



# Unmasking the Unexpected: A Case of Acute Pulmonary Edema in Heart Failure with Preserved Ejection Fraction

Nolly O. H. Rantung <sup>a,b\*</sup>, William Kurnia <sup>c</sup>,  
Lawrent E. Sumilat <sup>c</sup>, Dewa Gde Prema A. <sup>a</sup>  
and Welly Salutondok <sup>b,d</sup>

<sup>a</sup> Department of Cardiology and Vascular, Faculty of Medicine, Universitas Kristen Indonesia, Indonesia.

<sup>b</sup> General Hospital, Universitas Kristen Indonesia, Indonesia.

<sup>c</sup> Undergraduate Medical Study Program, Universitas Kristen Indonesia, Jakarta, Indonesia.

<sup>d</sup> Department of Internal Medicine, Faculty of Medicine, Universitas Kristen Indonesia, Indonesia.

## Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

## Article Information

DOI: <https://doi.org/10.9734/ajarr/2025/v19i3925>

## Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://pr.sdiarticle5.com/review-history/131524>

Case Report

Received: 17/12/2024

Accepted: 19/02/2025

Published: 24/02/2025

## ABSTRACT

Acute heart failure, particularly acute pulmonary edema, is a common cause of hospitalization and impacts life expectancy, especially with age. The mortality rate among patients in Southeast Asia is 13%. The severity of the condition is classified based on left ventricular ejection fraction (LVEF).

\*Corresponding author: Email: [nolly.rantung@uki.ac.id](mailto:nolly.rantung@uki.ac.id);

Cite as: Rantung, Nolly O. H., William Kurnia, Lawrent E. Sumilat, Dewa Gde Prema A., and Welly Salutondok. 2025. "Unmasking the Unexpected: A Case of Acute Pulmonary Edema in Heart Failure With Preserved Ejection Fraction". *Asian Journal of Advanced Research and Reports* 19 (3):109-19. <https://doi.org/10.9734/ajarr/2025/v19i3925>.

Only 21.5% of patients with acute pulmonary edema have preserved ejection fraction (HFpEF). This case involves a 63-year-old male patient with acute pulmonary edema due to heart failure with preserved ejection fraction, secondary to acute hypertension and community-acquired pneumonia. Initial management followed guidelines, including symptomatic therapy and observation in the intensive care unit. After three days of treatment, the patient's condition stabilized, with improved hemodynamics and alleviation of symptoms. We want to highlight an uncommon case of HFpEF in a patient presenting with acute pulmonary edema.

*Keywords: Acute heart failure; acute lung edema; heart failure with preserved ejection fraction.*

## 1. INTRODUCTION

Heart failure is a progressive disease associated with reduced life expectancy, regardless of its cause. It has become a significant public health issue, with its incidence and prevalence increasing worldwide. Heart failure is also the final stage of many disease processes, and its risk increases with age. To understand the risk and progression of this disease, a specific classification is used to define heart failure based on left ventricular ejection fraction (LVEF). Prognosis and treatment response in heart failure patients can differ significantly depending on the patient's LVEF (JACC 2022). Heart Failure with Preserved Ejection Fraction (HFpEF): Patients with an LVEF of  $\geq 50\%$  with evidence of increased left ventricular filling pressure (LVFP), either spontaneous or provoked, indicated by elevated natriuretic peptides or hemodynamic measurements. Particularly in cases of acute heart failure, attention is often directed towards heart failure with reduced ejection fraction (HFrEF) (Heidenreich et al., 2022). Heart failure is one of the most common causes of hospitalization in patients over 65 years old in developing countries. In developed countries such as the United States, approximately 3 million cases of acute heart failure are reported annually, with this figure tripling each year and expected to continue increasing in the future (Felker and Teerlink, 2021, McDonagh et al., 2021).

The prevalence of heart failure is estimated to be around 2–3% in Hong Kong, 5% in Indonesia, 1–2% in the Philippines, 0.6% in South Korea, 6% in Taiwan, and 0.4% in Thailand. With these numbers, Indonesia has a relatively high prevalence compared to other Asian countries. Therefore, heart failure warrants special attention, especially during exacerbations (Reyes et al., 2016). Acute lung edema is part of the acute heart failure syndrome characterized by severe shortness of breath, pulmonary congestion, and decreased oxygen saturation,

which develops within hours. In most cases, the cause is cardiogenic (cardiogenic ALO), a sudden increase in filling pressure in the left heart chambers, leading to increased pulmonary capillary pressure and stasis (Militaru et al., 2017). According to the European Society of Cardiology (ESC) guidelines, all patients with APE should be evaluated using echocardiography (Class I indication). This assessment should include the calculation of the left ventricular ejection fraction (LVEF). A study by C. Militaru et al. showed that 44.2% of patients had low LVEF ( $< 40\%$ ), 34.3% had moderately reduced LVEF (40-49%), and 21.5% had preserved ejection fraction ( $\geq 50\%$ ) (Cleland et al., 2006). Based on this data, we aim to report and describe a case of acute heart failure in acute lung edema with preserved ejection fraction, which occurs in about 1/5 of the acute lung edema patient population.

