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CASE REPORT

Dyspnea Secondary to Pneumonia with Suspected Acute Cardiogenic Pulmonary Edema Secondary to Heart Failure

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Abstract: Pneumonia remains one of the leading causes of morbidity worldwide and may present with clinical features that overlap with cardiogenic pulmonary edema secondary to heart failure. Distinguishing these conditions in primary healthcare can be challenging because of limited diagnostic resources, making appropriate clinical assessment and timely referral essential. A 51-year-old man presented to a rural primary healthcare facility with progressive dyspnea, productive cough, fever, and exertional fatigue. Physical examination revealed tachypnea, oxygen desaturation, bibasal coarse crackles, and suspected cardiomegaly. Laboratory findings demonstrated leukocytosis. Chest radiography suggested acute pulmonary edema with suspected pneumonia and cardiomegaly. The patient received supplemental oxygen, intravenous ampicillin, nebulization, corticosteroids, antipyretics, and supportive therapy. Referral to a higher-level hospital was strongly recommended. However, the patient declined referral and requested discharge against medical advice on the second day of hospitalization. This case emphasizes the diagnostic complexity of pneumonia and acute cardiogenic pulmonary edema in resource-limited primary healthcare settings. Comprehensive clinical assessment, appropriate initial stabilization, and evidence-based management remain essential. Timely referral is crucial for definitive diagnosis and specialist care, although patient refusal may limit therapeutic options and potentially affect clinical outcomes.

Keywords: Pneumonia, Acute Lung Edema, Primary Health Center, Declined Referral.

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INTRODUCTION

Pneumonia is a common acute respiratory infection that affects the alveoli and distal bronchial tree of the lungs. It can be caused by viruses, bacteria, fungi, or a combination of these pathogens, resulting in inflammation and fluid accumulation within the pulmonary parenchyma. Globally, pneumonia remains one of the leading causes of morbidity and mortality, particularly among children under five years of age (Torres et al., 2021; McDonough et al., 2014).

The clinical manifestations of pneumonia include cough, tachypnea, fever, although fever may be absent in some cases, and wheezing, which is more frequently associated with viral respiratory infections. Patients may also present with headache, myalgia, and sore throat. The diagnosis of pneumonia can be supported by the presence of pulmonary infiltrates or air bronchograms on chest radiography together with compatible clinical findings, including cough, changes in sputum characteristics or purulent sputum, body temperature >38°C or a history of fever, physical examination findings suggestive of pulmonary consolidation, bronchial breath sounds, crackles (rales), and abnormalities in the leukocyte count (Bickley, 2012; Torres et al., 2021).

According to the 2018 Basic Health Research (Riskesdas), the prevalence of pneumonia in Indonesia increased from 1.8% to 2.0%. Pneumonia can affect individuals across all age groups; however, its prevalence is higher among older adults. The prevalence of pneumonia increased with age, reaching 2.5% among individuals aged 55–64 years, 3.0% among those aged 65–74 years, and 2.9%

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among those aged ≥75 years 54(Badan Penelitian dan Pengembangan Kesehatan, 2019).

Edema is the accumulation of excess fluid within body tissues. Pulmonary edema is characterized by the accumulation of fluid within the lungs, particularly in the pulmonary interstitial spaces and alveoli. The accumulation of fluid within the alveoli impairs gas exchange and may result in respiratory distress. Pulmonary edema may occur through either cardiogenic or non-cardiogenic mechanisms. Cardiogenic pulmonary edema is most commonly associated with left ventricular dysfunction or left-sided heart failure. In contrast, non-cardiogenic pulmonary edema is characterized by increased pulmonary vascular permeability, severe hypoxemia, and diffuse pulmonary infiltrates in the absence of evidence of left-sided heart failure (Rampengan, 2014; Heidenreich et al., 2022).

