Jurnal Eduhealth
Volume 15 , Number 04, 2024, DOI 10.54209/eduhealth.v15i04
ESSN 2808-4608 (Online)
https://ejournal.seaninstitute.or.id/index.php/healt

Acute Decompensated Heart Failure+Atrial Fibrilation: Case Report

Ade Giriayu Anjani[1], Shofiatul Fajriyah[2]
[1,2]Departments Pharmacy, Institut Ilmu Kesehatan Bhakti Wiyata, Kediri, East Java

Article Info
Keywords:
Acute Decompensated Heart Failure,
Atrial Fibrilation,
Pharmacology therapy

ABSTRACT
Heart Failure is a health issue with high mortality and morbidity rates in both developed and developing countries, such as Indonesia. The prevalence of heart failure in Asia is generally similar to that reported in Europe (1–3%), while in Indonesia, the prevalence is reported to be greater than 5%. Heart failure increases among geriatric patients, affecting 6% of those aged 60-79 years and up to 14% of those over 80 years old. Acute Decompensated Heart Failure (ADHF) is the progressive worsening of symptoms and clinical signs of heart failure in patients who have been previously diagnosed with the condition. The underlying mechanisms of clinical deterioration in patients include increased congestion and disease progression. ADHF and atrial fibrillation (AF) often occur together and can lead to hemodynamic instability and death. AF is the most common supraventricular dysrhythmia in patients with ADHF, with a prevalence of 25%-40%. The combination of ADHF and AF results in adverse clinical outcomes, including prolonged hospitalization and increased mortality. A 50-year-old woman complained of shortness of breath accompanied by palpitations that started 10 days before hospital admission and worsened in the last 2 days. The patient has a history of an enlarged heart for the past 3 years. A transthoracic echocardiogram revealed atrial fibrillation with a rapid ventricular rate (RV) of 90-130 beats per minute, left ventricular dilation (LVIDd 5.5 cm), decreased right ventricular systolic function (TAPSE 1.4 cm), left atrial dilation (LAVI 64.45 ml/m²), and right atrial dilation (RA major 5.8 cm). The electrocardiogram showed atrial fibrillation, abnormal ST & T waves, and prolonged QT interval.

Corresponding Author:
Ade Giriayu Anjani
Institut Ilmu Kesehatan Bhakti Wiyata Kediri
ade.giriayu@iik.ac.id

INTRODUCTION

Heart failure is a health issue with high mortality and morbidity rates in both developed and developing countries, such as Indonesia. The prevalence of heart failure in Asia is generally similar to that reported in Europe (1—3%), while Indonesia's prevalence is reported to be over 5%. Heart failure increases in geriatric patients, with a prevalence of 6% in those aged 60-79 years and up to 14% in those over 80 years old (PERKI, 2023). *Acute Decompensated Heart Failure* (ADHF) is the progressive worsening of symptoms and clinical signs of heart failure in patients who have been previously diagnosed with the condition. The underlying mechanisms of clinical deterioration in

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patients include increased congestion and disease progression. Certain conditions can accelerate the progression of clinical deterioration in decompensated heart failure, such as *atrial fibrillation* (AF) with a rapid ventricular response. The clinical profile may be caused by progressive congestion with or without hypoperfusion (PERKI, 2023). *Acute Decompensated Heart Failure* (ADHF) and atrial fibrillation often occur together and can lead to hemodynamic instability and death. AF is the most common supraventricular dysrhythmia in patients with ADHF, with a prevalence of 25%-40%. The combination of ADHF and AF results in adverse clinical outcomes, including prolonged hospitalization and increased mortality (Joshua *et al.,* 2023). Atrial fibrillation is often found in patients with heart failure because the two conditions share similar pathophysiology and risk factors. Stable heart failure can become unstable with the onset of new AF or an increased ventricular rate of AF. Conversely, acute heart failure can also cause a change in the patient's rhythm from sinus to AF. Cardiac remodeling, neurohormonal activation, and worsening left ventricular function related to increased ventricular rate are the underlying mechanisms. The prognosis for patients withAF and heart failure can be found in those with heart failure with a reduced ejection fraction (<40%) or with a normal ejection fraction (>50%). The general management of patients with AF and heart failure is not much different from patients with heart failure, considering appropriate drug selection and anticoagulant use (PERKI, 2019).

