# Rapid Diagnosis and Management of Concomitant Acute Cardiogenic Pulmonary Edema and Community-Acquired Pneumonia Increase Patient Survival

**Adityo Wibowo[1], Tetra Arya Saputra[1]**
[1]Department of Pulmonology and Respiratory Medicine,
Faculty of Medicine, University of Lampung

**Abstract**

Acute pulmonary edema is a potentially life-threatening complication of cardiac disease that leads to acute respiratory failure. Cardiogenic pulmonary edema affects approximately 80% of patients with acute decompensated heart failure. Acute pulmonary edema caused by heart failure affects nearly a million individuals annually, and the number of cases is growing significantly as the ejection fraction reduces. The clinical presentation can be identified by a sudden onset of dyspnea and a quick accumulation of fluid inside the alveolar and lung interstitial secondary to an increase in hydrostatic pressure. Community-acquired pneumonia (CAP) is the most common respiratory infection caused by a microbial infection of the lung parenchyma and has almost similar clinical characteristics to acute pulmonary edema. The length of stay in hospital will increase nearly two times longer in these concomitant diseases. Comorbidity of cardiogenic lung edema and CAP increase mortality rate by nearly 25% in heart failure patients without early detection and proper management.

**Keywords**: Acute pulmonary edema, management, pneumonia

**Corresponding author**: Adityo Wibowo, Soemantri Bojonegoro Street No.1, email: aditpulmo@gmail.com

## Introduction

Acute pulmonary edema resulting from a cardiogenic problem is responsible for a large proportion of adult mortality across the world. High mortality and morbidity rates are directly associated with respiratory distress caused by either cardiovascular disease or complications from a lung infection that occur concurrently as an acute episode of pneumonia.[1]

The current study presents a recent perspective on the major pathophysiological mechanisms, prevalence, risk factors, outcomes, and appropriate therapy for pneumonia as a secondary infection in lung edema due to cardiovascular events. The pathogenesis of pulmonary edema generated predominantly by cardiac disease can happen in acute episodes of chronic decompensated heart disease, such as myocardial infarction or hypertensive heart disease.[2,3]

Patients with pulmonary edema are considered at a higher risk of developing pneumonia than various other groups in the population, and patients with such concomitant disease have a worse survival rate. As a result, pneumonia increases the likelihood of developing respiratory distress and is frequently seen as a factor in decompensation that leads to hospitalization. The death rate was 39.5 per 100 patient-years with pneumonia, indicating a more than four times increase in mortality risk, compared to the patient without pneumonia. Accordingly, precise management and intensive treatment are required to improve the patient's chances of survival.[4]

## Case presentation

A 59-year-old female patient arrived at the emergency room with the chief complaints of developing dyspnea on exertion and at rest started three weeks ago, which worsened within 8 hours before admission. The complaints had been associated with lower extremities edema and nocturnal dyspnea. The patient also experienced a cough over four days, with purulent sputum and a slightly higher body temperature. Uncontrolled hypertension and chronic hypertensive heart disease were among the most recent medical history.

The patient's clinical appearance was completely conscious, with vital signs indicating an increasing body temperature of 38.6°C, a heart rate of 116 beats per minute, a blood pressure of 162/92 mmHg, and a respiratory rate of 32 times per minute with an

oxygen saturation of 89%, subsequently increased to 95% following 10 L/min supplemental oxygen. The physical examination demonstrated symmetrical lung expansion, Sonor percussion, and bilateral lung coarse crackles from the middle to the lower thorax region. The heartbeat was regular, without abnormal heart sounds.

Laboratory tests confirmed leukocytosis (13,300/µL), neutrophilia (78%), and polycythemia (hemoglobin 16.2 mg/dL) with hematocrit at 45%. The chest X-ray revealed both-sided middle and lower lung inhomogeneous consolidations with cephalization in pulmonary vasculature (Figure 1), and the cardiac visualization could not be identified due to the consolidations. The ECG showed normal sinus rhythm and hypertrophy in the left ventricular wave. The patient was admitted with a working diagnosis of acute cardiogenic lung edema and subsequent infection of community-acquired pneumonia.

