Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522661

Epidemiology, Prevention, and Public Health Management of Legionnaires' Disease-An Updated Review for Healthcare Professionals.

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Abstract

Background: Legionnaires' disease (LD), a severe atypical pneumonia caused primarily by *Legionella pneumophila*, continues to pose significant public health challenges due to underdiagnosis, environmental persistence, and high morbidity and mortality.
Aim: This review aims to provide healthcare professionals with an updated synthesis of the epidemiology, pathophysiology, clinical features, diagnosis, management, and prevention of LD.
Methods: A comprehensive review of current literature and surveillance data was conducted, incorporating clinical, microbiological, and environmental findings related to LD presentations, diagnostic strategies, and therapeutic approaches.
Results: *Legionella* species remain widely distributed in natural and man-made water systems, with serogroup 1 of *L. pneumophila* responsible for most human infections LD often presents with fever, respiratory symptoms, and extrapulmonary manifestations such as gastrointestinal and neurological signs. Diagnosis requires a combination of urinary antigen testing, cultures, and imaging. Fluoroquinolones and macrolides represent first-line therapy, with early treatment significantly improving outcomes Complications include respiratory failure, shock, renal injury, and multiorgan dysfunction, particularly in high-risk populations
Conclusion: Early recognition, targeted antimicrobial therapy, and robust water-system management are crucial to reducing LD-related morbidity and mortality.
Keywords: Legionnaires' disease, *Legionella pneumophila*, atypical pneumonia, epidemiology, diagnosis, management, water systems.

Introduction

Legionellosis refers to human infections caused by bacteria belonging to the genus *Legionella*. Clinically, legionellosis manifests primarily in two forms: Pontiac fever, a self-limited, nonpneumonic, influenza-like illness, and Legionnaires' disease (LD), a severe, often life-threatening atypical pneumonia [1]. Legionnaires' disease is most frequently caused by *Legionella pneumophila*, which was first identified following a significant outbreak at an American Legion convention in Philadelphia, United States, in 1976. The outbreak affected 182 individuals and resulted in 29 deaths, and subsequent investigation traced the source of infection to a contaminated hotel air-conditioning system [2][3]. Despite recognition of *Legionella* as a pathogenic organism for over four decades, legionellosis—particularly LD—remains underdiagnosed and underreported, and the true global incidence is uncertain. Recent decades, however, have seen increasing reports of LD, a trend attributed to heightened clinical awareness, improvements in diagnostic techniques, and strengthened surveillance systems [4]. Epidemiological studies consistently identify *Legionella* species as one of the most frequent

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Receive Date: 07 December 2025, Revise Date: 29 December 2025, Accept Date: 31 December 2025

etiologies of severe community-acquired pneumonia, second only to *Streptococcus pneumoniae* [5]. *Legionella pneumophila* can induce rapidly progressive atypical pneumonia, frequently resulting in complications such as acute respiratory failure, septic shock, acute renal impairment, and multiorgan dysfunction within a short timeframe. Hospitalization is nearly universal among patients diagnosed with LD, with approximately one-third requiring intensive care support. Mortality rates range from 4% to 40%, depending on patient comorbidities, timeliness of diagnosis, and adequacy of treatment [6][7]. Legionnaires' disease represents a persistent and growing global health concern. Healthcare professionals must maintain a high index of suspicion for LD in patients presenting with compatible clinical features, as early recognition and prompt initiation of targeted therapy are critical determinants of outcomes. Effective management requires a multidisciplinary approach that integrates clinical evaluation, microbiological diagnostics, and supportive care interventions. This review synthesizes current knowledge regarding the epidemiology, clinical presentation, diagnostic strategies, management, and prevention of legionellosis, with particular emphasis on LD, highlighting the essential role of coordinated healthcare delivery in improving patient prognosis.

Etiology

Legionella species are widely distributed, aerobic, gram-negative, intracellular, motile bacilli that serve as the causative agents of legionellosis across the globe. Since the identification of *Legionella* in 1976 as the pathogen responsible for the atypical pneumonia outbreak now recognized as Legionnaires' disease (LD), over 60 distinct species and more than 70 serogroups have been described. Among these, *Legionella pneumophila* remains the predominant species implicated in human infections, accounting for the majority of legionellosis cases [1]. Within *L. pneumophila*, serotypes 1 through 6 are responsible for most infections in humans. Other species, including *L. micdadei*, *L. bozemanae*, *L. longbeachae*, and *L. dumoffii*, contribute to a smaller proportion of cases, collectively representing approximately 10% of human infections with Legionella-related pneumonia [8][9]. These organisms are ubiquitously present in aquatic and terrestrial environments, including natural water bodies such as rivers and ponds, soil, and man-made water systems like cooling towers, plumbing networks, and potable water supplies [10]. Once *Legionella* establishes colonization within an artificial water system, eradication becomes particularly challenging. The bacteria are capable of forming biofilms, which protect them from environmental stressors and standard disinfection measures, thereby enhancing their persistence and long-term survival within water systems [11]. These characteristics contribute to the ongoing risk of human exposure and outbreaks, especially in settings with complex water systems, including hospitals, hotels, and industrial facilities. The combination of environmental ubiquity, biofilm formation, and diverse species capable of causing disease underscores the complex epidemiology and resilience of *Legionella* spp.

