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Case Report

Case Report: Legionella Necrotising Pneumonia in a Diabetic Patient—A Clue to Co-Infection?

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Abstract

Aims/Background: Computerised tomography findings in patients with *Legionella* pneumonia usually demonstrate airspace consolidation and/or ground glass opacities (GGO). Associated necrosis, abscess formation, or cavitation are usually limited to patients who are clearly immunocompromised. **Case presentation**: We report a recent patient who was immunocompetent, other than controlled type 2 diabetes, and had hospital-acquired severe *Legionella* pneumonia. The patient was found to have pulmonary necrosis and bacterial co-infection with *Acinetobacter*. **Results**: The patient underwent endotracheal intubation and mechanical ventilation, and was treated with intravenous azithromycin and colistin. However, the patient's condition continued to deteriorate, and she eventually died from multiple organ failure. **Conclusion**: The unusual appearance of necrotising pneumonia in Legionnaires' disease patients without frank immunosuppression may signify a co-infection with other microorganisms, which must be sought.

Keywords: *Legionella* pneumonia; nosocomial infections; immunocompetent; *Acinetobacter*; necrotising pneumonia; co-infection; case report

1. Introduction

Legionnaires' disease (LD), as the infection with the Gram-negative *Legionella* species was termed, is a common but underdiagnosed cause of community-acquired pneumonia worldwide. It is a waterborne disease since bacteria are located in aquatic habitats, soil, and water distribution systems. Infection is contracted by aerosol inhalation, which is followed by severe pneumonia mandating intensive care admission in as many as 20–40% of cases [1]. Patients aged 65 years or older, a history of smoking or chronic lung disease, and those with immunocompromising conditions (chronic steroid use, organ transplant, solid tumour or haematological malignancy) are at increased risk of developing LD [2]. An epidemiologic study has also identified diabetes mellitus as a risk factor [3]. Sample cultures of the lower respiratory tract remain the gold standard for detecting LD, but being cumbersome and technically demanding, urinary antigen detection (fastest, 56–99% sensitive, and ≥99% specific), molecular techniques, and microscopy are important in diagnosis [4]. Most macrolides, tetracyclines, and quinolones are effective and active within the cells with a recommended duration of treatment of 5–10 days for levofloxacin and 3–5 days for azithromycin [4]. The imaging features of LD have been well-defined, and computed tomography (CT) usually reveals multifocal or multilobar airspace consolidation and ground glass opacities with common pleural effusions [5].

Cavitary Legionnaires' disease, as we report below, is not only unusual but mostly reported in the context of significant underlying disease and immunosuppression [6]. A recent case reported below, added to previous observations identified in a search of the literature, may suggest bacterial co-infection as an additional risk factor of this rare finding.

2. Case Report

A 76-year-old woman was admitted with a 1-day history of lassitude. She had Alzheimer's dementia, type 2 diabetes (glycated haemoglobin A1c [HbA1c] 6.7%) and osteoporosis and was an independent, past-smoker, living at home and treated with ginkgo bilboa extract 120 mg bid, quetiapine 25 mg, sitagliptin/metformin 50/1000 mg, atorvastatin 20 mg and risedronate 150 mg/month. On admission, she was febrile (39.4 °C) and acutely confused, but comfortable with normal vital signs, physical examination, chest X-ray, electrocardiogram (ECG), and head CT. Haemoglobin (Hb) was 11.2 g/dL (reference range: 12–15 g/dL), white blood cell count (WBC) 15 $\times$ 10^9/L (reference range: 3.8–9.8 $\times 10^9$/L), platelets 180 $\times$ 10^9/L (reference range: 140–400 $\times$ 10^9/L), and C-reactive protein (CRP) 71 mg/L (reference range: 0–5 mg/L) with normal electrolytes, liver enzymes, and urinalysis. Cultures (blood, urine, sputum) were negative, no viruses were detected on nasopharyngeal swabs by polymerase chain reaction (PCR), and the respiratory panel BioFire PCR was twice negative. The

Table 1. Individually-reported cases of five patients with co-infection and pulmonary cavitation in Legionnaires' disease*.

Age (years)	Gender	Acquisition	Background	Imaging	Microbiology	Outcome	Reference
73	M	Community	Smoker	Multiple cavitations	*Pseudomonas*	R.	[18]
75	W	Community	Aspiration, tsunami	Necrotising pneumonia + abscess	*Escherichia coli*	R.	[19]
28	M	Community	Smoker	Abscess	*Fusobacterium nucleatum*	R.	[20]
33	W	Community	Cushingoid?	Abscesses	*Candida, Aspergillus*	Died	[21]
76	W	Hospital	Diabetes	Necrotising pneumonia	*Acinetobacter*	Died	Current

* In a sixth likely case of a 34-year-old immunocompetent woman with *Legionella* pulmonary abscess and empyema, cultures were not obtained before antibiotic therapy [22].