## 2. CASE PRESENTATION

A 63-year-old male presented to the Emergency Department of UKI General Hospital with complaints of shortness of breath, which had been experienced 2 hours before hospital admission. The shortness of breath had been worsening progressively for the past week. Initially, the shortness of breath worsened with daily activities such as sweeping or walking from the porch to the bathroom. The patient reported wheezing during breathing for the past day. He also complained of worsening shortness of breath while lying down. The symptoms improved when the patient was seated, requiring four pillows for support to sleep. The patient also reported difficulty breathing during sleep and waking up feeling as though he was drowning. He mentioned that shortness of breath had been occurring intermittently for the past 10 years, although not as severe as his current symptoms, and he had never sought hospital care. Additionally, for the past 5 days, the patient had been experiencing a productive cough at night, with yellow-brown sputum that made it difficult to

sleep. He also described throbbing headaches when coughing. The patient reported a fever concurrent with the onset of coughing. He also experienced nausea and vomiting of food contents. He denied any nasal congestion, and his bowel and urinary habits were within normal limits.

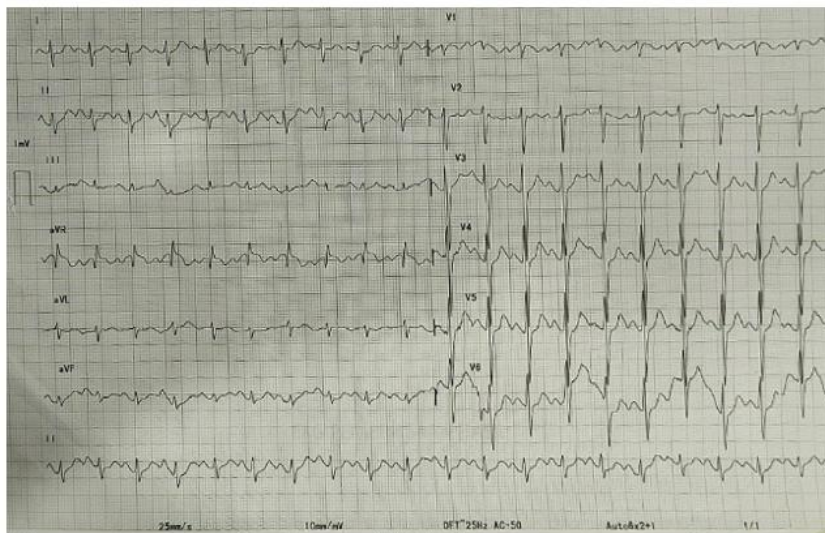
The patient reported a history of uncontrolled type 2 diabetes mellitus (T2DM) but could not recall when he was diagnosed. He also had a history of hypertension, diagnosed 4 years ago, and it was controlled with amlodipine 10 mg once daily. The patient had a history of asthma, diagnosed during adolescence and controlled with medication. There was a family history of hypertension. The patient was a smoker and had a habit of consuming salty foods. On physical examination, the patient appeared short of breath but was conscious and alert (*compos mentis*). His blood pressure was 200/110 mmHg, heart rate 129 beats per minute, respiratory rate 34 breaths per minute, temperature 37.8°C, and oxygen saturation 93%. On thoracic examination, palpation revealed asymmetrical vocal fremitus, weakened vesicular breath sounds, and the presence of rhonchi and wheezing in both lung fields. Cardiovascular examination revealed palpable *ictus cordis* at the 6<sup>th</sup> intercostal space, left anterior axillary line, regular heart sounds (S1 and S2), and no murmurs or gallops. Jugular venous pressure measurement showed visible distention. Extremities were warm, and pitting edema was noted in both lower limbs.

The patient was diagnosed with acute heart failure and suspected pneumonia, with asthma

as a differential diagnosis based on the history and physical examination. The patient was treated in the emergency room, with an IV line and a No. 16 Foley catheter inserted, yielding a urine output of 1.2 cc/hour. The patient was given 10 L of oxygen via a non-rebreathing mask (NRM) and placed in a semi-sitting position.

The complete blood count results revealed leukocytes  $9.0 \times 10^3/\mu\text{L}$ , hemoglobin 17.3 g/dL, hematocrit 52.0%, and platelets  $249.0 \times 10^3/\mu\text{L}$ . Hemostasis physiology showed D-Dimer 351 ng/mL. Blood chemistry tests indicated sodium 143 mmol/L, potassium 3.2 mmol/L, chloride 96 mmol/L, random blood glucose 163 mg/dL, urea 31 mg/dL, and creatinine 1.2 mg/dL. Arterial blood gas analysis showed pH 7.5, pCO<sub>2</sub> 47.6 mmHg, pO<sub>2</sub> 158.4 mmHg, BE 7.7 mmol/L, HCO<sub>3</sub> 35.70 mmol/L. Cardiac enzyme tests revealed CK-MB 35 U/L, and Troponin I was negative. NT-proBNP levels were 2333 pg/mL. The SARS-CoV-2 antigen test showed non-reactive results (Table 1).

