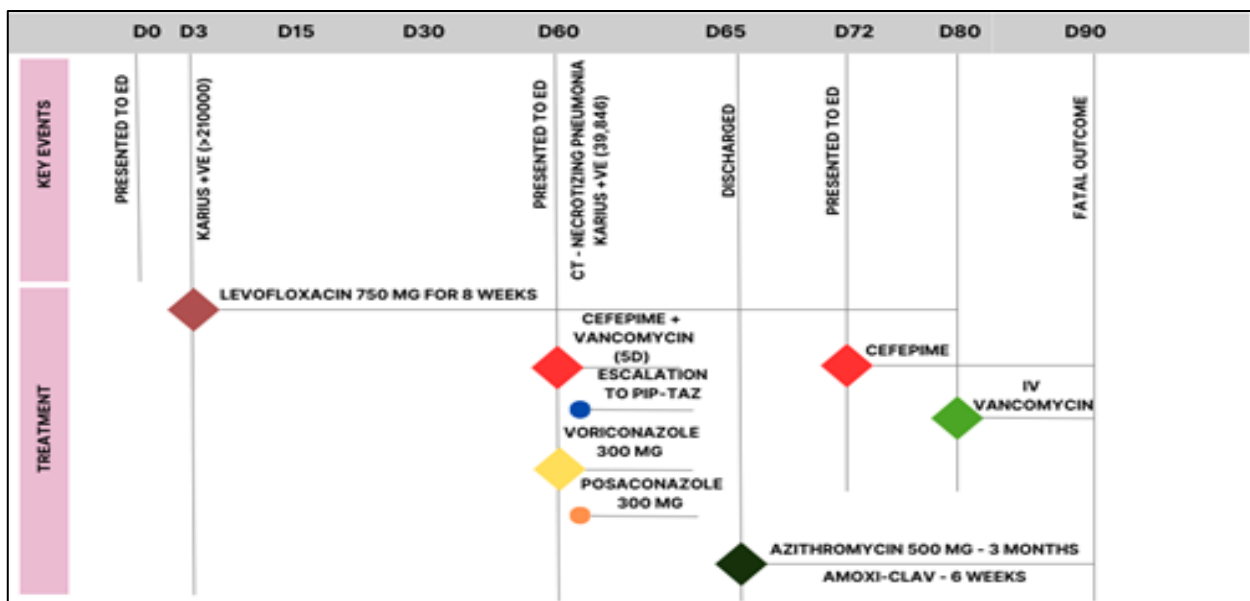


Figure 4: Timeline of patient: test performed, treatments given.

DISCUSSION

Legionella species are commonly found in the environment, where they colonize water sources and are transmitted through inhalation or aspiration of contaminated water. Among them, *L. pneumophila* is the most frequent cause of pneumonia, whereas *L. bozemanae* is a rare pathogen responsible for pulmonary and extra-pulmonary infections, primarily affecting immunocompromised patients.^{1,2} Reported risk factors for *Legionella* infection include immunosuppression (such as hematologic malignancies, corticosteroid therapy, and stem cell transplantation), advanced age, and chronic lung disease.^{8,10} In our patient, relapsed AML with

pancytopenia and prior corticosteroid exposure for Sweet's syndrome led to profound immunosuppression, making him particularly vulnerable to *L. bozemanae* infection.

Legionnaires' disease is clinically and radiologically indistinguishable from other forms of pneumonia, making microbiologic testing essential for accurate diagnosis. Culture of respiratory samples remains the gold standard and can detect most *Legionella* species, though it has limited sensitivity and may take up to 10 days. Direct fluorescent antibody testing offers faster results but with lower sensitivity. Urinary antigen testing is the most commonly used method, providing rapid and highly specific results, but it is limited to detecting *L.*

pneumophila serogroup 1. Traditional serology is mainly useful for retrospective studies because of delayed seroconversion, whereas newer PCR-based assays have shown greater sensitivity and faster detection of *Legionella* species.⁷ In our case, Karius test-a plasma-based next-generation sequencing assay that identifies microbial cell-free DNA (cfDNA)-was used for diagnosis.

This non-invasive approach is particularly valuable in immunocompromised patients, where invasive procedures carry significant risk and standard microbiological methods often lack sensitivity.^{11,12} To better understand diagnostic challenges, we reviewed published case reports that employed various methods, underscoring the need for advanced molecular tools in detecting *L. bozeman*.

Table 1: Characteristics of reported *L. bozeman* infections.

