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CASE REPORT

Community-Acquired Pneumonia with Acute Kidney Injury Complicated by Rhabdomyolysis-A Challenging Case Report in Resource Limit Setting

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Background: Community-acquired pneumonia (CAP) is a common infectious disease that can lead to complications such as rhabdomyolysis (RM), a rare but potentially life-threatening condition involving muscle breakdown. RM can further complicate the clinical course by causing acute kidney injury (AKI). We present a case of Community-acquired pneumonia with AKI complicated by rhabdomyolysis in a resource-limited setting.

Case Presentation: A 67-year-old male presented with high fever, cough, and shortness of breath. He had no significant medical history. On examination, he was febrile, tachypneic, and tachycardic, with right-sided lung crackles. Lab tests showed elevated inflammatory markers and impaired kidney function. Chest radiography revealed right upper lobe consolidation, confirming pneumonia and AKI. He was treated with fluids, antibiotics, and supportive care, but his condition worsened, requiring intensive care unit (ICU). In the ICU, dark urine and elevated creatine kinase confirmed rhabdomyolysis. After aggressive fluid therapy and antibiotics the patient improved over six days and was transferred to the ward. By day 10, he fully recovered and was discharged with follow-up.

Conclusion: This case underscores the importance of early recognition and prompt management of CAP complicated by AKI and rhabdomyolysis, even in resource-limited settings. Timely intervention can lead to favorable outcomes despite challenges.

Keywords: community-acquired pneumonia, acute kidney injury, rhabdomyolysis, resource limit setting

Introduction

Community-acquired pneumonia (CAP) is an inflammatory condition contracted outside of a hospital setting and is primarily characterized by symptoms such as fever, shortness of breath, cough, sputum production, and chest pain.[1] Community-acquired pneumonia is a major cause of hospitalization and mortality with significant healthcare costs and presents variably from mild outpatient illness to severe cases requiring intensive care, making early diagnosis and appropriate care level determination crucial for improving outcomes.[2]

Rhabdomyolysis (RM) is a clinical condition triggered by multiple factors that cause skeletal muscle damage and cell death, resulting in the breakdown of muscle cell membranes and the release of cellular contents into the bloodstream, which leads to disturbances in acid-base balance, electrolyte imbalances, myoglobin in the urine, and potentially acute kidney failure.[3] Rhabdomyolysis is commonly associated with trauma or muscle injury, it can also occur as a complication of severe Infections, including community-acquired pneumonia (CAP), are uncommon yet recognized triggers of rhabdomyolysis, pathogens such as Legionella pneumophila, which causes Legionnaires' disease, are particularly associated with this triad of CAP, rhabdomyolysis, and AKI.[4,5] Additionally, rhabdomyolysis is frequently

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overlooked in its early stages due to the absence of symptoms, and in clinical practice, mild rhabdomyolysis induced by community-acquired pneumonia (CAP) can be easily missed during examination.[6]

The combination of CAP, rhabdomyolysis, and AKI represents a serious clinical scenario, particularly in Resource-limited settings, where diagnostic and therapeutic options may be constrained. Here, we present the case of a 67-year-old male who developed acute kidney injury complicated by rhabdomyolysis as a result of severe community-acquired pneumonia in a resource-limited setting.

Case Presentation

A 67-Year-old male presented to the emergency department with complaints of intermittent high-grade fever that started two days ago relieved by antipyretics prior to admission, which was associated with productive cough and shortness of breath. He had no significant history or chronic illness. He denied recent travel. On examination, the patient looked ill, confused, febrile, had tachypnea, tachycardia, mild pallor, and no lower limb edema. The Glasgow Coma Scale (GCS) was 13 (Eye 3, Verbal 4, and Motor 6). On the chest, there was reduced air entry and crackles on the right side of the lung. On admission vital signs were as follows: SpO2 90% on room air; blood pressure, 105/68 mmHg; pulse, 116 beats/min; respiratory rate 24, blood glucose, 69 122 mg/dl.

The initial laboratory investigation revealed white blood cell count 18.04 x10^9/L (normal 4.00–71 10.00 x10^9/L), hemoglobin 12.1 g/dl (normal 12.0–16.0 g/dl), platelet 450x10^9/L (normal 100–300x10^9/L), C-reactive protein 230.73 mg/L (normal 2.5–10 mg/L), aspartate transaminase (AST) 60.7 U/I (normal 6–38 U/I), alanine transaminase (ALT) 36.0 U/I (normal 6–40 U/I), serum creatinine 2.1 mg/dl (normal 0.4–1.4 mg/dl), and serum urea 170.5 mg/dl (10–50 mg/dl), sodium 135.9 mmol/l ((normal 135.0–145.0 mmol/l), potassium 5.3 mmol/l (normal 3.5–5.5 mmol/l), calcium 2.4 mmol/l (normal 2.10–2.70 mmol/l), COVID-19 test was negative.