The ECG (Fig. 1) showed sinus tachycardia with left axis deviation and left ventricular hypertrophy (LVH) signs consistent with the Peguero-Lo Presti criteria. The chest X-ray (Fig. 2) showed normal bone and soft tissue. The right and left costophrenic sinuses were also within normal limits. Cardiomegaly was present with a cardiothoracic ratio (CTR) greater than 50% and consolidation in both lungs, especially in the central area. The conclusion from the chest X-ray was left ventricular cardiomegaly with alveolar edema, giving a “bat-wing” appearance and suspected pneumonia.



**Fig. 1. Electrocardiogram**

**Table 1. Laboratory Examination**

<b>Examination</b>	<b>Result</b>	<b>Reference Value</b>
<b>Routine Blood Test</b>		
Hemoglobin	17,3 g/dL	13 – 17 g/dL
Leukocytes	9 ribu/uL	5 – 10 ribu/uL
Hematocrit	52%	39 – 51%
Trombosit	249 ribu/uL	150 – 400 ribu/uL
<b>Clinical Chemistry</b>		
Pre-Prandial Blood Sugar	163 mg/dL	< 200 mg/dL
Creatinine	1,2 mg/dL	0,70 – 1,1 mg/dL
Ureum	31 mg/dL	15 – 45 mg/dL
<b>Electrolyte</b>		
Natrium	143 mmol/L	136 – 145 mmol/L
Kalium	3.2 mmol/L	3.5 – 5.1 mmol/L
Chloride	96 mmol/L	99 – 111 mmol/L
<b>Cardiac Enzymes</b>		
Nt-proBNP	2333 pg/mL	CHF in non-acute onset: < 125 CHF in acute onset: < 300
CK-MB	35 U/L	5 – 25 U/L
Troponin-I	Negatif	< 14 pg/L
<b>Blood Gas Analysis</b>		
Blood pH	7.5	7.35 – 7.45
PCO <sub>2</sub>	47.6	36 – 45
PO <sub>2</sub>	158.4	70 – 99
Base Excess	10.8	- 2.5 – 2.5
HCO <sub>3</sub>	35.7	21 – 25
TCO <sub>2</sub>	37.2	21 – 27
<b>Hemostasis</b>		
D-Dimer	351 ng/mL	0-500 ng/mL
	Antigen	
SARS CoV-2	Negative	Negative



**Fig. 2. Chest X-Ray**

Based on the anamnesis, physical examination, and supporting examinations, the patient was diagnosed with acute lung edema (warm-wet) caused by a hypertensive emergency, suspected asthma, and community-acquired pneumonia, classified as severity class IV, along with mild hypokalemia. The patient was treated with furosemide at a dose of 5 mg/hour and isosorbide dinitrate starting at 1 mg/hour, titrated to a target systolic blood pressure of 150-160 mmHg. Digoxin 0.25 mg/mL was administered intravenously. The patient was also treated with salbutamol and ipratropium bromide (2.5 mg/0.5 mg) combined with budesonide 0.5 mg via inhalation, along with methylprednisolone 62.5 mg, paracetamol 2x1 g, omeprazole 2x40 mg, ondansetron 2x8 mg, and his regular medication (amlodipine) continued at a dose of 1x10 mg.

After a few hours, the patient's shortness of breath improved, but his hemodynamic status remained elevated with blood pressure at 175/109 mmHg, heart rate at 129 bpm, and SpO<sub>2</sub> at 99% with 10 LPM oxygen. The patient

was then transferred from the emergency department to the ICVCU for further monitoring. Although his shortness of breath had decreased, he complained of fatigue and continued to have a productive cough with brown sputum. The patient was given additional medications, including lisinopril 1x10 mg and bisoprolol 1x1.25 mg as cardioprotective agents, and intravenous levofloxacin 1x750 mg along with N-acetylcysteine 3x200 mg. His blood pressure decreased to 115/83 mmHg, and his heart rate was 94 bpm. Due to his low potassium levels (3.2 mmol/L) and the potential worsening of hypokalemia with the use of furosemide, spironolactone 1x25 mg was added in combination with furosemide to prevent hypokalemia, along with KSR 2x600 mg, while monitoring potassium levels.

The echocardiogram results showed left atrial dilation with a left atrial volume index (LAVI) of 41 mL/m<sup>2</sup> and concentric left ventricular hypertrophy. The left ventricular systolic function was normal, with an ejection fraction of 67% (measured using the Teichholz method).