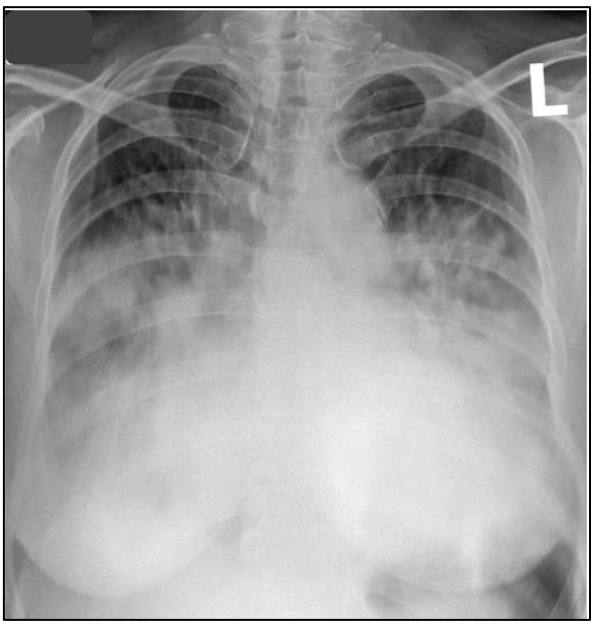


**Figure 1.** An initial chest X-ray revealed bilateral consolidations in the medial and lower regions of the lung. Cephalizations appeared in the pulmonary vascularization as a marker of volume overload in the pulmonary vein.

From the first day of treatment, the patient was given intravenous diuretics furosemide three times a day (40mg-20mg-20mg) with potassium blood monitoring to eliminate excess fluid and had urine output monitored via a urinary catheter, as well as fluid intake restriction. The patient was additionally lying in a 60-degree Fowler's position to support lung expansion, which was impaired due to restricting issues. Candesartan was used as hypertension treatment at 16 mg once daily. Respiratory fluoroquinolone levofloxacin 750 mg per 24 hours was administered as antibiotic therapy for infection treatment, with kidney function evaluation.

After 24 hours of treatment, the patient showed clinical improvement with a lower body temperature of 37.6°C, heart rate of 102 beats per minute, respiratory rate of 26 times per minute, and titration of oxygen supplementation to 5 L/min showed peripheral oxygen saturation of 97%. Following four days of antibiotic administration, a chest x-ray was performed to evaluate clinical improvements. Post-treatment chest x-ray evaluation confirmed improvement with the absence of consolidation in bilateral lung areas. Cardiac measurement after resolution was identified and the chest X-ray showed a cardiothoracic ratio exceeding 50% suggested cardiomegaly.

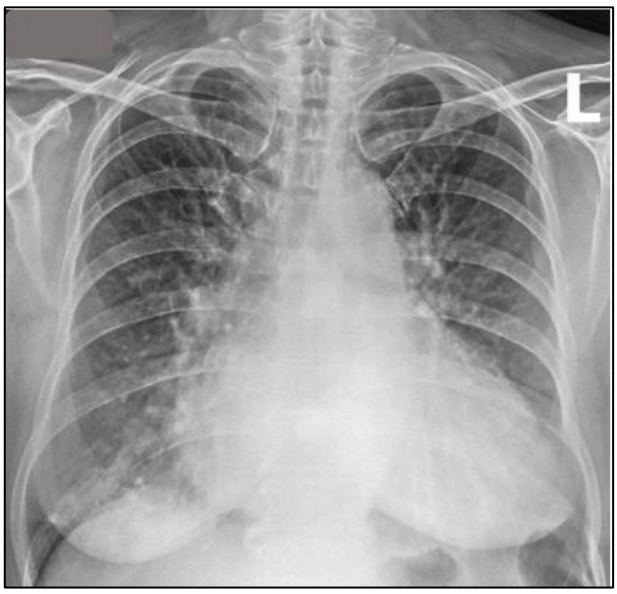


**Figure 2.** A chest X-ray taken four days after treatment showed no sign of bilateral consolidations. A cardiothoracic ratio of 71% indicated cardiomegaly and chronic heart disease.