Worsening heart failure can lead to increased atrial stretch and sympathetic tone. In patients diagnosed with *atrial fibrillation* (AF), additional treatment is required. The duration of AF episodes will affect the feasibility and potential for spontaneous cardioversion, pharmacological, or electrical treatment, and the selection of agents to control rhythm and rate. AF can trigger heart failure in previously stable patients or exacerbate heart failure and trigger acute AF episodes. In patients with this condition, thepotential for early recovery of sinus rhythm increases if heart failure symptoms can be controlled. When patients experience AF that is not immediately recognized, they may gradually progress to a state of *Acute Decompensated Heart Failure* (ADHF) and then present with severe symptoms. The interaction between ADHF and AF is highly complex,as AF can worsen heart failure, but heart failure also increases the risk of AF. The choicebetween rate control or rhythm control reflects the patient's symptoms and the potential for better ventricular function with sinus rhythm (Heidenreich *et al.*, 2022).

Although shock can temporarily restore sinus rhythm, the recurrence rate in patients still experiencing decompensation will be very high. Even if the resting heart rate in AF patients is between 60 and 100 bpm, a rate below 100 bpm cannot be achieved in patients with ADHF until volume overload and hypoxia are corrected. Therefore, the appropriate target is a heart rate below 120 bpm during the first few hours of treatment. Managing AF rate or rhythm control and maintaining stable blood pressure in ADHF conditions is complicated given the varying physiology of patients (Heidenreich *et al.*, 2022). The general goals of therapy for *Acute Decompensated Heart Failure* (ADHF) are to alleviate symptoms, restore oxygenation, improve organ perfusion, and limit damage to the heart and kidneys. In patients with sinus rhythm and ADHF, the use of vasodilators, oxygen, diuretics, positive

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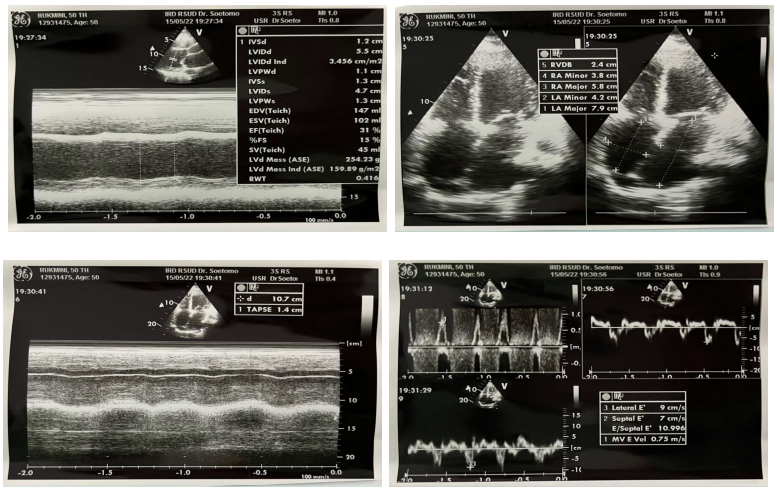
inotropes, and mechanical devices to support ventilation or cardiac output are the foundation of therapy. The combination of AF and ADHF is often encountered, but there is limited published research on this topic (Heidenreich *et al*., 2022).