Epidemiology

Legionella species are widely distributed environmental bacteria that have the capacity to infect humans through multiple transmission pathways. The term legionellosis encompasses infections caused by these organisms, which can range from the mild, self-limiting, influenza-like syndrome known as Pontiac fever to the more severe and potentially life-threatening Legionnaires' disease (LD), an atypical pneumonia. LD can manifest as community-acquired or healthcare-associated infections and primarily affects individuals with predisposing risk factors or compromised host defenses [7]. Since its initial identification in 1976, over 60 species of *Legionella* and more than 70 serogroups have been characterized. Among these, *Legionella pneumophila*, particularly serogroup 1, is the most frequently associated with human disease worldwide. In certain geographic regions, such as Australia and New Zealand, *L. longbeachae* is the predominant species causing legionellosis [1][12]. Epidemiological studies have consistently identified several risk factors for LD, including male sex, advanced age, cigarette smoking, immunosuppression, malignancy, alcohol misuse, iron overload, anti-tumor necrosis factor-α therapy, and residence in socioeconomically disadvantaged areas [13]. *Legionella* organisms are naturally present in both aquatic and terrestrial environments, including soil, rivers, ponds, and man-made water systems such as building water supplies, cooling towers, and plumbing networks [10]. Aerosol-generating devices using contaminated water represent a primary route for human exposure. Showers, faucets, whirlpool spas, and mechanical ventilators have been repeatedly implicated in the dissemination of *Legionella* aerosols [14]. Once colonization occurs within a water system, eradication proves challenging due to the bacteria's ability to form biofilms, which confer protection against standard disinfection measures and allow persistence over prolonged periods [11]. Seasonal fluctuations in incidence have been observed, with higher rates reported during periods of increased rainfall and elevated ambient temperatures, suggesting an environmental component to the disease dynamics [15].

Human-to-human transmission of *Legionella* is extremely rare, and there is no evidence supporting zoonotic spread. The predominant mode of transmission occurs through inhalation of aerosols containing the bacteria, which allows entry into the lower respiratory tract. Following inhalation, *L. pneumophila* invades alveolar macrophages, triggering infection and subsequent pulmonary inflammation [16][17]. Additional transmission

pathways include micro-aspiration of contaminated fluids, particularly in hospitalized patients with predisposing conditions for aspiration, emphasizing the importance of clinical vigilance in healthcare settings. Distinguishing between community-acquired and healthcare-associated LD is critical for both epidemiological tracking and infection control. Patients who develop LD after being hospitalized for ten or more days before symptom onset are classified as having definite hospital-acquired pneumonia (HAP) LD. HAP LD frequently occurs in clusters due to point-source exposures, such as contaminated water systems or medical equipment, necessitating prompt identification and targeted infection control measures to prevent ongoing transmission [18][19]. Mortality in LD is influenced by multiple factors, including the pneumonia severity index and nosocomial acquisition. Patients with healthcare-associated LD are at significantly higher risk of death compared with those with community-acquired disease. Contributing factors include delayed recognition due to atypical clinical presentations, postponed initiation of effective antimicrobial therapy, and the higher prevalence of comorbidities or immunocompromised status among hospitalized patients [20]. In addition to mortality, LD frequently results in severe morbidity, requiring intensive care unit admission in 20% to 27% of hospitalized cases. Complications may include respiratory failure, shock, acute renal injury, and multiorgan dysfunction, often developing rapidly within a matter of days [6][7][20].

Despite increased recognition over recent decades, LD remains underdiagnosed and underreported, and its true global incidence is difficult to determine. Enhanced clinical awareness, improved diagnostic methodologies, and strengthened surveillance systems have contributed to the apparent rise in reported cases worldwide [4]. *Legionella* spp are recognized as among the leading causes of severe community-acquired pneumonia (CAP), although *Streptococcus pneumoniae* remains the most common etiology overall [5]. A recent systematic review of seroprevalence studies revealed substantial heterogeneity across different populations, with an overall pooled prevalence of 13.7% (95% CI: 11.3–16.5%), highlighting widespread exposure and variable immunity in human populations [1]. Epidemiologically, the majority of LD cases are sporadic, with more than 70% classified as community-acquired. Other cases are associated with travel or healthcare exposure. LD accounts for approximately 2% to 15% of all CAP cases, ranking among the four most common causative pathogens [7][21]. The severity of *L. pneumophila* infection stems from its potential to precipitate a rapidly progressive atypical pneumonia, which can advance to respiratory failure, shock, renal injury, and multiorgan dysfunction. Intensive care admission remains frequent, and mortality rates range from 4% to 40%, reflecting both host and pathogen factors [6][7][20]. Understanding the epidemiology of LD is essential for public health surveillance, infection control, and clinical management. Identifying environmental sources of exposure, recognizing high-risk populations, and maintaining a high index of suspicion in patients presenting with compatible symptoms are critical strategies for early diagnosis and intervention. Public health measures, including routine monitoring of water systems, outbreak investigations, and the implementation of environmental controls, are pivotal in reducing the incidence and impact of Legionella infections globally.