CASE REPORT

A 51-year-old man Mr. J presented to the Emergency unit of Selaparang Pratama Regional Hospital with a chief complaint of shortness of breath. The patient reported progressive dyspnea for the past three days, which had worsened on the day of admission. The dyspnea occurred during moderate physical activity and was relieved by rest. The symptoms were not affected by weather conditions. The patient also complained of a productive cough with clear sputum for one week and intermittent fever for the previous four days. In addition, he reported epigastric pain that had started one day before admission without associated nausea or vomiting. He denied a history of night sweats, hemoptysis, or weight loss. There was no history of hypertension, diabetes mellitus, heart disease or asthma. The patient had a history of smoking for approximately 30 years, with consumption of about five cigarettes per day.

On arrival, the patient appeared dyspneic but was fully alert and oriented, with a Glasgow Coma Scale (GCS) score of 15 (E4V5M6). His blood pressure was 130/70 mmHg, heart rate was 108 beats/minute with a regular rhythm, respiratory rate was 28 breaths/minute, body temperature was 39.0°C, and Spo2 90% on room air, which improved to 98% with oxygen administered via a nasal cannula at 5 lpm. The patient weighed 80 kg and was 160 cm tall, corresponding to a body mass index (BMI) of 31.25 kg/m², consistent with obesity.

General physical examination showed a tachypnea. Head and neck examination was unremarkable. Chest examination demonstrated symmetrical chest expansion with mild intercostal retractions. Pulmonary auscultation revealed bilateral vesicular breath sounds with prominent coarse crackles over both lung fields, without wheezing. Cardiac auscultation demonstrated normal first and second heart sounds (S1 and S2) without murmurs or gallops. Cardiac percussion suggested an enlarged cardiac. Abdominal examination revealed a soft abdomen with normal bowel sounds and tenderness over the epigastric region. Examination of the extremities showed warm, well-perfused limbs with a capillary refill time of less than 2 seconds and no peripheral pitting edema.

Complementary diagnostic investigations performed on the day of admission (August 1st 2026) revealed the following objective results: Routine Hematology Evaluation: A complete blood count showed mild leukocytosis (WBC: 13,91× 10^3/µL; reference range: 4.–10) with a relative neutrophilia (NEUT%: 89,2%; reference range: 50.0–70.0) Hemoglobin levels (HGB: 14,1 g/dL), hematocrit (HCT: 43.9%), and platelet counts (PLT: 238 × 10^3/µL) were all within their respective normal limits. Random blood glucose (RBG) was measured at the upper limit of normal (210 mg/dL; reference range: < 200 mg/dL). Cholesterol total 204; reference range < 200 mg/dl. Radiological Assessment (PA Chest X-ray): Chest X-ray (AP View, Asymmetric): pneumonia; differential diagnosis pulmonary edema with cardiomegaly.

Table 1. Complete Blood Count: mild leukocytosis

Complete Blood Count 01/08/2026	
Total WBC	13.91 x 10^3/µL
Total RBC	4.85 x10^6/ µL
Hemoglobin	14.1 d/dL
Hematocrit	43.9%
Thrombocyte	238 x 10^3/µL

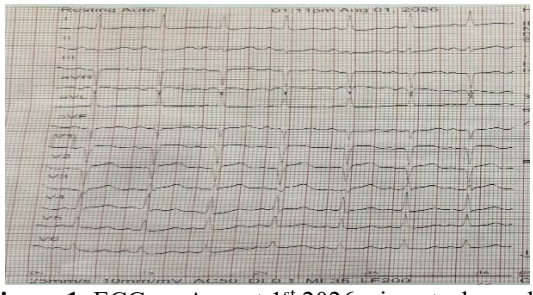


Figure 1. ECG on August 1st 2026: sinus tachycardia

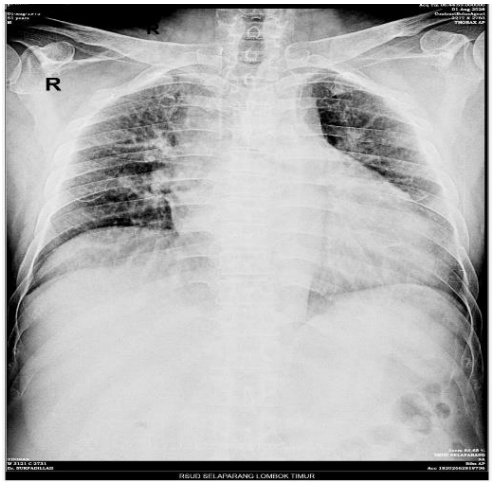


Figure 2. Chest X-ray (AP View)

Heart: Cardiac silhouette appears enlarged and there is calcification of the aortic knob. Lungs: Bilateral perihilar

haziness. Bilateral parahilar and paracardiac infiltrates. Right and left costophrenic angles are sharp. Right and left hemidiaphragms are well defined. Soft tissues are unremarkable. Trachea is midline. Bone structures are intact. Impression: Early pulmonary edema; differential diagnosis includes pneumonia with cardiomegaly.