**Discussion**

Acute pulmonary edema is a potentially life-threatening complication of cardiac disease that leads to acute respiratory failure. The clinical presentation can be identified by a sudden onset of dyspnea and a quick accumulation of fluid inside the alveolar and lung interstitial secondary to an increase in hydrostatic pressure. this condition is most likely caused by a sudden increase in heart-filling pressures. Cardiogenic pulmonary edema is mainly caused by acute decompensated heart failure (ADHF) and the main underlying cause of ADHF is left ventricular (LV) systolic failure, with or without diastolic dysfunction. Cardiogenic pulmonary edema may also be induced by a range of conditions other than cardiac dysfunction, including primary fluid overload, pulmonary disease, and severe kidney failure.[5]

Further medical conditions, when combined with pre-existing systolic or diastolic dysfunction, might cause hemodynamic decompensation and increase the risk of pulmonary edema. These can be classified based on the primary pathophysiologic cause characterized by a rapid increase in preload or afterload. Elevated preload related to volume excess is caused by an increase in LV filling pressure, which can be caused by any factor of intravascular volume expansion. On the other hand, patients with severe persistent hypertension would increase the afterload instead of having a preserved ejection fraction. Increased afterload, whether with or without increased preload, can induce the development of pulmonary edema and stimulate the adrenergic system, raising blood pressure and worsening the afterload increase.[6]

The pathophysiology of pulmonary edema due to hypertension has led to improved clinical management strategies. This form of pulmonary edema is triggered by an immediate increase of fluid induced by several factors like left ventricular dysfunction or extremely increased blood pressure. Patients with hypertension-induced cardiogenic pulmonary edema should be maintained with a combination of treatments involving cardiac preload and afterload. Although this type of cardiogenic pulmonary edema is typically treated initially with high-dose diuretics, a vasodilator Candesartan is also showing beneficial outcomes, according to the literature, to repair the effect of increased afterload capacitance. This medication shows efficacy in decreasing afterload and hence significantly

reducing systemic vascular resistance in conjunction with lowering blood pressure.[5-7]

Community-acquired pneumonia (CAP) is the most common respiratory infection regardless of the immune status of the patients. CAP is mostly caused by microbial infection of the lung parenchyma clinically characterized by fever, cough, and respiratory failure. The morbidity and mortality rate for CAP alone persists very high around 30% in hospitalized patients. The poor prognosis is linked to more serious complications such as respiratory failure, sepsis, and cardiovascular events, which significantly decrease the chances of survival.[8]

Comorbidity of heart failure and CAP and hospitalization rates for both concomitant diseases are increasing significantly among older individuals. The primary cause of hospital admission in heart failure relates to an acute respiratory tract infection. The symptoms of acute cardiogenic pulmonary edema may appear identical to those of CAP. Both conditions are frequently associated with cough, shortness of breath, fatigue, and/or peripheral edema. Clinical manifestation, standard laboratory testing, and chest X-ray findings appear to have a limited value in distinguishing acute pulmonary edema from CAP.[9]

Acute pulmonary edema chest X-rays may show left atrial and pulmonary venous enlargement, also peribronchial patterns. The first sign of hydrostatic pulmonary edema is an increase in vascularity in the precardiac portion of the cranial mediastinum above the aortic arch. The next manifestation is cephalization of pulmonary vessels, which occurs when blood is redistributed into the superior lobe. It can be identified when the upper lobe veins have the same or wider diameter than the lower lobe veins. Interstitial edema involves both the peribronchial interstitium in the middle lobe and the interstitial layer on the periphery lung area, known as Kerley B lines. Lung infiltrate, an objective marker of community-acquired pneumonia on a chest X-ray, is difficult to detect during acute lung edema. Thus, in this case, clinical and laboratory findings support the diagnosis.[10,11]