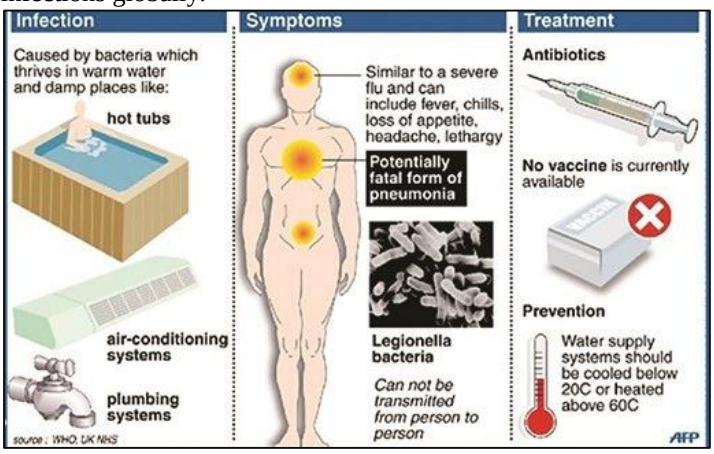

Fig. 1: Legionnaires' disease (LD).

Pathophysiology

Legionnaires' disease (LD) is predominantly caused by *Legionella pneumophila*, a gram-negative, facultative intracellular bacterium that thrives in aquatic environments. Infection occurs primarily through inhalation or micro-aspiration of aerosols generated from contaminated water sources, including showers, faucets, cooling towers, and respiratory equipment [16]. Once the bacterium reaches the alveoli, it is phagocytosed by alveolar macrophages. However, rather than being destroyed, *L. pneumophila* subverts the macrophage's bactericidal mechanisms, creating a protected intracellular niche for replication [22]. This intracellular lifestyle is facilitated by several virulence strategies, including type IV secretion systems, flagella, fimbriae, and iron-acquisition systems, which collectively enable the bacterium to evade host defenses and sustain replication within host cells [11]. Host susceptibility to infection is influenced by factors that compromise innate or adaptive immunity. Older age, cigarette smoking, chronic lung diseases such as chronic obstructive pulmonary disease, immunosuppressive therapies, and comorbidities like diabetes or malignancy increase the likelihood of symptomatic infection. These factors diminish the capacity of immune cells to respond effectively to *Legionella*, allowing bacterial proliferation and establishment of pulmonary disease. The pathophysiologic course of severe LD involves an initial hyperinflammatory phase characterized by extensive recruitment of neutrophils, macrophages, and lymphocytes to the site of infection. This

inflammatory cascade leads to alveolar and capillary injury, resulting in exudation, alveolar consolidation, and disruption of normal gas exchange. Histopathologic examination of affected lung tissue reveals diffuse alveolar damage, necrosis, neutrophilic infiltration, and involvement of alveolar ducts and respiratory bronchioles [24]. Following this hyperinflammatory response, patients may progress to a state of immunoparalysis, wherein immune cells exhibit reduced responsiveness to subsequent stimulation, further impairing the host's ability to control bacterial replication and increasing vulnerability to secondary infections [16][23]. The combination of bacterial virulence, intracellular replication, and host immune dysregulation accounts for the clinical severity observed in LD. Compromised gas exchange, inflammatory alveolar damage, and systemic immune responses contribute to the development of respiratory failure, shock, and multi-organ dysfunction in severe cases. Understanding the complex interplay between *L. pneumophila* virulence and host immune response is critical for guiding early diagnosis, risk stratification, and targeted therapeutic interventions.

Histopathology

Histopathological examination of lung tissue in patients with Legionnaires' disease demonstrates predominant involvement of the terminal bronchioles and adjacent alveoli. The alveolar spaces are typically filled with exudates comprising fibrin, proteinaceous material, and cellular debris, accompanied by septal thickening resulting from interstitial edema. *Legionella pneumophila* primarily replicates within alveolar macrophages, which serve as intracellular reservoirs, allowing the bacterium to evade host immune responses while propagating local infection [16][17]. The destruction of infected macrophages contributes to necrotic injury of the pulmonary parenchyma, which can be extensive in severe cases. Confluent lobular or lobar pneumonia is commonly observed, and abscess formation may occur, particularly in immunocompromised patients or those with delayed diagnosis. Histologically, lung tissue often reveals a dense leukocytoclastic inflammatory infiltrate, predominantly consisting of neutrophils and macrophages, which surrounds and infiltrates affected alveoli. Small vessel involvement is frequent, manifesting as "septic" vasculitis, characterized by endothelial injury, fibrinoid necrosis, and perivascular infiltration. Coagulative necrosis of pulmonary tissue and focal septal disruption are additional features, contributing to impaired gas exchange and ventilation [25]. Although these histopathological findings are characteristic of Legionella infection, they are not pathognomonic and may overlap with other atypical bacterial pneumonias or severe viral infections. In severe cases, the inflammatory response may extend to alveolar ducts and respiratory bronchioles, producing widespread consolidation and compromising oxygenation. The combination of intracellular bacterial replication, necrotic alveolar injury, and intense neutrophilic inflammation underlies the rapid progression and severity of the disease. Recognition of these histopathological features can support clinical diagnosis, guide therapeutic interventions, and provide insight into the mechanisms of lung injury in Legionnaires' disease, particularly in patients with comorbidities or immunosuppression that exacerbate disease progression [16][17][25].