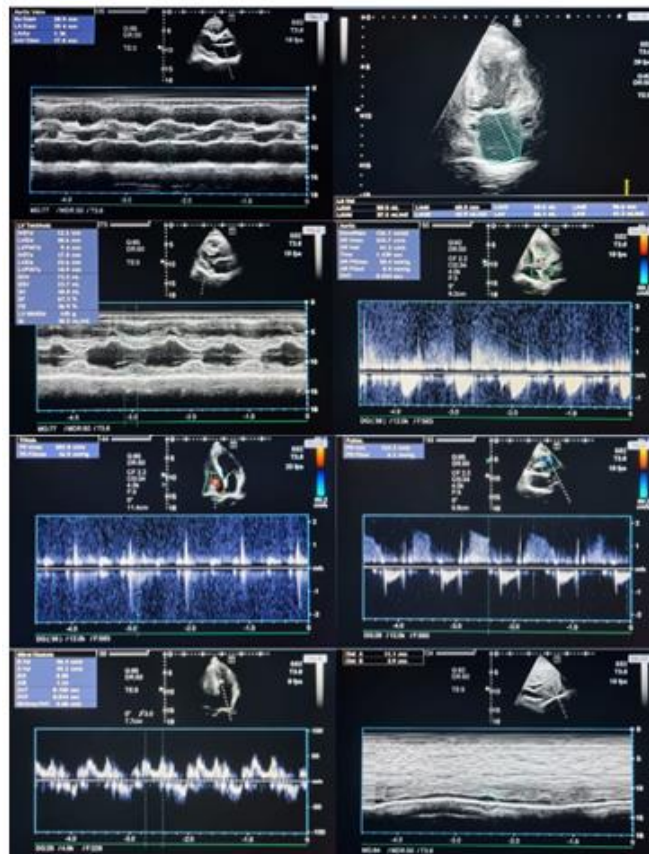
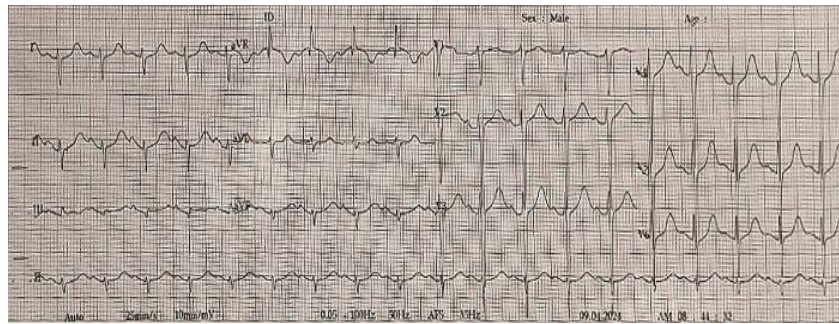


Fig. 3. Echocardiogram Results



**Fig. 4. ECG Evaluation**

A segmental analysis showed globally normal left ventricular kinetics, with diastolic dysfunction indicating impaired relaxation. The aorta had three cusps and no calcifications were observed. Mild aortic regurgitation was noted (ARVCD 1 mm), along with mild tricuspid regurgitation (TVG 16 mmHg) and mild pulmonary regurgitation (low probability of pulmonary hypertension). The right ventricular contractility was normal, with a TAPSE of 2.6 cm, and the inferior vena cava (IVC) measured 1.1 cm with >50% collapsibility, suggesting an estimated right atrial pressure (RAP) of 3 mmHg.

The patient was diagnosed with acute lung edema in the context of heart failure with preserved ejection fraction due to hypertensive emergency and pneumonia. Treatment was continued for approximately one day. The patient's hemodynamic condition remained stable (blood pressure 118/72 mmHg, heart rate 103 bpm, respiratory rate 22 breaths/min, temperature 37°C, SpO<sub>2</sub> 100% on 5 LPM nasal cannula), with no complaints, and the general condition improved significantly.

On physical examination, no rhonchi or wheezing were heard in both lung fields. Heart sounds 1 and 2 were regular, with no murmurs or gallops detected. No pitting edema was found in the patient's lower extremities. A follow-up ECG (Fig. 4) showed a sinus rhythm with findings suggestive of left ventricular hypertrophy (LVH).

The patient was discharged on the third day of treatment. The patient was educated about his condition and advised to attend regular follow-up appointments with a cardiologist and an internal medicine specialist.

### 3. DISCUSSION

Heart failure is a clinical syndrome resulting from a complex process that disrupts the structure or

function of the ventricles, impairing their ability to fill or pump blood effectively. A specific classification is used to define heart failure, which is categorized based on the left ventricular ejection fraction (LVEF). The prognosis and response to treatment in patients with heart failure significantly differ based on this classification. Heart failure classifications based on LVEF include Heart Failure with Reduced Ejection Fraction (HFrEF), which occurs when LVEF  $\leq$  40%. Heart Failure with Improved Ejection Fraction (HFimPEF) occurs when the previous LVEF was  $\leq$  40% but increases to >40%. Heart Failure with Mid-Range Ejection Fraction (HFmrEF) involves LVEF 41-49% with evidence of increased left ventricular filling pressure. Heart Failure with Preserved Ejection Fraction (HFpEF) occurs when LVEF  $\geq$  50%, accompanied by increased left ventricular filling pressure (Heidenreich et al., 2022).