The severity of CAP symptoms in acute pulmonary edema is determined by numerous factors. Pneumonia may cause a systemic inflammatory response, leading to life-threatening hypoperfusion and multiorgan dysfunction. The pathogens that cause bacterial pneumonia may vary epidemiologically. Studies have confirmed the most frequent bacteria associated with CAP, including *Streptococcus pneumoniae*, *Klebsiella pneumoniae*, *Hemophilus influenza*, and *Pseudomonas aeruginosa*. Guidelines recommend a combination of a beta-lactam (such as amoxicillin-clavulanate, ampicillin-sulbactam, cefotaxime, or ceftriaxone) with a macrolide (azithromycin or clarithromycin). Fluoroquinolones (such as levofloxacin and moxifloxacin) may be a potential alternative treatment in penicillin-sensitive individuals. Immediate antibiotic administration in CAP may prevent sepsis and early hospital discharge. In contrast, several studies revealed that delaying antibiotic therapy before hospitalization was related to poor clinical outcomes and increased mortality.[12-14]

**Conclusion**

Early management and treatment based on simple diagnostic procedures may raise the patient's survival in this case of severe respiratory conditions such as acute pulmonary edema and community-acquired pneumonia. Secondary bacterial pneumonia is common during an acute clinical episode in chronic heart disease and increases the mortality rate by about 7.3%.

**References**

1. Zanza C, Saglietti F, Tesauro M, Longhitano Y, Savioli G, Balzanelli MG, et al. Cardiogenic Pulmonary Edema in Emergency Medicine. Advances in Respiratory Medicine. 2023; 91(5):445-63.
2. Nishiga M, Wang DW, Han Y, Lewis DB, Wu JC. COVID-19 and cardiovascular disease: from basic mechanisms to clinical perspectives. Nat Rev Cardiol. 2020 Sep;17(9):543-58.
3. King KC, Goldstein S. Congestive heart failure and pulmonary edema. [Updated

2022 Sep 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554557/
4. Shen L, Jhund PS, Anand IS, Bhatt AS, Desai AS, Maggioni AP, et al. Incidence and outcomes of pneumonia in patients with heart failure. J Am Coll Cardiol. 2021;77(16):1961-73.
5. Zanza C, Saglietti F, Tesauro M, Longhitano Y, Savioli G, Balzanelli MG, et al. Cardiogenic pulmonary edema in emergency medicine. Adv Respir Med. 2023;91(5):445-63.
6. Arrigo M, Jessup M, Mullens W, Reza N, Shah AM, Sliwa K, et al. Acute heart failure. Nat Rev Dis Primers. 2020;6(1):16.
7. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021;42(36):3599-726.
8. Tokgoz Akyil F, Yalcinsoy M, Hazar A, Cilli A, Celenk B, Kilic O, et al. Prognosis of hospitalized patients with community-acquired pneumonia. Pulmonology. 2018;(17):30156-2.
9. Malek R, Soufi S. Pulmonary edema. [Updated 2023 Apr 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557611.
10. Barile M. Pulmonary edema: A pictorial review of imaging manifestations and current understanding of mechanisms of disease. Eur J Radiol Open. 2020;7:100274.
11. Rachina S, Bobylev A, Lazarev P, Mladov V, Carrouel F, Avdeev S, et al. Biomarker value in the diagnosis of community-acquired pneumonia with concomitant chronic heart failure. J Clin Med. 2021;10(19):4570.
12. Stotts C, Corrales-Medina VF, Rayner KJ. Pneumonia-induced inflammation, resolution and cardiovascular disease: causes, consequences and clinical opportunities. Circ Res. 2023;132(6):751-74.
13. Pahal P, Rajasurya V, Sharma S. Typical bacterial pneumonia. [Updated 2023 Jul 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534295/
14. Darraj A, Hudays A, Hazazi A, Hobani A, Alghamdi A. The association between emergency department overcrowding and delay in treatment: A systematic review. Healthcare (Basel). 2023;11(3):385.