History and Physical

A thorough history is essential in patients suspected of having Legionnaires' disease. Key elements include recent exposure to potential sources of contaminated water, such as showers, hot tubs, fountains, cooling towers, or medical devices within healthcare settings. Travel history is critical, especially in individuals staying in hotels, cruise ships, or healthcare facilities, as these environments have been linked to outbreaks. Occupational exposure among maintenance personnel, plumbers, or workers involved in water system management also increases risk. Symptom onset typically occurs 2 to 10 days following exposure, often presenting insidiously with fever, chills, malaise, and generalized myalgias [16][26]. Respiratory manifestations frequently include dry cough, dyspnea, and pleuritic chest pain, while extrapulmonary features may encompass gastrointestinal symptoms such as diarrhea, nausea, vomiting, and abdominal discomfort. Neurological involvement, though less common, can present as headache, confusion, or ataxia. Patients with underlying chronic lung disease, diabetes, immunosuppression, or recent hospitalization are particularly vulnerable to more severe disease. Clinicians should suspect Legionella infection in cases of community-acquired pneumonia unresponsive to β-lactam antibiotics or when extrapulmonary manifestations are prominent [27]. On physical examination, patients often appear acutely ill and febrile, sometimes displaying relative bradycardia. Respiratory findings may include crackles or rales, although overt lobar consolidation may be absent. Severe disease can manifest with tachypnea, hypoxia, or cyanosis. Additional findings may include confusion, focal neurological deficits, or abdominal tenderness, and less commonly, dermatologic or musculoskeletal signs. Cardiovascular examination is usually unremarkable, although myocarditis or arrhythmias can occasionally be observed. Awareness of these characteristic clinical features, in conjunction with detailed exposure history, is crucial for prompt recognition, early diagnostic evaluation, and initiation of targeted therapy in Legionnaires' disease [16].

Evaluation

The diagnosis of Legionnaires' disease (LD) requires an integrative approach that combines epidemiologic context, clinical assessment, laboratory

evaluation, and radiologic imaging, as the clinical manifestations are often nonspecific and mimic those of other pneumonias [27]. Clinicians should maintain a high index of suspicion for LD in patients presenting with community-acquired pneumonia (CAP), particularly in severe or rapidly progressive cases, because early identification and initiation of targeted therapy are strongly associated with improved clinical outcomes [27]. Consideration of patient-related and environmental risk factors is critical. Key epidemiological elements include recent hospitalization, exposure during known outbreaks, occupational or travel history, and underlying comorbidities that increase susceptibility. Environmental and seasonal trends are particularly relevant, as Legionella infections demonstrate a well-established correlation with rainfall, temperature, and seasonality, which distinguishes it from other CAP pathogens [26]. A supportive clinical sign is relative bradycardia in febrile patients who are not on bradycardia-inducing medications and do not have pacemakers; this can reinforce suspicion for LD [28]. Laboratory evaluation is typically nonspecific but can provide supportive evidence for LD. Common findings include hyponatremia, hypophosphatemia, elevated creatine kinase levels secondary to rhabdomyolysis, renal impairment, microscopic hematuria, leukocytosis with relative lymphopenia, and elevated inflammatory markers such as ferritin or C-reactive protein [26][27]. While these abnormalities are not diagnostic, their presence in the appropriate clinical and epidemiological context can raise suspicion for LD and prompt further confirmatory testing.

Microbiological confirmation is critical for definitive diagnosis. The urinary antigen test is widely utilized due to its rapid results and high sensitivity; however, it primarily detects *Legionella pneumophila* serogroup 1, which accounts for the majority of cases [26][27]. Culture of respiratory specimens—including sputum and bronchoalveolar lavage—remains the gold standard, as it allows definitive species identification and epidemiological typing. Limitations include low sensitivity, the requirement for specialized culture media, and occasionally the need for invasive specimen collection. Direct fluorescent antibody staining is less commonly used due to technical complexity and reduced sensitivity [27]. Optimally, a combination of diagnostic modalities enhances sensitivity and enables timely and accurate identification of the pathogen, facilitating prompt and effective management [26]. Radiologic evaluation plays a supportive but essential role in the diagnosis of LD. Chest radiographs usually demonstrate early patchy alveolar infiltrates with consolidation, often predominating in the lower lobes, though any lobe may be involved [26][27]. Rapid progression to bilateral interstitial pneumonia can occur, and pleural effusions are observed in approximately 15% to 50% of cases. Radiographic abnormalities may progress even during the early phase of clinical improvement, reflecting ongoing pulmonary inflammation [26][27]. Cavitation and abscess formation are uncommon but can develop in immunocompromised patients, emphasizing the importance of close monitoring in high-risk populations [26]. This multifaceted evaluation—including epidemiologic assessment, clinical suspicion, laboratory analysis, microbiological confirmation, and radiologic imaging—forms the cornerstone for accurate diagnosis of LD and underpins timely initiation of targeted antimicrobial therapy.

