Legionella infection with renal and muscular complications: a clinical case with a focus on clinical aspects and imaging diagnostics

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ABSTRACT

Legionellosis is a serious respiratory infection caused by the bacterium *Legionella pneumophila*, transmitted through the inhalation of contaminated aerosol. The most common form is pneumonia, but rare complications such as rhabdomyolysis and acute kidney injury (AKI) may occur, increasing both morbidity and mortality. These require early recognition and targeted treatment.

A 54-year-old man with a history of chronic alcoholism presented to the Emergency Department with fever, diarrhea, and confusion. Vital signs were within normal limits, but laboratory tests revealed rhabdomyolysis and AKI with compensated metabolic acidosis. In the following days, renal function deteriorated, and muscle damage persisted. Chest computed tomography (CT) revealed bilateral pulmonary consolidations and pleural effusion, while brain CT showed chronic vascular leukoencephalopathy.

The diagnosis of legionellosis was confirmed by a positive urinary antigen test for *Legionella pneumophila* serogroup 1. The patient began targeted antibiotic therapy with azithromycin and levofloxacin, combined with corticosteroids. Due to worsening renal function, he underwent hemodialysis. During hospitalization, clinical and laboratory improvements were observed, with recovery of renal function and resolution of rhabdomyolysis, although pulmonary lesions persisted. This case highlights the importance of considering legionellosis in cases of pneumonia complicated by rhabdomyolysis and AKI. Timely diagnosis and appropriate treatment are crucial to prevent severe complications and improve outcomes.

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Introduction

Legionellosis is an infectious disease caused by aerobic, Gram-negative bacteria of the *Legionella* genus. The species most commonly involved in human cases is *Legionella pneumophila* (LP), which is the etiological agent of a severe pneumonia that can lead to significant systemic impairment and may have a fatal outcome. It is ubiquitous in both natural and artificial aquatic environments, such as domestic water systems, cooling towers, and air conditioning units. Transmission to humans occurs primarily through the inhalation of contaminated aerosols.

LP infection is recognized as a rare cause of rhabdomyolysis. It can be further complicated by acute renal failure, which may result from acute tubular necrosis (ATN) or acute tubulo-interstitial nephritis. This triad of pneumonia, renal

failure, and rhabdomyolysis is associated with high morbidity and mortality.

Case Report

A 54-year-old male patient, resident in an assisted living facility and a chronic alcoholic, with no other notable comorbidities, presented to the Emergency Department of our facility with confusion, fever, and diarrhea. Physical examination included the following. Blood pressure: 130/75 mmHg; heart rate: 90 bpm; temperature: 38.5°C; oxygen saturation: 95%; no peripheral edema; and chest examination: decreased breath sounds and mild dullness at the mid-to-lower left lung field.

Chest computed tomography (CT), performed without intravenous (IV) contrast administration, showed parenchymal consolidation in the left upper lobe and partially confluent amorphous patches of parenchymal consolidation in the right lower lobe; dense lamellar streaks in the right upper lobe; right apico-mid-basal pleural effusion (Figures 1 and 2).

Brain CT, also performed without IV contrast, revealed chronic hypoperfusion in the periventricular area (Figure 3). Abdominal ultrasound was unremarkable.

The patient also underwent blood cultures, stool cultures, Widal-Wright test, serologic testing for *Mycoplasma pneumoniae* antibodies, testing for *Escherichia coli* O157 toxin, *Clostridium difficile* A/B toxin, and urinary antigen testing for *Legionella*.

The clinical history, combined with the diagnosis of pneumonia complicated by rhabdomyolysis and acute kidney injury (AKI), raised a strong suspicion of legionellosis, which was promptly confirmed by a positive urinary antigen test for *Legionella* serogroup 1. This test has a sensitivity ranging from 70-100% and a specificity approaching 100%.

The patient therefore began targeted IV antibiotic therapy with azithromycin (500 mg/day IV for 8 days) and levofloxacin (500 mg/day IV for 12 days, followed by oral administration for an additional 8 days), combined with methylprednisolone 20 mg twice daily IV (discontinued after 5 days).