The patient was managed with the following treatment regimen:

- Intravenous maintenance fluid with 0.9% normal saline, 500 mL over 24 hours.
- Intravenous Furosemide, 1 ampoule every 12 hours.
- Intravenous Dexamethasone, 1 ampoule every 8 hours.
- Intravenous Ranitidine, 50 mg every 12 hours.
- Nebulized Combivent® (1 respule diluted with 1.5 mL of 0.9% normal saline) every 8 hours.
- Intravenous paracetamol 3x500 mg
- Intravenous ampicillin, 1 g every 12 hours.
- Oral Ambroxol, one tablet three times daily.
- Oral Antacid, one tablet three times daily.
- Oral Simvastatin, 20 mg once daily.

The patient was counseled regarding the need for referral to a higher-level healthcare facility. However, he declined referral because the referral hospital was located far from his residence, and he had no family member available to accompany him during hospitalization. The patient was also counseled regarding the possibility of clinical deterioration and the potential complications associated with delaying definitive treatment. After 2 days treatment, the patient requested discharge against medical advice (DAMA) and consequently, the patient was unable to receive definitive standard-of-care treatment.

DISCUSSION

A 51-year-old man presented with dyspnea, productive cough, fever, and fatigue during physical activity, which were suggestive of pneumonia with concomitant suspected cardiac disease. The cardinal clinical manifestations of pneumonia include dyspnea, fever, cough, chest pain, and fatigue, although the clinical presentation may vary according to the causative pathogen and host characteristics (Torres et al., 2021). Cough results from irritation of the tracheobronchial tree and serves as a protective mechanism to clear the airways when mucociliary clearance is impaired due to excessive mucus production, inflammation, infection, or ciliary dysfunction. The inflammatory response leads to the accumulation of inflammatory exudates within the alveoli. It promotes the accumulation of secretions within the airways, thereby stimulating the cough reflex (Bickley, 2012; Torres et al., 2021).

Increased pulmonary capillary permeability and inflammatory exudation may cause alveolar and interstitial fluid accumulation, producing pulmonary infiltrates and crackles and impairing gas exchange, which can result in hypoxemia and dyspnea (Torres et al., 2021). The patient's 30-year smoking history is a relevant risk factor for respiratory infection and cardiovascular disease. Obesity is also independently associated with increased cardiovascular morbidity and mortality (Powell-Wiley et al., 2021).

Physical examination revealed tachypnea, oxygen desaturation (SpO_2 90% on room air), and high-grade fever (39.0°C). Pulmonary examination demonstrated mild intercostal retractions and bibasal coarse crackles, while cardiac examination suggested possible cardiomegaly. Pulmonary congestion increases pulmonary capillary hydrostatic pressure, resulting in the movement of fluid into the pulmonary interstitium and alveolar spaces. This process leads to pulmonary edema, impaired gas exchange, exertional dyspnea, and bibasal crackles (Rampengan, 2014; Heidenreich et al., 2022).

As pulmonary edema progresses, worsening alveolar fluid accumulation contributes to hypoxemia and increasing respiratory distress, often manifested clinically by tachypnea. Laboratory investigations demonstrated leukocytosis with a white blood cell count of $13.9 \times 10^3/\mu L$, suggesting a possible bacterial infection. Electrocardiography revealed sinus tachycardia with P mitral, indicating possible left atrial enlargement and underlying cardiac abnormalities. Chest radiography demonstrated findings consistent with acute pulmonary edema, suspected pneumonia, and cardiomegaly.