Treatment / Management

Management of Legionnaires' disease (LD) requires prompt recognition and initiation of targeted antimicrobial therapy, particularly in patients hospitalized with severe community-acquired pneumonia (CAP). Early diagnostic testing for *Legionella pneumophila* is strongly recommended, as delays in appropriate therapy are associated with worsened clinical outcomes, including higher rates of intensive care unit (ICU) admission and mortality [27][29][30][31]. Empirical therapy for severe CAP should include coverage for Legionella species due to the nonspecific clinical presentation and potential rapid progression of disease. Fluoroquinolones, such as levofloxacin or moxifloxacin, and macrolides, including azithromycin, constitute first-line treatment options. Evidence from recent meta-analyses indicates that fluoroquinolones and macrolides are similarly effective in reducing mortality, ICU length of stay, and clinical complications, though the choice may depend on patient comorbidities, drug interactions, and local resistance patterns [27][29][30][31]. In addition to antibiotic therapy, supportive care is essential. Patients with severe pneumonia may require supplemental oxygen, ventilatory support, hemodynamic stabilization, or renal replacement therapy in cases of multiorgan dysfunction. Adjunctive measures, such as fluid resuscitation and careful monitoring of electrolytes, are critical, particularly for patients presenting with hyponatremia or rhabdomyolysis. Immunocompromised patients or those with comorbidities may benefit from closer monitoring due to higher risks of rapid clinical deterioration. The management strategy should also consider the source of infection, implementing infection control measures to prevent nosocomial transmission when relevant. Early empiric therapy with coverage against *Legionella* is particularly important because this pathogen can lead to rapidly progressive atypical pneumonia, systemic complications, and high mortality if untreated. Combining antimicrobial therapy with supportive and intensive care interventions ensures the best possible outcomes. Clinicians should maintain vigilance for treatment response, adjust therapy based on microbiological confirmation, and anticipate complications to optimize recovery and reduce the likelihood of adverse outcomes [27][29][30][31].

Differential Diagnosis

The differential diagnosis of Legionnaires' disease encompasses a range of infectious and noninfectious conditions that present with pneumonia-like syndromes. Typical bacterial pneumonias caused by pathogens such as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Staphylococcus aureus* usually manifest with acute onset of symptoms, productive cough, pleuritic chest pain, and lobar consolidation visible on imaging studies [9][27]. These infections often have a relatively predictable clinical course and respond to standard β-lactam antibiotics, distinguishing them from LD, which frequently demonstrates atypical presentations. Atypical bacterial pathogens, including *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*, generally produce a milder, subacute respiratory illness accompanied by extrapulmonary manifestations, such as pharyngitis, hoarseness, hemolytic anemia, or bullous myringitis [9][27]. Viral pneumonias, including influenza and SARS-CoV-2, are characterized by predominant upper respiratory tract symptoms, generalized myalgias, fatigue, and, in some cases, anosmia or ageusia. These infections often follow seasonal or epidemic patterns, which can assist in distinguishing them from sporadic LD cases [26]. Legionnaires' disease, however, is frequently associated with systemic and extrapulmonary manifestations that are less common in other respiratory infections. High fever, relative bradycardia, gastrointestinal disturbances such as diarrhea and abdominal pain, and neurological symptoms including confusion, headache, or encephalopathy are hallmark features. Laboratory abnormalities, such as elevated liver transaminases or hyponatremia, further support the diagnosis [26]. Noninfectious etiologies, such as pulmonary embolism, chemical pneumonitis, or autoimmune pneumonitis, may also present with fever, dyspnea, and infiltrates on imaging, highlighting the need for careful evaluation. Accurate differentiation relies on integrating exposure history, clinical signs, laboratory parameters, radiologic findings, and microbiological testing. Travel history, hospital exposures, and environmental risk factors, including recent use of aerosol-generating water systems, can further guide the clinician in narrowing the differential diagnosis and identifying LD [26]. Recognizing these distinguishing features ensures timely initiation of appropriate therapy, which is critical given the rapid progression and potential severity of LD.

Prognosis

The prognosis of Legionnaires' disease varies widely and depends on multiple patient-related and disease-specific factors. Both the pneumonia severity index and nosocomial acquisition are independent predictors of increased mortality in Legionella pneumonia [20]. Patients with hospital-acquired LD are at significantly higher risk of adverse outcomes compared with those with community-acquired infections due to delayed recognition, atypical clinical presentation, and postponement of effective antimicrobial therapy. Furthermore, individuals with nosocomial infection are more likely to be immunocompromised, have multiple comorbidities, or require invasive procedures, all of which further exacerbate morbidity and mortality [20]. Clinical progression can be rapid, with severe atypical pneumonia leading to respiratory failure, shock, acute renal injury, and multiorgan dysfunction within days. ICU admission is common, ranging from 20% to 27% of hospitalized patients, and overall mortality spans 4% to 40%, depending on the severity of illness, timeliness of therapy, and presence of underlying conditions [6][7][20]. Early diagnosis and initiation of appropriate antimicrobial therapy are therefore critical to improving outcomes. Long-term prognosis also depends on host factors, including age, preexisting chronic lung disease, diabetes, or immunosuppressive states. Patients who survive severe LD may experience prolonged convalescence and residual pulmonary dysfunction. The extent of organ involvement, rapidity of disease progression, and the presence of extrapulmonary manifestations such as renal, hepatic, or neurological complications further influence recovery trajectories [6][7][20]. Overall, while effective early management can significantly reduce mortality, the disease remains associated with substantial risk, emphasizing the importance of rapid recognition, timely intervention, and comprehensive supportive care.