Case Illustrations

A 50-year-old woman weighing 70 kg and measuring 155 cm in height with a BMI of 25.71 kg/m² complained of shortness of breath accompanied by heart palpitations that started 10 days before hospital admission and worsened in the past 2 days. She also experienced dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. The patient has a history of an enlarged heart for the past 3 years, with regular hospital check-ups and occasional visits to a general practitioner. However, because she had no complaints in the last 3 years, she decided to stop taking medication. The patient's medical history includes hypertension with the highest systolic blood pressure of 150 mmHg for more than 10 years and an enlarged heart for the last 3 years. The patient had been taking furosemide 40 mg orally once daily and bisoprolol 2.5 mg orally once daily. She has no history of allergies. The examination revealed atrial fibrillation with a rapid ventricular rate of 90-130 beats per minute, left ventricular dilation (LVIDd 5.5), eccentric left ventricular hypertrophy (LVDMi 159.8 g/m²; RWT 0.415), decreased left ventricular systolic function(EF by TECH 31%, by mod A4C 32%, by mod A2C 33%, by Biplane 32%), and global hypokinesia on segmental left ventricular analysis. Left ventricular diastolic function was reduced, and right ventricular systolic function was decreased (TAPSE 1.4 cm). The left atrium was dilated (LAVI 64.45 ml/m²), and the right atrium was dilated (RA major 5.8 cm, RA minor 3.8 cm) on transthoracic echocardiogram examination. The electrocardiogram showed atrial fibrillation, abnormal ST & T waves, and prolonged QT interval. The primary diagnosis was *acute decompensated heart failure* (ADHF) with a Forrester wet and warm profile, and the secondary diagnoses included dilated cardiomyopathy, atrial fibrillation with moderate rapid ventricular response (CHA2DS2VASC score 3, HAS-BLED score 1),and right ventricular failure. Subsequently, the patient developed hypokalemia and an ischemic stroke differential diagnosis of *transient ischemic attack* (TIA).



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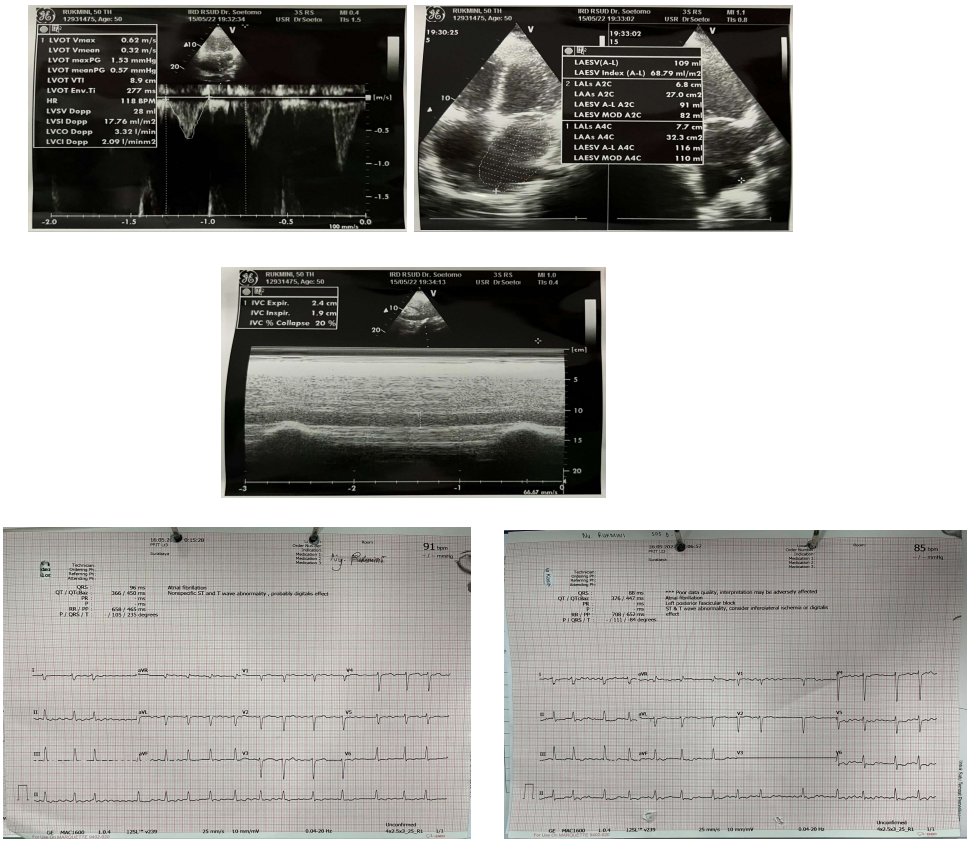
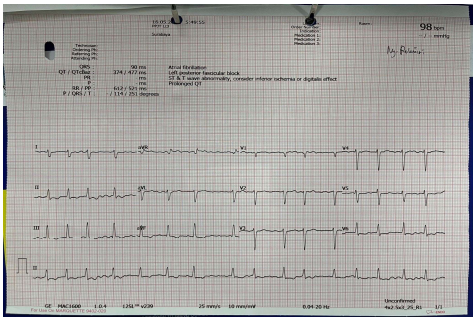