Chest radiography was performed immediately and showed right upper lobe consolidation (Figure 1). The patient was admitted to the ward with community-acquired pneumonia and acute kidney injury (AKI) and started Ceftriaxone 1 g two times a day, intravenous fluid of normal saline (NS) 1 Liter per 24 h, ipratropium with ventolin inhaler four times a day,paracetamol 1000 mg infusion once a day, and nasal oxygen support 2 L per hour. After 12 hours, the patient's condition deteriorated and developed respiratory distress and hypoxia with oxygen saturation 80% on a simple mask of 6L/hour, and was transferred to the

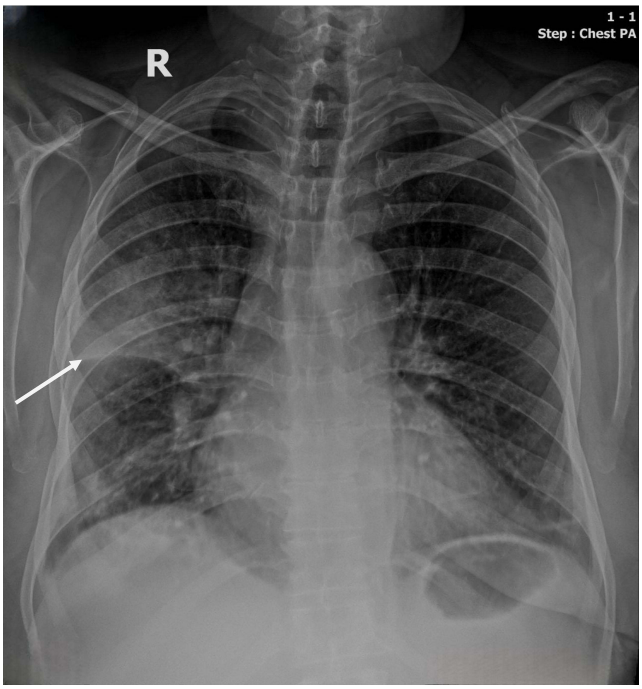

Figure 1 Chest x-ray demonstrates pneumonia. The arrow indicates the right upper lobe consolidation.

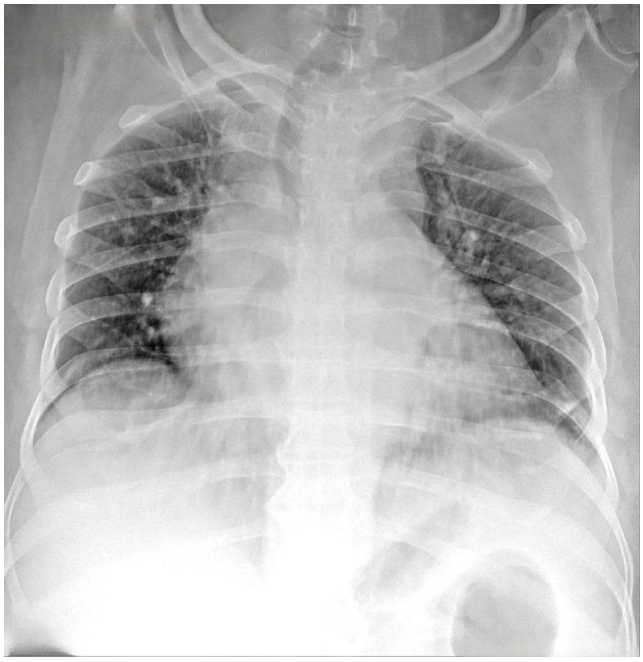

Figure 2 PA view chest x-ray shows that the previous consolidation has resolved, and the lungs now appear normal.

intensive care unit (ICU) for supplemental oxygen therapy and further management. 24 hours later in the ICU, the patient's urine appeared dark-colored and urine analysis revealed no hematuria, suggesting rhabdomyolysis. Laboratory investigation revealed white blood cell count 28.04 x10^9/L (normal 4.00–10.00 x10^9/L), 87 hemoglobin 11.2 g/dl (normal 12.0–16.0 g/dl), platelet 430x10^9/L (normal 100–300x10^9/L), C-reactive protein 300.73 mg/L (normal 2.5–10 mg/L),Creatinine kinase (CK) 4450 u/l (normal 1–171 u/l) serum myoglobin 389 ng/mL (normal 0–80 ng/mL),serum creatinine 2.5 mg/dl (normal 0.4–1.4 mg/dl), and serum urea 194.3 mg/dl (10–50 mg/dl),sodium 137.2 mmol/l ((normal 135.0–145.0 mmol/l), potassium 5.9 mmol/l (normal 3.5–5.5 mmol/l), calcium 1.9 mmol/l (normal 2.10–2.70 mmol/l),aspartate transaminase (AST) 84.7 U/I (normal 6–38 U/I), alanine transaminase (ALT) 40.5 U/I (normal 6–40 U/I) after that our previous diagnose changed for community acquired pneumonia associated with acute kidney injury complicating rhabdomyolysis and electrolyte imbalance so we change our plan for immediate and started with intravenous fluid Normal saline (ns) 5 liter per 24 hour along with adding sodium bicarbonate, piperacillin and tazobactam 2.25 g three times a day, moxifloxacin 400 mg for once a day. We maintained the intravenous fluid according to the dehydration status and urine output. After six days in the ICU, the patient improved and was switched to a nasal oxygen cannula, and laboratory investigations improved.