Complications

Legionnaires' disease (LD) can extend beyond pulmonary involvement, producing a range of extrapulmonary complications that increase morbidity and can be life-threatening. Cardiovascular complications include myopericarditis and, more rarely, endocarditis, which may manifest with chest pain, arrhythmias, or signs of heart failure. Neurological involvement can present as encephalitis, brain abscesses, or cerebellar ataxia, leading to confusion, impaired coordination, or focal neurological deficits. Gastrointestinal complications are also notable; patients may develop pancreatitis, colitis, or hepatic and splenic involvement, which can exacerbate systemic illness and prolong recovery. Musculoskeletal manifestations, such as reactive arthritis, may occur during or after the acute phase of infection. Dermatologic complications, including cellulitis and, in rare cases, necrotizing fasciitis, reflect systemic bacterial dissemination or secondary infection. Hematologic involvement may include disseminated intravascular coagulation, particularly in severe or fulminant cases, contributing to multiorgan failure. These extrapulmonary effects underscore the importance of early recognition, aggressive supportive care, and multidisciplinary management to reduce

morbidity and prevent progression to multiorgan dysfunction [16][26].

Consultations

The management of LD often requires a multidisciplinary approach tailored to disease severity and patient comorbidities. Mild or early cases are usually managed by the primary clinician or hospitalist, with infectious disease specialists providing guidance on diagnosis and optimal antibiotic selection. Pulmonology may be consulted for patients with preexisting chronic lung disease or significant respiratory compromise. In cases of severe or rapidly progressing LD, consultations expand to include critical care specialists, as patients may require intensive support, including mechanical ventilation or extracorporeal membrane oxygenation. Infectious disease clinicians oversee antimicrobial therapy and advise on pathogen-specific management strategies. Cardiology input is warranted in cases of myocarditis, arrhythmias, or heart failure, whereas nephrology may be involved in managing acute kidney injury, rhabdomyolysis, or fluid-electrolyte disturbances. High-risk populations, including older adults and immunocompromised patients, may benefit from geriatric or oncology consultation to optimize overall care. Coordination among specialists ensures timely interventions, reduces complications, and improves patient outcomes in severe Legionella infections [27][29].

Patient Education

Patient education is essential in preventing Legionnaires' disease and mitigating exposure risk. Individuals should be informed that *Legionella* bacteria proliferate in warm, stagnant water and are primarily transmitted through inhalation of contaminated aerosols. High-risk populations include older adults, smokers, and those with chronic illnesses or compromised immune systems. Preventive measures focus on proper maintenance of water systems, including regular cleaning and disinfection of showers, faucets, and humidifiers, avoiding stagnant water, and employing point-of-use filters where appropriate. Patients should be advised to seek immediate medical attention if they experience fever, cough, dyspnea, or gastrointestinal symptoms following potential exposure to contaminated water sources. Maintaining overall health through smoking cessation, proper nutrition, and regular exercise may further reduce susceptibility. Effective patient education empowers individuals to take proactive steps in reducing exposure risk while promoting early recognition and timely intervention if symptoms arise [26][27].

Other Issues

Preventing Legionnaires' disease relies on rigorous water system management and awareness of environmental risk factors. Maintaining appropriate water temperatures, regularly flushing unused taps, and disinfecting cooling towers, plumbing, and other water reservoirs are critical strategies. Point-of-use filters provide additional protection for high-risk individuals. Common challenges in managing LD include missed early diagnosis due to nonspecific symptoms, inadequate maintenance of water systems, failure to identify the infection source during outbreaks, and administration of antibiotics lacking activity against *Legionella*. Clinicians and public health authorities must be alert to these challenges, ensuring that high-risk populations are educated and that water systems are routinely monitored and maintained. Prompt recognition, targeted treatment, and preventive strategies collectively reduce the incidence of LD and minimize severe disease outcomes [26][27].

Enhancing Healthcare Team Outcomes

Effective management of LD requires coordinated efforts across healthcare teams to reduce morbidity and mortality associated with this rapidly progressing infection. Clinicians should maintain high suspicion for Legionella infection in patients with severe CAP or atypical presentations and work closely with infectious disease, critical care, pulmonology, and nephrology specialists when complications arise. Public health authorities play a vital role in detecting cases, identifying environmental sources, and preventing outbreaks. In healthcare-associated cases, collaboration with hospital infection control teams is essential, particularly after changes in water systems or positive environmental testing for Legionella. Interdisciplinary communication ensures that patient care is optimized, infection control measures are implemented promptly, and outbreaks are contained. By combining clinical vigilance, environmental management, and public health interventions, healthcare teams can significantly improve outcomes, reduce transmission, and enhance overall preparedness for Legionella-associated illness [27][29].