Figure 1. Transthoracic Echocardiogram

(a) (b)



(c)

Figure 2. Electrocardiogram (a)00.15 WIB; (b)03.06 WIB ; (c)05.49

WIB Clinical data showed the patient had a body temperature of 36.5°C, a heart rate of 130 beats per minute, a respiratory rate of 28 breaths per minute, and blood pressure that was relatively normal at 120/89 mmHg. The patient had an SpO2 of 97%, a Glasgow Coma Scale (GCS) of E4V5M6, a mean arterial pressure (MAP) of 83, and a quick Sequential Organ Failure Assessment (qSOFA) score of 1. Laboratory data revealed hemoglobin at

13.5 g/dL, hematocrit at 41.8%, platelet count at 271,000 µ/L, blood urea nitrogen (BUN) at 9 mg/dL, serum creatinine at 0.7 mg/dL, glomerular filtration rate (GFR) at

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88.6 mg/min, serum glutamic oxaloacetic transaminase (SGOT) at 34 U/L, serum glutamic pyruvic transaminase (SGPT) at 18 U/L, and serum albumin at 3.83 g/dL. The patient's coagulation factors showed an international normalized ratio (INR) of 1.01, prolonged prothrombin time (PTT) of 14.3 seconds, and activated partial thromboplastin time (aPTT) of 26.4 seconds. The patient's lipid levels were LDL 207 mg/dL, HDL 31 mg/dL, cholesterol 255 mg/dL, and triglycerides 110 mg/dL, indicating hyperlipidemia marked by elevated LDL and total cholesterol and decreased HDL levels. The patient's arterial blood gas analysis revealed HCO3- at 35.9 mmol/L, FiO2 at 21%, SO2C at 98%, base excess in extracellular fluid (BEecf) at 13.4 mmol/L, TCO2 at 37.2 mmol/L, pO2 at 188 mmHg, pCO2 at 42 mmHg, and blood gas pH at 7.54, indicating a basic pH level and showing metabolic alkalosis. HBsAg was non-reactive, and C-reactive protein (CRP) was 0.23 mg/dL. Upon initial hospital admission, the patient received therapy with 0.9% NaCl, furosemide at 5 mg/hour via pump, candesartan 8 mg orally every 24 hours, warfarin 4 mg every 24 hours in 500cc KN2, and digoxin 0.25 mg intravenously as a bolus every 24 hours.