Abbreviations: M, man; W, woman; R, recovered.

cerebrospinal fluid was normal. Intravenous ceftriaxone (Rocephin, Hoffman-La Roche Inc., Nutley, NJ, USA) was started, and the patient quickly improved.

However, on the fourth hospital day, she became increasingly dyspneic (respiratory rate 38/minute), desaturated (89% oxygen saturation on ambient air), and developed tachycardia (heart rate 112/minute, sinus). Temperature spiked at 39.9 °C, and crackles were heard over both lungs. Chest X-ray demonstrated bilateral opacities, and CT revealed bibasilar consolidations, with necrosis in the left lower lobe (Fig. 1). WBC doubled (27.4×10^9/L), CRP rose to 345 mg/L, and serum albumin fell (4.1 to 2.6 g/dL). Bacterial culture results remained negative, but the repeat BioFire respiratory panel now detected *Legionella* and *Acinetobacter* on several different samples. Urine was also positive for *Legionella* antigen. Each of these tests is rapid, inexpensive, and approximately 100% specific [7]. Her protein electrophoresis showed no monoclonal spike, the kappa to lambda free light chains ratio was normal, and serum immunoglobulins showed no deficiency. At the same time, the department and its air conditioning system underwent renovation, and another patient was diagnosed with in-hospital *Legionella* pneumonia. This other patient had no co-infection and no atypical features on imaging. Due to acute hypoxemic respiratory failure, our patient was intubated, ventilated and treated with intravenous azithromycin (for *Legionella*) (Azenil, Pfizer, Kalamazoo, MI, USA) and colistin (for *Acinetobacter*, according to sensitivity tests) (Coly-Mycin M Parenteral, JHP Pharmaceuticals, Rochester, MI, USA). Nevertheless, she worsened (WBC 34.3×10^9/L, CRP 450 mg/L, albumin 1.6 g/dL), became hypotensive, developed acute kidney injury, and sustained type 2 myocardial infarction. She died of multiorgan failure despite intensive treatment.

The Care Checklist has been attached as **Supplementary Material** associated with this article.

3. Discussion

Legionella pneumophila (*L. pneumophila*) are aerobic, Gram-negative, intracellular pathogens causing pneumonia in hosts that are frequently elderly, smokers, diabetic, or immunocompromised—especially where impaired cellular immunity is concerned [7,8]. Travel or occupational exposures (e.g., truck drivers) are other established risk factors, but the diabetes association is especially intriguing. Type 2 (also type 1) diabetes has been demonstrated to carry an increased relative risk for hospitalisation with pneumonia (1.23, 95% confidence interval [CI] 1.19–1.28), increasing in patients with poorly-controlled disease and showing a gradient with increasing HbA1c levels [9]. In that study, the microbiology of pneumonia was not investigated, but patients with diabetes are particularly susceptible to *L. pneumophila* infection, although it was not mentioned in a review published in a leading journal [4]. A recent study demonstrated an important pathogenetic pathway, using both *in vitro* and *in vivo* experiments in infected diabetic guinea pigs to show that high glucose reduced tumour necrosis factor-alpha and interleukin-6 secretion in response to *Legionella* [10]. Our patient had three risk factors (age, diabetes, past-smoker), but though she was admitted with an acute febrile illness, her pneumonia was nosocomial, one of two cases diagnosed in our department within a fortnight, likely due to imperative ongoing construction work.

Legionella contaminates manmade water reservoirs or air conditioning systems, and waterborne-associated aerosol inhalation exposure underlies both community-acquired and health care-associated pneumonia. The majority of cases are sporadic and contracted in the community. However, *L. pneumophila* outbreaks and clusters are not uncommon, such as the well-known American Legion 1976 convention in Philadelphia, or the Melbourne aquarium cooling tower contamination affecting 125 visitors. The global epidemiological situation of *Legionella* can be summarised as follows: *Legionella* causes a meaningful fraction (4–5%) of community-acquired pneumonia that requires hospital admission, suggesting an estimated incidence of 2.8/100,000 population, though heterogeneity is high between countries and societies [11]. Interestingly, the incidence has increased from 0.48 (1992–2002) to 2.71 per 100,000 (2018) in the USA, and a less pronounced rise was also reported in Europe [12]. Nosocomial (i.e. hospital-acquired) *L. pneumophila* pneumonia was identified by the Centres for Disease Control and Prevention (CDC) national surveillance in 463/1284 (36.1%) of confirmed healthcare exposures in 2019, including 'pos-