Conclusion:

Legionnaires' disease remains a globally significant respiratory infection due to its environmental resilience, diverse clinical manifestations, and potential for rapid clinical deterioration. The review underscores that *Legionella pneumophila*, particularly serogroup 1, continues to dominate reported cases and poses diagnostic challenges because of its nonspecific presentation and overlap with other infectious and noninfectious pneumonias. The disease's severity lies in its ability to cause rapidly progressive atypical pneumonia, leading to complications such as respiratory failure, shock, renal injury, and multiorgan dysfunction, especially among hospitalized, elderly, or immunocompromised patients. Early clinical suspicion remains central to improving outcomes, particularly in patients presenting with severe community-acquired pneumonia or when epidemiologic risk factors suggest possible exposure. The availability of rapid urinary antigen testing and improved microbiological methods enhances early detection, but these tools must be

paired with timely initiation of effective antimicrobial therapy to reduce mortality. Beyond clinical management, prevention remains essential; maintaining safe water systems, regular monitoring, and effective disinfection strategies are pivotal in avoiding environmental colonization and outbreaks. Strengthening interdisciplinary coordination among clinicians, infection control teams, and public health authorities is crucial to mitigating the burden of Legionnaires' disease and improving patient and community health outcomes

References:

1. Graham FF, Hales S, White PS, Baker MG. Review Global seroprevalence of legionellosis - a systematic review and meta-analysis. Sci Rep. 2020 Apr 30;10(1):7337.
2. Fraser DW, Tsai TR, Orenstein W, Parkin WE, Beecham HJ, Sharrar RG, Harris J, Mallison GF, Martin SM, McDade JE, Shepard CC, Brachman PS. Legionnaires' disease: description of an epidemic of pneumonia. N Engl J Med. 1977 Dec 01;297(22):1189-97.
3. McDade JE, Shepard CC, Fraser DW, Tsai TR, Redus MA, Dowdle WR. Legionnaires' disease: isolation of a bacterium and demonstration of its role in other respiratory disease. N Engl J Med. 1977 Dec 01;297(22):1197-203.
4. Barskey AE, Derado G, Edens C. Rising Incidence of Legionnaires' Disease and Associated Epidemiologic Patterns, United States, 1992-2018. Emerg Infect Dis. 2022 Mar;28(3):527-538.
5. Martin-Loeches I, Torres A, Nagavci B, Aliberti S, Antonelli M, Bassetti M, Bos LD, Chalmers JD, Derde L, de Waele J, Garnacho-Montero J, Kollef M, Luna CM, Menendez R, Niederman MS, Ponomarev D, Restrepo MI, Rigau D, Schultz MJ, Weiss E, Welte T, Wunderink R. ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia. Intensive Care Med. 2023 Jun;49(6):615-632.
6. 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. Clin Infect Dis. 2020 May 23;70(11):2428-2431.
7. Viasus D, Gaia V, Manzur-Barbur C, Carratalà J. Legionnaires' Disease: Update on Diagnosis and Treatment. Infect Dis Ther. 2022 Jun;11(3):973-986.
8. Miyashita N. Legionella prediction score. Respir Investig. 2024 Mar;62(2):305-306.
9. Cunha BA. Legionnaires' disease: clinical differentiation from typical and other atypical pneumonias. Infect Dis Clin North Am. 2010 Mar;24(1):73-105.
10. Niculita-Hirzel H, Vanhove AS, Leclerc L, Girardot F, Pourchez J, Allegra S. Risk Exposure to *Legionella pneumophila* during Showering: The Difference between a Classical and a Water Saving Shower System. Int J Environ Res Public Health. 2022 Mar 10;19(6)
11. Palusińska-Szysz M, Simões M. Editorial: *Legionella pneumophila*-transmission, pathogenesis, host-pathogen interaction, prevention and treatment. Front Microbiol. 2024;15:1364620.
12. Priest PC, Slow S, Chambers ST, Cameron CM, Balm MN, Beale MW, Blackmore TK, Burns AD, Drinković D, Elvy JA, Everts RJ, Hammer DA, Huggan PJ, Mansell CJ, Raeder VM, Roberts SA, Robinson MC, Sathyendran V, Taylor SL, Thompson AW, Ussher JE, van der Linden AJ, Williams MJ, Podmore RG, Anderson TP, Barratt K, Mitchell JL, Harte DJ, Hope VT, Murdoch DR. The burden of Legionnaires' disease in New Zealand (LegiNZ): a national surveillance study. Lancet Infect Dis. 2019 Jul;19(7):770-777.
13. Ishikawa K, Nakamura T, Matsuo T, Kawai F, Murakami H, Aoki K, Nagasawa T, Uehara Y, Mori N. Clinical Presentation of Legionella pneumophila Serogroup 1-Associated Pneumonia and Diffuse Alveolar Hemorrhage: A Case Report and Literature Review. Am J Case Rep. 2022 Jul 12;23:e936309.
14. Rebellato S, Lee C, Gardner C, Kivilahti K, Wallace J, Hachborn D, Fenik J, Majury A, Kim J, Murphy A, Minnery J. Community *Legionella* outbreak linked to a cooling tower, 2022. Can Commun Dis Rep. 2023 Sep 01;49(9):380-386.
15. Kirpich A, Shishkin A, Lhewa P, Yang C, von Fricken ME, Norris MH, Weppelmann TA. An investigation of the seasonal relationships between meteorological factors, water quality, and sporadic cases of Legionnaires' disease in Washington, DC. Epidemiol Infect. 2023 May 15;151:e88.
16. Rello J, Allam C, Ruiz-Spinelli A, Jarraud S. Severe Legionnaires' disease. Ann Intensive Care. 2024 Apr 02;14(1):51.
17. Fu Y, Macwan V, Heineman RE, Terebiznik MR, Harrison RE. *Legionella pneumophila* Infection of Human Macrophages Retains Golgi Structure but Reduces O-Glycans. Pathogens. 2022 Aug 12;11(8)
18. Beauté J, Plachouras D, Sandin S, Giesecke J, Sparén P. Healthcare-Associated Legionnaires' Disease, Europe, 2008-2017. Emerg Infect Dis. 2020 Oct;26(10):2309-2318.
19. Soda EA, Barskey AE, Shah PP, Schrag S, Whitney CG, Arduino MJ, Reddy SC, Kunz JM, Hunter CM, Raphael BH, Cooley LA. Vital Signs: Health Care-Associated Legionnaires' Disease Surveillance Data from 20 States and a Large