The prevalence of heart failure (HF) is estimated to be around 5% in Indonesia. In the China Hypertension Survey, the prevalence of HF was found to be 1.3%, with 40% of these cases being HFrEF, 23% HFmrEF, and 36% HFpEF (Heidenreich et al., 2022, Reyes et al., 2016, Cleland et al., 2006). In the prospective ASIAN HF registry, the all-cause mortality rate for the entire population was 9.6% at one year, which was higher in patients with HFrEF (10.6%) compared to those with HFpEF (5.4%). The all-cause mortality rate at one year was significantly higher in Southeast Asian patients (13.0%) compared to South Asian patients (7.5%) and Northeast Asian patients (7.4%) (Cleland et al., 2006).

Acute heart failure (ESC, 2021) classifies acute heart failure into four types: acute decompensated heart failure, acute lung edema, right ventricular heart failure, and cardiogenic shock, each with distinct clinical profiles. The clinical profiles are described as 'wet' or 'dry'

based on their fluid status, and as 'cold' or 'warm' based on the assessment of the patient's perfusion status. Among the four classifications of acute heart failure, acute decompensated heart failure and acute lung edema are the most common reasons patients present to the emergency department, with congestion symptoms dominating the clinical profile (McDonagh et al., 2021). Acute lung edema is a life-threatening condition, with an estimated incidence of 75,000 to 83,000 cases per 100,000 individuals with heart failure and reduced ejection fraction. A trial indicated a very high prevalence of pulmonary edema, reaching 80% in patients with heart failure. This is a highly concerning condition, with a patient discharge rate of 74% and a one-year survival rate of 50%. The mortality rate after six years of follow-up reached 85% in patients with congestive heart failure. Men are typically more affected than women, and the elderly are at a higher risk of developing pulmonary edema (Flather et al., 2005).

Acute lung edema is characterized by sudden and distinct shortness of breath, which includes orthopnea (worsening dyspnea when the patient is lying down and improvement when sitting), paroxysmal nocturnal dyspnea (PND, sudden shortness of breath that awakens the patient at night), and dyspnea on exertion (DOE, which is exacerbated by physical activity) (Stotts et al., 2023, Arrigo et al., 2020). In this case, the symptoms that prompted the patient to visit the emergency department were severe shortness of breath that developed suddenly within hours. The dyspnea experienced while lying down improved when the patient was in a sitting position, worsened with light activities over the past week, and was severe enough to wake the patient at night. These complaints align with the symptoms of acute lung edema, which may be due to suspected increased left atrial pressure and mitral valve regurgitation, to be evaluated through echocardiography. This leads to increased hydrostatic pressure in the pulmonary capillaries, elevating fluid filtration rate from the capillaries into the pulmonary interstitium, causing pulmonary stiffness and dyspnea (Arrigo et al., 2020). If the pulmonary interstitium cannot accommodate the fluid accumulation, the pressures in the right ventricle and atrium will increase, leading to edema in the digestive system and extremities. This condition is often caused by respiratory infections, which are characterized by clinical symptoms such as fever, productive cough, and an elevated white blood cell count ( $> 4-11 \times 10^9/L$ ), especially

neutrophilic leukocytosis ( $>75\%$ ). Chronic obstructive pulmonary disease (COPD), asthma, and myocardial infarction can also contribute. This explains why the patient reported symptoms of nausea and vomiting, which are signs of gastrointestinal congestion due to lung infection, accompanied by brownish sputum and dyspnea that began a week ago, along with fever.

The most common precipitants and etiologies of acute heart failure overall include acute coronary syndrome, arrhythmias (especially atrial fibrillation), infections (particularly respiratory infections), uncontrolled hypertension, hypertensive heart disease, and noncompliance with dietary and medication recommendations. It is important to note that in the majority of patients (40-50%), no identifiable trigger can be found, while a combination of several factors is observed in 5-20% of patients (Arrigo et al., 2020).

In this case, the patient and their family have a history of hypertension, diabetes mellitus, asthma, and smoking. Other comorbidities such as diabetes and asthma (obstructive lung disease) also play a role as precipitating factors. In the development of acute heart failure, particularly acute lung edema, which has a poor prognosis and necessitates hospitalization.

In the study by (Aparna et al., 2022), it was explained that 26% of patients with acute heart failure have comorbidities (Current trends, 2022). This high number of comorbidities contributes to an inpatient occurrence rate 5.8 times greater due to acute heart failure, as stated in the research by Meireles (MA et al., 2020), which indicates that if there are  $\geq$  three comorbidities present in acute heart failure, the odds ratio is 5.81, with a 95% CI of 2.77 - 12.16,  $p < 0.001$  (Meireles et al., 2020).

In addition to the findings of symptoms and medical history supporting the condition of acute lung edema, the findings from vital signs and physical examinations also elucidate the profile of a patient with acute lung edema. The patient presented with hypertensive crisis (200/110 mmHg), accompanied by symptoms and signs of pneumonia, such as shortness of breath and fever (respiratory rate of 34 breaths per minute, temperature of  $37.8^\circ\text{C}$ , and SpO<sub>2</sub> of 93%). The contribution of precipitating factors related to infection is only 5-10%. The largest precipitating factor in this patient is uncontrolled hypertension (Asghar et al., 2016, Meireles et al., 2020).