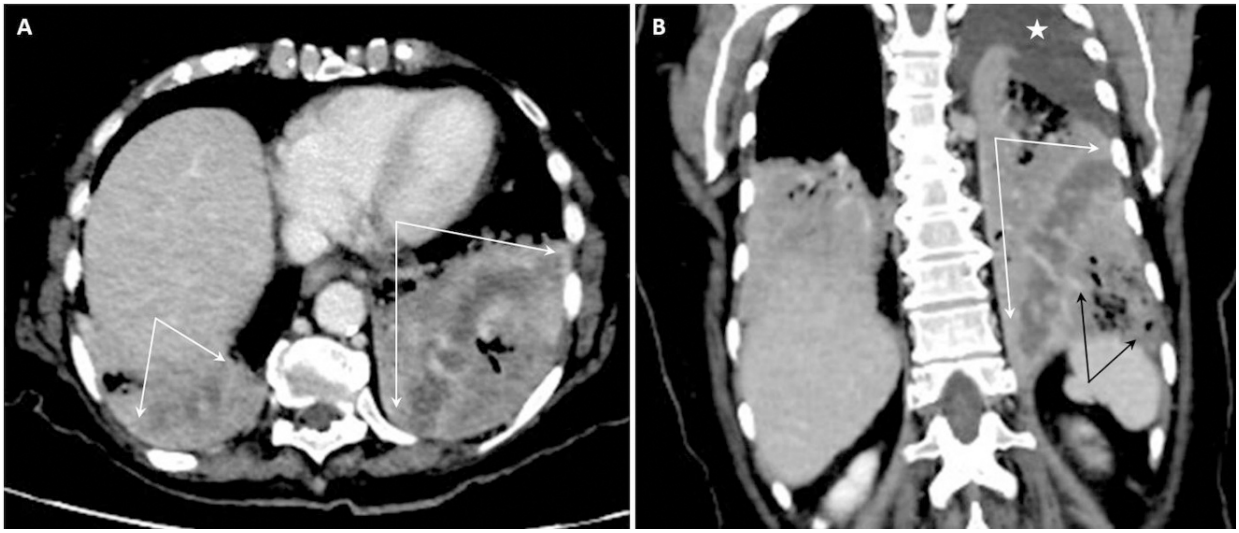

Fig. 1. Contrast-enhanced chest computed tomography (CT) of a 76-year-old patient with *Legionella* and *Acinetobacter* pneumonia. Axial (A) and coronal reformat (B) demonstrate bilateral lower lobes airspace consolidations with air bronchogram and areas of decreased attenuation highly suspicious for early pulmonary necrosis (A and B, white arrows). A well-demarcated stripe-shaped decreased attenuation is seen in the left lower lobe (B, white arrows). The partially aerated low attenuation lung parenchyma in the left lower lobe (B, black arrows) demonstrated ground glass opacities (GGO) on the lung window (B, black arrow). Mediastinal lymphadenopathy and pleural effusions were also present (B, asterisk).

sible' exposures [13]. Thus, despite the steadily increasing incidence of Legionnaires' disease in the USA (more than 6-fold since 2000) [13], cases of nosocomial *Legionella* pneumonia remain a minority. In an analysis centred on the radiographic features of nosocomial *Legionella* infections, only 2/71 (2.8%) had cavitation at presentation, but this number increased to 6/71 (8.45%) after 8–10 days [14]. All these patients had either received a transplant or been treated with high doses of steroids. Cavitation has been reported only rarely in previous large series of LD [8,15]. Thus, while the cause of our cluster of 2 nosocomial LD cases may be related to ongoing construction work nearby, the imaging features (Fig. 1) are rarely encountered. CT findings in patients with *Legionella* pneumonia usually demonstrate airspace consolidation and/or ground glass opacities (GGO). In contrast, abscess formation, necrosis, or cavitation are distinctly unusual, except in immunosuppressed patients [5,16]. In one series of 12 patients (of 1686) with slowly-resolving Legionnaires' disease, 10/12 (83%) had been immunosuppressed and 5 had lung abscesses [17]. Otherwise, *Legionella* infection causing lung abscess has been described in isolated case reports. Our patient's co-infection with *Acinetobacter* could have been the cause of pulmonary cavitation as observed in almost one-half of a series of patients with cavitary *Legionella* infections accrued from the literature over many years [6]. Co-infection was not discovered in our second non-immunosuppressed patient, who had *Legionella* pneumonia and unremarkable imaging features. In a recent extensive review of the literature focusing on cavitary Legionnaires' disease, only 29 patients were identified, and two local cases were reported. The majority were receiving one or more immunosuppressive treatments (steroids 85%, chemotherapy 45%, immunomodulatory drug 40%), and in 11/29 (38%), a co-infection was identified, including an almost equal number of aerobic and anaerobic bacteria, and 2 fungal infections with *Aspergillus* [6]. Table 1 (Ref. [18–22]) summarises individually-reported cases of co-infection and pulmonary cavitation in LD, supporting the suggested association. Although a few cases of LD in non-immunocompromised patients may develop cavitation without an identified co-infection, this remains a rare occurrence [23]. The mortality in nosocomial *Legionella* pneumonia was found to be more than double that of community-acquired cases [24], and pulmonary necrosis may be a contributing factor, although it was not studied.