Metropolitan Area - United States, 2015. MMWR Morb Mortal Wkly Rep. 2017 Jun 09;66(22):584-589.
20. Dagan A, Epstein D, Mahagneh A, Nashashibi J, Geffen Y, Neuberger A, Miller A. Community-acquired versus nosocomial Legionella pneumonia: factors associated with Legionella-related mortality. Eur J Clin Microbiol Infect Dis. 2021 Jul;40(7):1419-1426.
21. 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. Int J Environ Res Public Health. 2022 Feb 08;19(3)
22. Shames SR, Liu L, Havey JC, Schofield WB, Goodman AL, Roy CR. Multiple *Legionella pneumophila* effector virulence phenotypes revealed through high-throughput analysis of targeted mutant libraries. Proc Natl Acad Sci U S A. 2017 Nov 28;114(48):E10446-E10454.
23. Allam C, Mouton W, Testaert H, Ginevra C, Fessy N, Ibranosyan M, Descours G, Beraud L, Guillemot J, Chapalain A, Albert-Vega C, Richard JC, Argaud L, Friggeri A, Labeye V, Jamilloux Y, Freymond N, Venet F, Lina G, Doublet P, Ader F, Trouillet-Assant S, Jarraud S. Hyper-inflammatory profile and immunoparalysis in patients with severe Legionnaires' disease. Front Cell Infect Microbiol. 2023;13:1252515.
24. Lattimer GL, Rachman RA, Scarlato M. Legionnaires' disease pneumonia: histopathologic features and comparison with microbial and chemical pneumonias. Ann Clin Lab Sci. 1979 Sep-Oct;9(5):353-61.
25. Winn WC, Myerowitz RL. The pathology of the Legionella pneumonias. A review of 74 cases and the literature. Hum Pathol. 1981 May;12(5):401-22.
26. Bai L, Yang W, Li Y. Clinical and Laboratory Diagnosis of Legionella Pneumonia. Diagnostics (Basel). 2023 Jan 12;13(2)
27. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, Cooley LA, Dean NC, Fine MJ, Flanders SA, Griffin MR, Metersky ML, Musher DM, Restrepo MI, Whitney CG. Diagnosis and Treatment of Adults with Community-acquired Pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America. Am J Respir Crit Care Med. 2019 Oct 01;200(7):e45-e67.
28. Cunha BA. The diagnostic significance of relative bradycardia in infectious disease. Clin Microbiol Infect. 2000 Dec;6(12):633-4.
29. Jones BE, Ramirez JA, Oren E, Soni NJ, Sullivan LR, Restrepo MI, Musher DM, Erstad BL, Pickens C, Vaughn VM, Helgeson SA, Crothers K, Metlay JP, Bissell B, Cao B, Chalmers JD, Dela Cruz CS, Gendlina I, Hojat LS, Laguio-Vila M, Liang SY, Waterer GW, Paine M, Hawkins C, Wilson K. Diagnosis and Management of Community-acquired Pneumonia. An Official American Thoracic Society Clinical Practice Guideline. Am J Respir Crit Care Med. 2025 Jul 18;
30. Falcone M, Russo A, Tiseo G, Cesaretti M, Guarracino F, Menichetti F. Predictors of intensive care unit admission in patients with Legionella pneumonia: role of the time to appropriate antibiotic therapy. Infection. 2021 Apr;49(2):321-325.
31. Woodhead M, Blasi F, Ewig S, Garau J, Huchon G, Ieven M, Ortqvist A, Schaberg T, Torres A, van der Heijden G, Read R, Verheij TJ., Joint Taskforce of the European Respiratory Society and European Society for Clinical Microbiology and Infectious Diseases. Guidelines for the management of adult lower respiratory tract infections--full version. Clin Microbiol Infect. 2011 Nov;17 Suppl 6(Suppl 6):E1-59.