Discussion

A 50-year-old woman came to Dr. Soetomo Regional Hospital in Surabaya, complaining of shortness of breath accompanied by palpitations that started 10 days before hospital admission and worsened in the last 2 days. The patient has a history of an enlarged heart for the past 3 years. A transthoracic echocardiogram revealed atrial fibrillation with a rapid ventricular rate (RV) of 90-130 beats per minute, left ventricular dilation (LVIDd 5.5 cm), decreased right ventricular systolic function (TAPSE 1.4 cm), left atrial dilation (LAVI 64.45 ml/m²), and right atrial dilation (RA major 5.8 cm). The electrocardiogram showed atrial fibrillation, abnormal ST & T waves, and prolonged QT interval. The initial diagnosis was *acute decompensated heart failure* (ADHF) with a Forrester wet and warm profile, and the secondary diagnoses included dilated cardiomyopathy, moderate atrial fibrillation with rapid ventricular response (CHA2DS2VASC score 3, HAS-BLED score 1), and right ventricular failure. ADHF is the progressive worsening of symptoms and clinical signs of heart failure in patients who have been previously diagnosed with the condition. Certain conditions can accelerate the clinical deterioration of ADHF, such as atrial fibrillation with a rapid ventricular response. The clinical profile may be caused by progressive congestion with or without hypoperfusion (PERKI, 2023).

The shortness of breath experienced by the patient began 10 days before hospital admission and worsened over the last 2 days, accompanied by palpitations without chestpain. As a result, the patient was given furosemide therapy via a pump at a dose of 5 mgper hour, which was reduced to 2.5 mg per hour after 2 days. Loop diuretics can provide vasodilator and rapid diuretic effects, thereby reducing the heart's preload burden on theleft ventricle and alleviating symptoms of shortness of breath or dyspnea (Heidenreich *et al.*, 2022). Furosemide was administered with an IV pump at 5 mg/hour (120 mg/day). The dose for ADHF conditions is an IV bolus of 40–80 mg. The effectiveness of furosemide therapy is assessed by the daily urine volume, which should

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indicate a negative fluid balance. Diuretics increase the renal excretion of salt and water to manage fluid overload and congestion in most AHF patients. The use of diuretics is favored for their rapid onset of action. The oral or intravenous dose of furosemide is 1–2 times daily or administered intravenously at ≥20–40 mg (McDonagh *et al.,* 2021). Treatment starts with low doses (furosemide 20 to 40 mg, bumetanide 1 mg, torsemide 10 to 20 mg). The dose can be doubled every 2-4 hours up to the maximum recommended dose (Heidenreich *et al*., 2022).

Furosemide is administered in combination with candesartan. Candesartan is an antihypertensive of the ARB class that works by blocking the AT1 receptor, resulting in vasodilation and increased excretion of sodium and fluid (reducing plasma volume). The condition of *acute decompensated heart failure* (ADHF) will trigger the *renin-angiotensin-aldosterone* (RAA) system to maintain cardiac output. However, excessive compensation can negatively affect the patient's condition. Therefore, the administration of candesartan helps control cardiac function and reduces the potential for morbidity and mortality (Heidenreich *et al*., 2022). The combination of furosemide and candesartan is considered appropriate according to ESC guidelines for ADHF in the warm and wet category, which requires vasodilators and diuretics to lower intravascular pressure, alleviating dyspnea in patients (McDonagh *et al.,* 2021). The administration of ARBs alsohelps reduce morbidity and mortality in patients with ADHF (Heidenreich *et al*., 2022).