The coexistence of pneumonia and cardiovascular abnormalities may increase diagnostic and therapeutic complexity. A recent Indonesian study of 334 hospitalized adults with CAP reported cardiovascular disease in 30.2% of patients and an in-hospital mortality rate of 35.9%; higher CURB-65 scores were also associated with mortality (Rosdiana et al., 2025)

Based on the patient's history and physical examination, the clinical findings were suggestive of pneumonia with possible cardiomegaly. Because pneumonia and cardiogenic pulmonary edema may both present with dyspnea, hypoxemia, crackles, and pulmonary infiltrates, differentiation requires integration of clinical, laboratory, electrocardiographic, and radiological findings (Torres et al., 2021; Heidenreich et al., 2022)

Initial management was provided according to the treatment resources available at the primary healthcare facility. Supplemental oxygen was administered via nasal cannula at 5 L/min to improve oxygenation. Intravenous paracetamol (500 mg) was administered as an antipyretic and analgesic for fever control. Empirical antibiotic therapy with intravenous ampicillin (1 g every 12 hours), a penicillin-class antibiotic available at the primary healthcare center, was initiated to treat the suspected bacterial infection. Intravenous ranitidine (50 mg every 12 hours) was administered for stress ulcer prophylaxis and to relieve the patient's epigastric pain. Nebulized Combivent® (ipratropium bromide/salbutamol) was administered every 8 hours to relieve dyspnea through bronchodilation. In addition, nebulized corticosteroid therapy and intravenous dexamethasone every 8 hours were administered to reduce airway inflammation. However, corticosteroids are not routinely recommended for non-severe pneumonia and should be reserved for specific indications

(Metlay et al., 2019). Oral ambroxol (30 mg three times daily) was prescribed as a mucolytic agent to decrease sputum viscosity and facilitate expectoration. Simvastatin was continued to reduce the patient's long-term cardiovascular risk.

Management of acute pulmonary edema requires prompt identification and treatment of the underlying cause, together with supportive measures to maintain adequate airway, ventilation, and oxygenation (Jufan et al., 2020). In cardiogenic pulmonary edema, definitive management may additionally require therapies directed at pulmonary congestion and the underlying cardiac dysfunction (Parissis et al., 2010; Heidenreich et al., 2022). However, the patient declined referral and therefore was unable to receive definitive evaluation and advanced treatment at a tertiary care center. Despite repeated counseling regarding the risk of clinical deterioration and the potential consequences of delaying definitive treatment, the patient chose to remain at the primary healthcare facility. On the second day of hospitalization, he requested discharge against medical advice (DAMA), precluding further diagnostic evaluation and definitive management.

CONCLUSION

Despite the limited diagnostic and therapeutic resources available at the rural primary healthcare facility, patient management adhered to established clinical standards. Diagnostic and therapeutic decisions were guided by the patient's clinical presentation, physical examination findings, available investigations, and current evidence-based guidelines and literature.

STRENGTHS AND LIMITATION

The primary strength of this report lies in its detailed, real-time documentation of the acute primary-care presentation and the immediate risk stratification applied in a low-resource setting. This case demonstrates that appropriate initial management can be delivered in a rural primary healthcare facility despite limited diagnostic and therapeutic resources. The healthcare team followed standard clinical protocols and provided optimal treatment based on the resources available, while strongly recommending referral to a higher-level hospital for definitive evaluation and specialist care. However, the patient refused referral despite repeated counseling regarding the potential risks of clinical deterioration.

CONFLICT OF INTEREST

The authors declare that there are no financial or personal conflicts of interest regarding the publication of this case report.

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ETHICAL APPROVAL

Patient identity was heavily protected by omitting personal registration numbers and using clinical pseudonyms. Institutional clearance and informed verbal consent from the patient's family for the utilization of anonymized clinical data were secured prior to manuscript compilation.

DECLARATION OF USING AI

The authors affirm that artificial intelligence (AI) tools were used to assist in the writing process solely for language enhancement purposes, such as grammar checking, paraphrasing, and improving clarity. No Al tools were employed to generate original content, conduct data analysis, or interpret research findings. The authors take full responsibility for the content, interpretations, and conclusions presented in this manuscript.

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