The strength of our study is in identifying a novel association of a concurrent bacterial co-infection in LD patients with necrotising pneumonia who are not immunosuppressed, supported by a literature review identifying 4 more similar cases, and a potential fifth case (Table 1). Limitations are those of a single case report and PCR-based identification (although this was repeatedly positive, known to be accurate, and negative for all other respiratory organisms).

4. Conclusion

Pulmonary necrosis is unusual in Legionnaires' disease patients who are not immunosuppressed, and its occurrence in these patients may suggest the presence of a concurrent bacterial infection, which should be sought and treated.

Learning Points

• *Legionella* causes 4–5% of community-acquired pneumonia that requires hospital admission, and may cause severe disease mandating intensive care admission.

• *Legionella* pneumonia usually demonstrates airspace consolidation and/or ground glass opacities on imaging, whereas cavitation/abscess mostly occur in patients with human immunodeficiency virus, cancer, post-transplant, and on immunosuppressive medications.

• Legionnaires' disease patients who are not immunosuppressed and develop necrotising pneumonia/cavitation should be investigated for bacterial co-infection, an important, treatable association.

Availability of Data and Materials

All the data of this study are included in this article.

Author Contributions

AS, ID and LU designed the work. AS primarily researched and wrote the manuscript. All authors contributed to the critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Research was conducted in strict accordance with the ethical principles outlined in the Declaration of Helsinki. Informed consent was obtained from and signed by the participant's next of kin.

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Conflict of Interest

AS is serving as one of the Editorial Board Members of this journal. We declare that AS had no involvement in the review of this article and has no access to information regarding its review. Full responsibility for the editorial process for this article was delegated to Fraser Russell Millar. Other authors declared no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/BJHM55140.

References

[1] Rello J, Allam C, Ruiz-Spinelli A, Jarraud S. Severe Legionnaires' disease. Annals of Intensive Care. 2024; 14: 51. https://doi.org/10.1186/s13613-024-01252-y.

[2] Berrington WR, Hawn TR. Human susceptibility to legionnaires' disease. Methods in Molecular Biology. 2013; 954: 541–551. https://doi.org/10.1007/978-1-62703-161-5_33.

[3] Cooley LA, Pondo T, Francois Watkins LK, Shah P, Schrag S, Active Bacterial Core Surveillance Program of the Emerging Infections Program Network. Population-Based Assessment of Clinical Risk Factors for Legionnaires' Disease. Clinical Infectious Diseases. 2020; 70: 2428–2431. https://doi.org/10.1093/cid/ciz771.

[4] Cunha BA, Burillo A, Bouza E. Legionnaires' disease. Lancet. 2016; 387: 376–385. https://doi.org/10.1016/S0140-6736(15)60078-2.

[5] Yu H, Higa F, Hibiya K, Furugen M, Sato Y, Shinzato T, et al. Computed tomographic features of 23 sporadic cases with Legionella pneumophila pneumonia. European Journal of Radiology. 2010; 74: e73–e78. https://doi.org/10.1016/j.ejrad.2009.04.011.

[6] Moretti M, De Boek L, Ilsen B, Demuyser T, Vanderhelst E. Therapeutical strategies in cavitary legionnaires' disease, two cases from the field and a systematic review. Annals of Clinical Microbiology and Antimicrobials. 2023; 22: 105. https://doi.org/10.1186/s12941-023-00652-5.

[7] Bai L, Yang W, Li Y. Clinical and Laboratory Diagnosis of Legionella Pneumonia. Diagnostics. 2023; 13: 280. https://doi.org/10.3390/diagnostics13020280.

[8] Broome CV. Pneumonia due to Legionella species. Seminars in Infectious Diseases. 1983; 5: 16–35.