Subsequently, the patient was transferred to the ward for continued management. A 10^{th} days on admission a chest X-ray was performed and showed normal (Figure 2), clinically improved, and normal laboratory investigations (Table 1). Finally, the patient was discharged, and follow-up was planned.

Discussion

This case report describes a rare presentation of community-acquired pneumonia with acute kidney injury complicated by rhabdomyolysis and highlights the challenges faced in managing such cases in resource-limited settings, which is different from previous cases reported in developed countries.

Rhabdomyolysis, although not commonly caused by infectious agents, can occur due to various bacteria, notably Legionella species, which account for 2–15% of community-acquired pneumonia cases requiring hospitalization and is the second most common cause of severe pneumonia requiring intensive care unit admission, followed by Streptococcus species, Francisella tularensis, and Salmonella species, with bacterial causes leading to significant morbidity (57% incidence of acute renal failure) and mortality (38% of cases).[7]

Table 1 The Result of the Laboratory Investigations

Tests	Reference Range	On ward Admission	During ICU	On ICU Discharge	On Ward Discharge
White cell count (WBC, *10^9 /L)	4.00–10.00	18.04	28.4	10.56	8.62
Hemoglobin (HB, g/dl)	12.0–16.0	12.1	11.2	11.9	12.3
Platelet (PLT, *10^9 /L)	100–300	450	430	310	111
C-reactive protein (CRP, mg/L)	2.5–10	230.73	300.73	11.3	7.4
Aspartate transaminase (AST, U/L)	6–38	60.7	84.7	45	25
Alanine transaminase (ALT, U/L)	6–40	36.0	40.5	38.2	23.3
Creatinine (Crt, mg/dl)	0.4–1.4	2.1	2.5	1.3	0.97
Blood urea (Blood urea, mg/dl)	10–50	170.5	194.3	84.1	32.3
Creatinine kinase (CK, u/l)	1–171	----------	4450	154	94
Serum myoglobin (S. myoglobin, ng/mL)	0–80	----------	389	75.4	38.2
Sodium (Na+, mmol/l)	135–145	135.9	137.2	140	137.1
Potassium (K+, mmol/l)	3.5–5.5	5.3	5.9	4.3	3.6
Calcium (Ca+, mmol/l)	2.10–2.70	2.4	1.9	2.1	2.3

Note: *Multiplication Sign.

Although the exact mechanism underlying rhabdomyolysis secondary to community-acquired pneumonia (CAP) is not fully understood, it may involve various factors such as direct invasion of muscle tissue by pathogenic bacteria, toxic effects of bacterial metabolites, and cytotoxicity mediated by inflammatory transmitters and necrosis factors contributing to tissue damage.[8] The etiology of acute kidney injury (AKI) related to Legionella may be immune or infection-related, with AKI potentially occurring indirectly through rhabdomyolysis or directly from Legionella infection.[9] Any patient with the triad of pneumonia, rhabdomyolysis, and AKI should be suspected for Legionella infection.[10]

Numerous case reports have documented the association between Legionella, rhabdomyolysis, and renal failure in developed countries, with Legionnaires' disease causing acute kidney injury (AKI) in 13% to 15% of cases, with a mortality rate of 40% to 53%, while 33% to 50% of patients with Legionnaires' disease complicated by rhabdomyolysis develop AKI.[11–15]

A study of 392 cases of community-acquired pneumonia found that 12.5% were due to Legionella, with 20.4% of these cases requiring ICU admission. Overall mortality associated with Legionella was 54%, with nosocomial cases showing a mortality rate of 79% and community-acquired cases of 42%.[16,17]

The study analyzed 11 cases of community-acquired pneumonia complicated by rhabdomyolysis, which often results in higher rates of acute kidney injury and poorer prognosis than exercise-induced rhabdomyolysis.[6]

Rhabdomyolysis secondary to Legionella was initially reported by Posner et al in 1980. Since then, more than 20 case reports have documented the association between rhabdomyolysis and acute renal failure.[17] Notably, patients with traumatic rhabdomyolysis who develop acute kidney injury (AKI) exhibit significantly higher mortality rates than those who do not (59% vs 22%).[18]

The classic triad of rhabdomyolysis symptoms includes muscle pain, weakness, and dark urine.[19] However, this triad is observed in only about 10% of patients, with up to 50% of patients not experiencing muscle pain or weakness, presenting instead with non-specific symptoms.[18] In this particular case, the initial sign was the appearance of dark urine, ranging in color from pink to brown to black, which necessitated a differential diagnosis from hematuria.