Due to significant renal impairment, despite adequate diuresis and hydration, hemodialysis was initiated. The patient underwent a total of 3 hemodialysis sessions. During hospi-

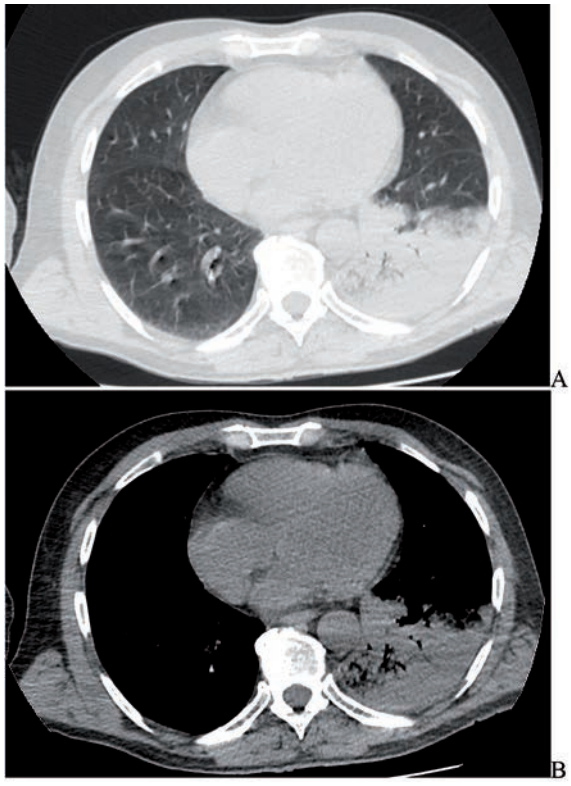

Figure 1. A,B) Presence of extensive parenchymal consolidation with associated air bronchogram in the left lower lobe (images dated June 15).

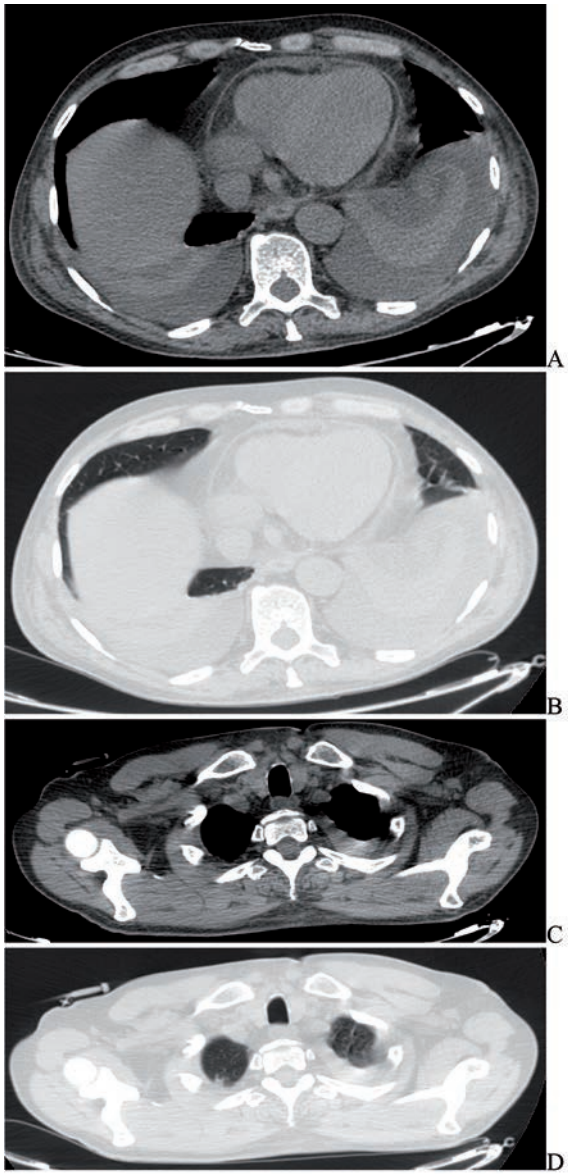

Figure 2. A-D) Onset of left apico-parieto-basal and right parieto-basal pleural effusion with passive atelectasis of the adjacent lung parenchyma. Pericardial effusion was also noted (June 26).

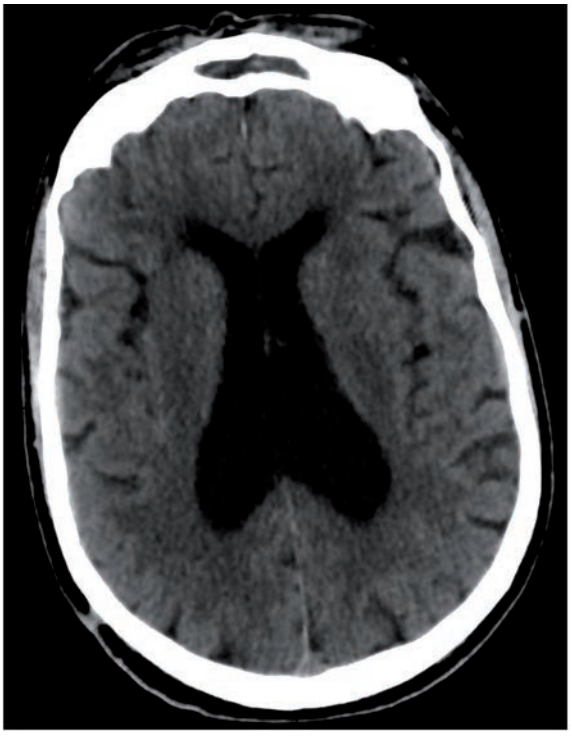

Figure 3. Presence of signs of chronic vascular leukoencephalopathy of hypoxic-ischemic origin, predominantly involving the periventricular and subcortical white matter.

talization, there was progressive improvement in clinical condition, cytolysis markers, and renal function parameters.