[9] Kornum JB, Thomsen RW, Riis A, Lervang HH, Schønheyder HC, Sørensen HT. Diabetes, glycemic control, and risk of hospitalization with pneumonia: a population-based case-control study. Diabetes Care. 2008; 31: 1541–1545. https://doi.org/10.2337/dc08-0138.

[10] Zhang Y, Liang S, Deng Z, Zhao Z, Han X. High-glucose conditions attenuate the response of macrophages to Legionella pneumophila infection by inhibiting NOD1 and MAPK signaling. International Immunopharmacology. 2024; 134: 112254. https://doi.org/10.1016/j.intimp.2024.112254.

[11] Graham FF, Finn N, White P, Hales S, Baker MG. Global Perspective of *Legionella* Infection in Community-Acquired Pneumonia: A Systematic Review and Meta-Analysis of Observational Studies. International Journal of Environmental Research and Public Health. 2022; 19: 1907. https://doi.org/10.3390/ijerph19031907.

[12] Barskey AE, Derado G, Edens C. Rising Incidence of Legionnaires' Disease and Associated Epidemiologic Patterns, United States, 1992-2018. Emerging Infectious Diseases. 2022; 28: 527–538. https://doi.org/10.3201/eid2803.211435.

[13] Barskey A, Lee S, Hannapel E, Smith J, Edens C, Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, CDC. Legionnaires' Disease Surveillance Summary Report, United States 2018-2019. 2024. Available at: https://www.cdc.gov/legionella/health-depts/surv-reporting/2018-19-surv-report-508.pdf (Accessed: 27 August 2025).

[14] Domingo C, Roig J, Planas F, Bechini J, Tenesa M, Morera J. Radiographic appearance of nosocomial legionnaires' disease after erythromycin treatment. Thorax. 1991; 46: 663–666. https://doi.org/10.1136/thx.46.9.663.

[15] Kirby BD, Peck H, Meyer RD. Radiographic features of Legionnaires' disease. Chest. 1979; 76: 562–565. https://doi.org/10.1378/chest.76.5.562.

[16] Nakanishi M, Shiroshita A, Nakashima K, Takeshita M, Kiguchi T, Yamada H. Clinical and computed tomographic features of Legionella pneumonia with negative urine antigen test results. Respiratory Investigation. 2021; 59: 204–211. https://doi.org/10.1016/j.resinv.2020.11.005.

[17] Pouderoux C, Ginevra C, Descours G, Ranc AG, Beraud L, Boisset S, et al. Slowly or Nonresolving Legionnaires' Disease: Case Series and Literature Review. Clinical Infectious Diseases. 2020; 70: 1933–1940. https://doi.org/10.1093/cid/ciz538.

[18] Gibney RT, Herbert FA, King EG, Elliot JF. Prolonged cavitating pneumonia in a patient with serologic evidence of Legionnaires' disease. Chest. 1980; 78: 671–672. https://doi.org/10.1378/chest.78.4.671.

[19] Ebisawa K, Yamada N, Okada S, Suzuki Y, Satoh A, Kobayashi M, et al. Combined Legionella and Escherichia coli lung infection after a tsunami disaster. Internal Medicine. 2011; 50: 2233–2236. https://doi.org/10.2169/internalmedicine.50.5800.

[20] Descours G, Tellini C, Flamens C, Philit F, Celard M, Etienne J, et al. Legionellosis and lung abscesses: contribution of legionella quantitative real-time PCR to an adapted followup. Case Reports in Infectious Diseases. 2013; 2013: 190183. https://doi.org/10.1155/2013/190183.

[21] Serio G, Fortarezza F, Pezzuto F, Santacroce L, Nazzaro P, Bellitti E, et al. Legionnaires' disease arising with hirsutism: case report of an extremely confusing event. BMC Infectious Diseases. 2021; 21: 532. https://doi.org/10.1186/s12879-021-06247-9.

[22] Flint KL, Lloyd MR, van den Berg P, Kanjee Z. *Legionella* pulmonary abscess and pleural space infection in an immunocompetent patient. BMJ Case Reports. 2021; 14: e243026. https://doi.org/10.1136/bcr-2021-243026.

[23] Edwards D, Finlayson DM. Legionnaires' disease causing severe lung abscesses. Canadian Medical Association Journal. 1980; 123: 524–527.

[24] Dagan A, Epstein D, Mahagneh A, Nashashibi J, Geffen Y, Neuberger A, et al. Community-acquired versus nosocomial Legionella pneumonia: factors associated with Legionella-related mortality. European Journal of Clinical Microbiology & Infectious Diseases. 2021; 40: 1419–1426. https://doi.org/10.1007/s10096-021-04172-y.