The laboratory diagnosis of rhabdomyolysis predominantly relies on the measurement of serum or plasma creatine kinase (CK), which remains the most sensitive test despite being considered a "surrogate" marker.[18] Although there is no

universally established cut-off threshold, a CK concentration ranging from five to ten times the upper reference limit (approximately 1000 U/L) is commonly utilized for diagnosis.[19] While CK values above 5000 U/L are generally considered predictive of acute kidney injury (AKI) and closely associated with kidney damage, recent evidence suggests that myoglobin, with its more rapid kinetics, plays a more critical role in the pathogenesis of myoglobinuric AKI and that monitoring myoglobin levels over time may more accurately reflect disease activity and therapeutic efficacy.[18,20] CK remains the biochemical "gold standard" for diagnosing rhabdomyolysis, while myoglobin should be regarded as the "gold standard" for prognostication, particularly in patients with non-traumatic rhabdomyolysis.[18]

In our case initially was diagnosed as community acquired pneumonia with acute kidney injury As the ward, after his condition deteriorated, transferred to the ICU and reassessed clinically, and available investigations which we finally diagnosed the triad of community-acquired pneumonia, AKI and Rhabdomyolysis. In this case, there was elevated creatine kinase (CK) and myoglobin in the serum with the appearance of dark urine, which confirmed rhabdomyolysis after the patient had CAP with AKI. In developed settings, cases such as Legionella pneumonia-related rhabdomyolysis and AKI are most often diagnosed at an early stage because of advanced diagnostic tools like metagenomic next-generation sequencing (mNGS) and urinary antigen tests that could rapidly identify pathogens while developing countries do not available or scarcity.[5,10]

The management of rhabdomyolysis with acute kidney injury involves aggressive isotonic fluid administration to correct electrolyte and metabolic disturbances, and the first-line treatment for Legionnaires' disease is empiric therapy with fluoroquinolones or macrolides, either alone or in combination with beta-lactams.[21] Intravenous fluid administration in rhabdomyolysis should be tailored to the patient, starting at 400 cc/hour (adjustable between 200 and 1000 cc/hour) with sodium bicarbonate for urine alkalization, and titrated to urine output to ensure adequate resuscitation without risking volume overload.[22]

In this case, the management was intravenous fluid sat 200 mL/hr with sodium bicarbonate tailoring according to the urine output and combination antibiotics of moxifloxacin 400 mg once a day with piperacillin-tazobactam 2.25 g three times a day. Similarly, studies conducted in resource-limited settings in America and China revealed that a combination of β-lactams with fluoroquinolone or macrolides in rhabdomyolysis-induced pneumonia with AKI significantly improved.[5,10,23] In resource-limited settings, managing severe AKI and respiratory failure is challenging due to limited access to continuous renal replacement (CRRT), advanced ventilatory support, and targeted antibiotics such as fluoroquinolones or macrolides, which are commonly available in developed countries.

The limitations of this study include the lack of availability of diagnostic tests, such as myoglobinuria for rhabdomyolysis, urinary antigen test (UAT) for Legionnaires 'disease, blood culture, and sputum gram stain/culture for confirmation of the exact etiologies. This case is also limited because of the lack of generalizability.

The study indicated that early detection through clinical and available diagnostic tests and treatment of CAP-induced AKI and RM are critical to avoid missed diagnosis or misdiagnosis in resource-limited settings, ultimately reducing complications and shortening the treatment course.

Conclusion

This case report highlights the importance of early recognition and aggressive management of CAP-related complications in order to prevent adverse outcomes. Clinicians should be aware of the potential for rare but serious complications such as rhabdomyolysis in patients with CAP and AKI, especially in resource-limited settings.

Abbreviations

RM, rhabdomyolysis; AKI, acute kidney injury; CAP, community-acquired pneumonia; CK, Creatinine kinase; CPK, creatinine phosphokinase; mNGS, metagenomic next-generation sequencing.

Ethics and Consent

Informed Consent: Written consent was obtained from the patient for the publication of this case report, including permission to use any accompanying images. At our institution, SIMAD University, ethical approval from the Institutional Review Board is not required for case reports.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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