Physical examination revealed distension of the jugular veins, indicating increased pressure in the right atrium. Additionally, rhonchi and wheezing were found. These findings can suggest several diagnoses, including pneumonia and exacerbation of asthma, which are considered differential diagnoses alongside acute lung edema. However, the presence of cardiac enlargement, evidenced by the displacement of the left heart border to the left anterior axillary line and the discovery of pitting edema in both lower extremities, indicates that the patient's issues involve cardiac function, leaning the diagnosis towards acute heart failure with acute lung edema. The diagnoses of pneumonia, asthma exacerbation, or even acute respiratory distress syndrome (ARDS) cannot be completely ruled out and should be considered as differential diagnoses.

Routine blood tests revealed hemoglobin and hematocrit levels exceeding normal values. Epidemiologically, patients with acute heart failure are typically associated with anemia, but in this case, the results were contrary. This can be attributed to relative dehydration due to fluid shifting from the intravascular space to the interstitial or alveolar spaces, resulting in increased red blood cell concentration in the blood and, consequently, elevated hematocrit and hemoglobin levels (Meireles et al., 2020, Figueras and Weil, 1978). This phenomenon has also been observed in case reports concerning patients with acute lung edema and community-acquired pneumonia, as described by (Wibowo A. et al., 2024).

A point of concern is that the leukocyte count for this patient was only 9,000/uL. This laboratory finding does not strongly support the occurrence of pneumonia; however, the limitation of this case is that a complete leukocyte count was not performed, and only the D-Dimer test was conducted, which returned normal results. Atypical pneumonia or viral infections can present with normal leukocyte levels. Research by Muller B. et al. indicated that among 545 patients presenting with dyspnea and cough, 373 were diagnosed with community-acquired pneumonia, yielding a leukocyte count of  $12.9 \pm 6.7$  ( $\times 10^9/L$ ). This study supports that the underlying conditions include coronary artery disease, hypertensive heart disease, and congestive heart failure (Müller et al., 2007).

In this case, the potassium level was found to be 3.2 mmol/L, categorized as mild hypokalemia.

According to a cohort study by Perez PC et al., patients with hypokalemia at the time of hospital admission did not show a higher mortality rate during hospitalization when defined as serum potassium levels  $< 3.5$  mEq/L. Potassium levels  $< 3.5$  mEq/L are more specific in identifying heart failure patients with a high-risk profile, although this remains a hypothesis. However, persistent hypokalemia from admission to discharge is associated with higher mortality rates, which may be explained by more severe heart failure episodes, greater diuretic requirements, and significant potassium loss through urine (Caravaca et al., 2022).

Arterial blood gas analysis also indicated respiratory alkalosis in the patient. Acute heart failure patients at high risk often show respiratory alkalosis; however, only acidosis is associated with worse clinical outcomes. A decrease in serum bicarbonate levels is also a significant predictor of long-term mortality in high-risk patients with acute heart failure (Park et al., 2015).

Furthermore, cardiac enzyme tests were found to be within normal limits, allowing for the exclusion of conditions such as coronary artery disease. The ECG examination only revealed tachycardia and left ventricular hypertrophy (LVH) consistent with the Peguero-Lo Presti criteria (Yu et al., 2021). The ECG findings were supported by chest X-ray results, which showed hypertrophy of the left ventricle and prominent opacities in the central region, resembling the "batwings appearance" typically seen in alveolar edema (Siwik et al., 2023).

Echocardiography showed a left ventricular ejection fraction of 67%, indicating that the systolic function of the heart is still functioning well. However, there was also a decrease in diastolic function, left atrial dilation, and mild regurgitation in the tricuspid, aortic, and pulmonary valves. In the study by Militaru et al., it was found that echocardiographic parameters in acute lung edema are most frequently observed in the HFrEF group (44.2%), while only 21.5% of patients experience acute lung edema in the HFpEF group (Militaru et al., 2017).

It's important to note that even though the ejection fraction in this patient was normal, it is known that patients with an ejection fraction  $> 50\%$  can have minimal impairment in systolic function, which may only be detected through Doppler tissue imaging or myocardial strain



imaging (Harada et al., 2022). Diastolic dysfunction and increased filling pressures can be common and specific characteristics in patients with acute lung edema. In some cases, diastolic dysfunction becomes the primary pathophysiological mechanism underlying cardiac damage in HFpEF (Militaru et al., 2017). The increase in left atrial dimension can be expressed by indexed volume, and in this patient, a Left Atrial Volume Index (LAVI) of 41 mL/m<sup>2</sup> was found. Furthermore, mitral regurgitation can indicate remodeling of the left atrium. Left atrial dilation is also strongly associated with pulmonary hypertension. Although mild aortic regurgitation (ARVCD < 1mm) suggests a low likelihood of pulmonary hypertension, many patients with HFpEF develop pulmonary hypertension during exercise, which typically goes undetected at rest. Exercise-induced pulmonary hypertension can occur even at light exercise intensity in patients with HFpEF (Harada et al., 2022).

In this patient, the presence of left ventricular hypertrophy (LVH), left atrial dilation, and regurgitation in the pulmonary and tricuspid valves are significant prognostic indicators for the progression of heart failure.