The patient also complained of palpitations, and the ECG results showed *atrial fibrillation* (AF), a typical supraventricular tachyarrhythmia, with uncoordinated atrial activation resulting in impaired atrial mechanical function. On the electrocardiogram, AF is characterized by the absence of consistent P waves, replaced by fibrillation waves that vary in amplitude, shape, and duration. In normal atrioventricular node (AV) function, AF is usually followed by an irregular and often rapid ventricular response (PERKI, 2019). Atrial fibrillation is characterized by irregular atrial electrical activation and uncoordinated atrial contractions. The patient's heart rate was initially elevated and returned to normal after 2days of treatment. The patient was given digoxin therapy at 0.25 mg intravenously every24 hours as a bolus upon hospital admission and warfarin at 4 mg every 24 hours for 3 days. Digoxin administration on the first day was used to quickly address AF. In heart failure patients with AF, digoxin can be used to slow the rapid ventricular rate. It increases myocardial contractility to enhance cardiac output and decreases AV conduction to slow the ventricular rate in atrial fibrillation. The initial dose of digoxin is 0.25 mg/day (PERKI, 2019). The digoxin dose given to the patient was appropriate. Next, warfarin was administered as an anticoagulant. Warfarin works by inhibiting vitamin K synthesis in the liver, affecting clotting factors II, III, IX, and X by converting glutamic acid residues into gamma-carboxyglutamic acid residues. Additionally, it helps control ventricular rate and prevent systemic embolism. The recommended dose of warfarin is 1–6 mg/day, targeting an INR of 2-3 (PERKI, 2019). The dose for this patient was consistent with PERKI's recommendation of 4 mg/day. Warfarin was given here to prevent stroke in atrial fibrillation patients following the 2019 PERKI

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recommendations, where patients with a CHA2DS2VASc stroke risk score of 1 or >2 are indicated for oral anticoagulants. A CHA2DS2VASc score of 3 indicates oral anticoagulant use. The effectiveness of warfarin therapy is assessed by the INR value, with the target for AF patients being 2-3. Stroke prevention is effective when the time in therapeutic range (TTR) is >70%. TTR is the proportion of time when an INR of 2-3 is achieved compared to the total time on warfarin. Therefore, monitoring the INR is necessary for dose adjustment (PERKI, 2019). For the next 2 days, the patient was given bisoprolol tablets at 1.25 mg every 24 hours. Bisoprolol is a cardioselective β-blocker antihypertensive. The use of bisoprolol in AF for rate control aligns with AF therapy management (Heidenreich *et al*., 2022).

The patient also had the potential for an ischemic stroke versus transient ischemic attack (TIA) with decreased consciousness and left hemiparesis, suspecting an acute stroke. Therefore, the patient was given mecobalamin and citicoline intravenously as neuroprotectants to protect nerve cells from damage due to stroke. Mecobalamin is a form of vitamin B12 used to treat peripheral neuropathy caused by vitamin B12 deficiency (Kleindorfer et al., 2021). Citicoline acts as a neuroprotectant. Citicoline can reduce the severity of stroke symptoms by increasing acetylcholine production and reducing fatty acid accumulation in the damaged nerve area, thereby decreasing the infarct size (Dávalos et al., 2012).

Laboratory tests showed an increase in lipid profile, and blood gas parameters indicated metabolic alkalosis. Therefore, it is recommended to address the increased lipid profile by administering high-intensity statins such as atorvastatin $\geq$ 40 mg or rosuvastatin 20 mg to lower lipid levels. According to the ESC Guideline, high-intensity statin therapy can reduce LDL-C by $\geq$ 50%, while moderate-intensity statin therapy can reduce LDL-C by 30% to <50% (Colin *et al.,* 2020). Monitoring the patient's lipid profile and administering sodium bicarbonate are suggested to manage metabolic alkalosis.

CONCLUSION

The interaction between acute decompensated heart failure (ADHF) and atrial fibrillation (AF) is very complex, as AF can worsen heart failure, but heart failure also increases the risk of AF. The patient was diagnosed with ADHF and AF, having previously been diagnosed with heart disease, and had not consistently taken medication, which worsened the patient's clinical condition. After receiving treatment at Dr. Soetomo Regional Hospital in Surabaya, the patient's clinical condition improved. It is important to understand the factors influencing the worsening of the patient's heart disease and the prevention strategies. It is crucial to understand the risks, treatment, and prevention of progression to stroke.

ACKNOWLEDGEMENT

We would like to thank Mochamad Yusuf Alsagaff, doctor of the Cardiology and Vacular Medicine from Dr. Soetomo General Hospital, for the opportunity to fulfil our accomplishment on this case report.

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