At discharge, the patient was in good overall clinical condition, although worsening of pulmonary lesions was observed on follow-up chest CT (performed without IV contrast). There was a good recovery of renal function and cytolysis markers, with the following lab values: blood urea nitrogen 52 mg/dL, creatinine 1.45 mg/dL, creatine kinase (CK) 30 U/L, lactate dehydrogenase 613 U/L, and C-reactive protein 4.7 mg/L.

Discussion

LP can cause a spectrum of illnesses ranging from Legionnaires' disease, a severe multisystem disorder characterized by pneumonia along with gastrointestinal, musculoskeletal, and neurological involvement, to Pontiac fever, a self-limiting flu-like illness.

LP accounts for only 2-9% of all cases of community-acquired pneumonia. Age, chronic lung diseases, smoking, and immunocompromised states are all known risk factors for the disease.

Rhabdomyolysis secondary to LP infection is a rare but potentially life-threatening complication of legionellosis.[1] It is a clinical syndrome characterized by the breakdown of skeletal muscle tissue and the release of intracellular components such as CK, myoglobin, potassium, and phosphate into the bloodstream. Among the infectious causes of rhabdomyolysis, infection with LP has been documented, although it remains an uncommon occurrence.

The pathogenesis of rhabdomyolysis associated with *Legionella* infection is not fully understood. Proposed mechanisms include direct invasion of skeletal muscle by the bacteria, the action of endotoxins, and an exaggerated systemic inflammatory response, leading to cellular injury and muscle necrosis.[2,3]

Endotoxins produced by LP may induce ischemic muscle damage through vasoconstriction of small muscular vessels, resulting in local hypoperfusion and ischemia, a mechanism also described in other severe bacterial infections.[3] This ischemic injury leads to muscle cell disintegration and the release of myoglobin, which is potentially nephrotoxic and may cause obstruction of the distal renal tubules by myoglobin casts.[2]

The hypothesis of direct myocyte invasion by LP is based on similarities with other intracellular pathogens; however, direct histopathological evidence remains limited.[4] Nonetheless, autopsy findings and renal biopsies have demonstrated the presence of myoglobin pigments within the tubules, confirming secondary muscle-renal pathogenesis.[3]

Massive release of myoglobin into the bloodstream results in its precipitation in renal tubules, leading to ATN and AKI.[4] Approximately 55% of patients affected by this triad (pneumonia, rhabdomyolysis, and AKI) require dialysis, and mortality may reach 51% in cases with AKI, compared to 15% in those without AKI.[5,6]

Early recognition of rhabdomyolysis in the context of legionellosis is essential to initiate appropriate antibiotic therapy and supportive renal care, which can significantly improve prognosis.[2]

From a clinical standpoint, patients may present with a combination of respiratory symptoms (fever, cough, dyspnea) typical of *Legionella* pneumonia, and systemic manifestations such as myalgia, muscle weakness, and dark-colored urine. Serum CK levels are typically elevated, sometimes exceeding 100,000 U/L, and myoglobinuria is frequently observed.[4,5]

Renal complications are common. The release of myoglobin can cause acute tubular injury and, in more severe cases, AKI, which may require the initiation of renal replacement therapy.[6,7] In immunocompromised patients, the risk of complications is even higher.[8]

Diagnosis is based on urinary antigen testing for *Legionella*, respiratory cultures, and molecular techniques such as polymerase chain reaction and, in selected cases, next-generation sequencing.[9,10]

Treatment involves the administration of antibiotics active against *Legionella* (macrolides and fluoroquinolones), along with intensive rehydration to prevent AKI.[11] The pathogenic mechanisms are not fully understood, but the main hypotheses include systemic toxic effects mediated by endotoxins and, less frequently, direct invasion of muscle tissue by the bacterium.[2]

Conclusions

The presented clinical case highlights the importance of a thorough medical history, which, when supported by clinical and laboratory findings, can guide the diagnostic and therapeutic process toward conditions that are not frequently encountered in daily clinical practice, but which, thanks to prompt and targeted treatment, can achieve a favorable and complete resolution.

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