In the study by Bajwa EK et al. concerning NT-proBNP in ARDS patients, it was found that seventy patients (40%) died with NT-proBNP levels at 3181 ng/L (interquartile range 723–9246 ng/L). NT-proBNP levels were significantly higher among patients who did not survive ( $p < 0.0001$ ). The optimal NT-proBNP cutoff level to predict mortality was 6813 ng/L, with a sensitivity of 80% and specificity of 51%, yielding an odds ratio of 4.24 (95% confidence interval 2.17–8.27,  $p = 0.0001$ ) (Bajwa et al., 2008).

In this patient, the NT-proBNP level was found to be 2333 pg/mL. According to the Japanese Heart Failure Society (JHFS) 2017 guidelines, an NT-proBNP level above 900 suggests a high likelihood of heart failure requiring urgent management and comprehensive supportive care (Tsutsui et al., 2019). Elevated NT-proBNP levels in patients with respiratory distress syndromes such as ARDS align with the severity of the syndrome and the potential for morbidity and mortality, indicating this biomarker's prognostic utility in such conditions.

Initial management of acute heart failure, particularly acute lung edema, was conducted in the emergency department (ED). According to

the JHFS 2017 guidelines, the initial treatment for acute lung edema includes diuretics and vasodilators. In this case, furosemide was administered at 5 mg/hour, and isosorbide dinitrate was given as a vasodilator at 1 mg/hour, titrated up to achieve a target systolic blood pressure of 150-160 mmHg (Tsutsui et al., 2019), so it was given in the acute setting in the ED and its continuation will be reconsidered after a HFpEF diagnosis is confirmed. Following the management guidelines for acute heart failure in the ED, the patient showed clinical improvement with stable hemodynamics requiring observation. Given the suspected asthma, treatments with salbutamol, ipratropium bromide, and methylprednisolone were administered. Due to complaints of fever and nausea, symptomatic management included paracetamol, ondansetron, and omeprazole.

In the ICVCU, lisinopril (an ACE inhibitor) and bisoprolol (a beta-blocker) were given. ACE inhibitors and beta-blockers are beneficial in heart failure management, particularly in HFrEF. While ACE inhibitors do not yield significant clinical outcomes in HFpEF ( $P=0.35$ ) (Darraj et al., 2023). they have a cardioprotective role, and beta-blockers have been shown to reduce mortality or cardiovascular hospitalization by 14% ( $P=0.04$ ) (Darraj et al., 2023). In this case, the administration of an ACE inhibitor and beta-blocker resulted in stable hemodynamic clinical outcomes (McDonagh et al., 2023).

Because of the persistent productive cough, the patient was treated symptomatically with N-acetylcysteine as a mucolytic agent and received the fluoroquinolone antibiotic levofloxacin. Fluoroquinolones can be a potential alternative treatment for individuals with penicillin sensitivity. Prompt antibiotic administration in cases of community-acquired pneumonia can prevent sepsis and allow for earlier hospital discharge. Several studies have shown that delays in antibiotic therapy before hospital admission are associated with poor clinical outcomes and increased mortality (Darraj et al., 2023). Given the patient's low potassium level, which is still classified as mild hypokalemia, potassium-sparing diuretics (KSR tablets) were administered along with spironolactone to prevent further hypokalemia. Notably, spironolactone has been associated with improved left ventricular systolic function, as assessed by LV longitudinal strain (Harada et al., 2022).

#### 4. CONCLUSION

In conclusion, we want to highlight an uncommon case of HFpEF in a patient presenting with acute pulmonary edema. Despite the preserved systolic function, we were able to improve the patient's condition in an emergency setting by administering medical therapy typically used for treating HFrEF.

#### DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

#### CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

#### ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

#### COMPETING INTERESTS

The authors have declared that no competing interests exist

#### REFERENCES

- Arrigo, M., Jessup, M., Mullens, W., Reza, N., Shah, A. M., Sliwa, K., et al. (2020). Acute heart failure. *Nature Reviews Disease Primers*, 6(1), 16.
- Asghar, N., Ahmad, S., Nazim, M., Ilyas, H. M. F., & Ahmad, M. N. (2016). Acute heart failure patients: Clinical profile. *Professional Medical Journal*, 23(10), 1281–1288.
- Bajwa, E. K., Januzzi, J. L., Gong, M. N., Thompson, B. T., & Christiani, D. C. (2008). Prognostic value of plasma N-terminal probrain natriuretic peptide levels in the acute respiratory distress syndrome. *Critical Care Medicine*, 36(8), 2322–2327.
- Caravaca Pérez, P., González-Juanatey, J. R., Nucho, J., Morán Fernández, L., Lora Pablos, D., Alvarez-García, J., et al. (2022). Serum potassium dynamics during acute heart failure hospitalization. *Clinical*