Authors	Patient	Diagnostic method	Outcome
Just et al ⁴	Immunocompromised, joint infection	Culture + species identification	Survived (treated monoarthritis)
Khan et al ¹³	Renal transplant recipient, brain abscess	Molecular methods (16S/culture)	Fatal
Caldararo et al ¹⁰	Immunocompromised, pulmonary infection	Karius plasma cfDNA NGS + concordant tissue PCR	Improved after targeted therapy
Xu et al ³	Immunocompromised, severe pneumonia	SGS of plasma and BALF	Recovered after targeted therapy
Siddiqui et al ¹⁴	Immunocompetent, pneumonia	Culture from lung tissue on autopsy	Fatal

*SGS-Second-generation sequencing, BALF-bronchoalveolar lavage fluid, NGS-Next-generation sequencing.

The patient had a history of relapsed AML and Sweet's syndrome; hence invasive procedures were deferred. Karius testing identified *L. bozeman* (>210,000), and treatment was started with an 8-week course of levofloxacin 750 mg daily. Despite this, after several weeks of antibiotics, his overall condition deteriorated, and repeat CT imaging showed worsening pneumonia, with Karius again detecting *L. bozeman* (39,846). So, in our case Karius was used to assess overall response to therapy. This suggested that the amount of *Legionella* was decreasing but might still have been present in the patient's lungs, leading to necrotizing pneumonia or the possibility of a secondary infection. However, no secondary infection was identified in culture. The absence of typical pathogens such as *S. aureus*, *Pseudomonas*, *Klebsiella*, or *Nocardia* further supports the consideration that the necrosis could have been due to *Legionella*. There are reported cases where *Legionella* has caused cavitary Legionnaires' disease, including necrotizing pneumonia, particularly in patients with hematologic malignancies and those receiving immunosuppressive therapy such as steroids or chemotherapy.¹⁵ As Montero-Arias and others reported a rare case of necrotizing pneumonia in a patient having sweet syndrome, in our patient this could be a cause of unusual course of disease, which ended up as necrotizing pneumonia.¹⁶ During admission, cefepime and vancomycin were added, later escalated to piperacillin-tazobactam. He was subsequently discharged on azithromycin 500 mg daily for 3 months and amoxicillin-clavulanate 875 mg twice daily for 6 weeks, with instructions for follow-up. There is no clear consensus among infectious disease specialists on the best treatment approach for persistent *Legionella* infection. However, based on a systematic review published on therapeutic strategies in cavitary Legionnaires' disease, common therapies include monotherapy with levofloxacin or azithromycin, followed by bi-therapy such as a

combination of a fluoroquinolone and a macrolide and Triple therapy with a fluoroquinolone, a macrolide, and rifampicin has also been shown to be effective.¹⁵

Table 2: Antimicrobials used for cavitary legionnaires' diseases.¹⁵

Monotherapy	Bi-Therapy	Triple- therapy
Levofloxacin	Levofloxacin + spiramycin	Rifampicin + erythromycin + levofloxacin
Azithromycin	Rifampicin + ciprofloxacin	Rifampicin + spiramycin + levofloxacin
Moxifloxacin	Levofloxacin + azithromycin	
Clarithromycin	Ciprofloxacin + azithromycin	

Despite being on multiple antimicrobials, his condition worsened, and widespread lung consolidation was identified. In the literature, recurrent *L. bozeman* infection with macrolide resistance has been documented.^{8,17} Although the mechanism of resistance is not clear, it has been reported that *Legionella* species, including *L. bozeman*, produce beta-lactamases that make them resistant to aminoglycosides. Furthermore, after being phagocytosed, *L. bozeman* multiplies within leukocytes and macrophages; therefore, drugs with higher penetration into phagocytes, such as azithromycin, erythromycin, and rifampicin, have shown promising results.⁸ So, in our patient, the poor response to antibiotics could be attributed to immunosuppression or antimicrobial resistance. Although microbiological testing showed improvement, his clinical condition worsened due to necrotizing pneumonia, and he eventually succumbed to the illness. This raises questions regarding management in

similar cases as patient exhibited a slow, progressive necrotizing infection in lungs despite being on treatment.

CONCLUSION

This case highlights the severity of the rare organism *L. bozemanii*, despite treatment with multiple antimicrobials. Failure of therapy or a slow response can worsen the condition and progress to necrotizing pneumonia, especially in patients with multiple comorbidities such as hematologic malignancies and/or immunosuppression, which may lead to a fatal outcome. Furthermore, the use of advanced diagnostic methods like Karius enables earlier detection of the organism, particularly in immunocompromised patients where culture results may take many days. Managing such patients is crucial; therefore, the therapeutic regimen should include multiple antimicrobials, as poor response to monotherapy with levofloxacin or azithromycin has been reported due to resistance in *L. bozemanii*. This case presents a significant clinical challenge with no straightforward management strategies, underscoring the need for further research on atypical *Legionella* infections.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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Cite this article as: Hirpara A, Batukbhai BP, Ribadiya FN, Undhad H, Greene J. A rare case of *Legionella bozemanii* leading to necrotizing pneumonia in an immunocompromised patient: a case study. Int J Res Med Sci 2025;13:4941-5.