- Research in Cardiology*, 111(4), 368–379.
- Cleland, J. G., Tendera, M., Adamus, J., Freemantle, N., Polonski, L., & Taylor, J. (2006). The perindopril in elderly people with chronic heart failure (PEP-CHF) study. *European Heart Journal*, 27, 2338–2345.
- Current trends in prescription pattern and risk factor analysis of patients admitted with acute heart failure in a tertiary care teaching hospital. (2022). *Journal of Pharmaceutical Negative Results*, 4, 955–964.
- Darraj, A., Hudays, A., Hazazi, A., Hobani, A., & Alghamdi, A. (2023). The association between emergency department overcrowding and delay in treatment: A systematic review. *Healthcare (Basel)*, 11(3), 385.
- Felker, G. M., & Teerlink, J. R. (2021). Diagnosis and management of acute heart failure. In *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine* (12th ed.).
- Figueras, J., & Weil, M. H. (1978). Blood volume prior to and following treatment of acute cardiogenic pulmonary edema. *Circulation*, 57(2), 349–355.
- Flather, M. D., Shibata, M. C., Coats, A. J., Van Veldhuisen, D. J., Parkhomenko, A., Borbola, J., et al. (2005). Randomized trial to determine the effect of nebivolol on mortality and cardiovascular hospital admission in elderly patients with heart failure (SENIORS). *European Heart Journal*, 26, 215–225.
- Harada, T., Kagami, K., Kato, T., & Obokata, M. (2022). Echocardiography in the diagnostic evaluation and phenotyping of heart failure with preserved ejection fraction. *Journal of Cardiology*, 79(6), 679–690.
- Heidenreich, P. A., Bozkurt, B., Aguilar, D., et al. (2022). 2022 AHA/ACC/HFSA guideline for the management of heart failure: A report of the American College of Cardiology/American Heart Association joint committee on clinical practice guidelines. *Journal of the American College of Cardiology*, 79(17), e263–e421.
- McDonagh, T. A., Metra, M., Adamo, M., et al. (2021). 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. *European Heart Journal*, 42(36), 3599–3726.
- McDonagh, T. A., Metra, M., Adamo, M., Gardner, R. S., Baumbach, A., Böhm, M., et al. (2023). 2023 focused update of the 2021 ESC guidelines for the diagnosis and

- treatment of acute and chronic heart failure. *European Heart Journal*, 44(37), 3627–3639.
- Meireles, M. A., Golçalves, J., & Neves, J. (2020). Acute heart failure comorbidome: The impact of everything else. *Acta Medica Portuguesa*, 33(2), 109–115.
- Militaru, C., Deliu, R., Donoiu, I., et al. (2017). Echocardiographic parameters in acute pulmonary edema. *Current Health Sciences Journal*, 43(4), 317-324.
- Müller, B., Harbarth, S., Stolz, D., Bingisser, R., Mueller, C., Leuppi, J., et al. (2007). Diagnostic and prognostic accuracy of clinical and laboratory parameters in community-acquired pneumonia. *BMC Infectious Diseases*, 7, 10. <https://doi.org/10.1186/1471-2334-7-10>
- Park, J. J., Choi, D. J., Yoon, C. H., Oh, I. Y., Lee, J. H., Ahn, S., et al. (2015). The prognostic value of arterial blood gas analysis in high-risk acute heart failure patients: An analysis of the Korean Heart Failure (KorHF) registry. *European Journal of Heart Failure*, 17(6), 601–611.
- Reyes, E. B., Ha, J. W., Firdaus, I., et al. (2016). Heart failure across Asia: Same healthcare burden but differences in organization of care. *International Journal of Cardiology*, 223, 163–167.
- Siwik, D., Apanasiewicz, W., Żukowska, M., Jaczewski, G., & Dąbrowska, M. (2023). Diagnosing lung abnormalities related to heart failure in chest radiogram, lung ultrasound, and thoracic computed tomography. *Advances in Respiratory Medicine*, 91(2), 103–122.
- Stotts, C., Corrales-Medina, V. F., & Rayner, K. J. (2023). Pneumonia-induced inflammation, resolution and cardiovascular disease: Causes, consequences and clinical opportunities. *Circulation Research*, 132(6), 751-774.
- Tsutsui, H., Isobe, M., Ito, H., Okumura, K., Ono, M., Kitakaze, M., et al. (2019). JCS 2017/JHFS 2017 guideline on diagnosis and treatment of acute and chronic heart failure - Digest version. *Circulation Journal*, 83(10), 2084–2184.
- Wibowo, A., & Saputra, T. A. (2024). Rapid diagnosis and management of concomitant acute cardiogenic pulmonary edema and community-acquired pneumonia increase patient survival. *Medical Professionals Journal of Lampung*, 14(2), 371–375.
- Yu, Z., Song, J., Cheng, L., Li, S., Lu, Q., Zhang, Y., et al. (2021). Peguero-Lo Presti criteria for the diagnosis of left ventricular hypertrophy: A systematic review and meta-analysis. *PLOS One*, 16(1), e0246305. <https://doi.org/10.1371/journal.pone.0246305>

**Disclaimer/Publisher's Note:** The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of the publisher and/or the editor(s). This publisher and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.

© Copyright (2025): Author(s). The licensee is the journal publisher. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:

<https://pr.sdiarticle5.com/review-history/131524>