Acute Decompensated Heart Failure+Atrial Fibrilation: Case Report

by Perpustakaan IIK Bhakti Wiyata

Submission date: 12-Jul-2025 11:20AM (UTC+0700)
Submission ID: 2501139310
File name: IASI_Acute_Decompensated_Heart_Failure_-_Ade_Giriayu_Anjani.docx (631.17K)
Word count: 2766
Character count: 15357

Acute Decompensated Heart Failure+Atrial Fibrilation: Case Report

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 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.

INTRODUCTION

Heart failure is a medical problem with elevated rates of mortality and morbidity in both developed and developing nations, including Indonesia. The occurrence of heart failure in Asia is typically comparable to that observed in Europe (1–3%), but Indonesia's prevalence is noted to exceed 5%. Heart failure rises among older patients, with a prevalence of 6% in individuals aged 60-79 and as high as 14% in those over 80 years old (PERKI, 2023). ADHF refers to the gradual deterioration of symptoms and clinical indicators of heart failure in individuals who have already received a diagnosis of the illness. The fundamental causes of clinical decline in patients consist of heightened congestion and the advancement of the disease. Certain factors can speed up the advancement of clinical decline in decompensated heart failure, like AF with a rapid ventricular response. The clinical profile might result from ongoing congestion, with or without hypoperfusion (PERKI, 2023). HF is a medical problem with elevated rates of mortality and morbidity in both developed and developing nations, including Indonesia. The occurrence of heart failure in Asia is typically comparable to that observed in Europe (1–3%), but Indonesia's prevalence is noted to exceed 5%. Heart failure rises among older patients, with a prevalence of 6% in individuals aged 60-79 and as high as 14% in those over 80 years old (PERKI, 2023). ADHF refers to the gradual deterioration of symptoms and clinical indicators of heart failure in individuals who have already received a diagnosis of the illness. The fundamental causes of clinical decline in patients consist of heightened congestion and the advancement of the disease. Certain factors can speed up the advancement of clinical decline in decompensated heart failure, like AF with a rapid ventricular response. The clinical profile might result from ongoing congestion, with or without hypoperfusion (PERKI, 2023).

Deteriorating heart failure may result in heightened atrial distension and sympathetic activity. Patients with AF need further treatment. The length of AF episodes will influence the

possibility and likelihood for spontaneous cardioversion, pharmacological, or electrical therapies, as well as the choice of medications to manage rhythm and rate. AF can initiate heart failure in patients who were previously stable or worsen existing heart failure and provoke acute AF episodes. In individuals with this condition, the likelihood of regaining sinus rhythm early improves if heart failure symptoms are managed effectively. When patients have AF that goes unrecognized initially, they can slowly advance to ADHF and later show significant symptoms. The relationship between ADHF and AF is very intricate, as AF has the potential to exacerbate heart failure, while heart failure simultaneously elevates the risk of developing AF. The decision between rate control or rhythm control depends on the patient's symptoms and the possibility of improved ventricular function with sinus rhythm (Heidenreich et al., 2022).

Even though shock can briefly restore sinus rhythm, the recurrence rate in patients still undergoing decompensation will be extremely high. Although the resting heart rate in AF patients ranges from 60 to 100 bpm, patients with ADHF cannot have a rate under 100 bpm until volume overload and hypoxia are resolved. Consequently, the ideal goal is a heart rate under 120 bpm in the initial hours of treatment. Controlling AF rate or rhythm and ensuring stable blood pressure in ADHF patients is challenging due to their differing physiology (Heidenreich et al., 2022). The primary objectives of treatment for ADHF are to relieve symptoms, enhance oxygenation, boost organ perfusion, and reduce harm to the heart and kidneys. In individuals with sinus rhythm and ADHF, the foundation of treatment includes vasodilators, oxygen, diuretics, positive inotropes, and mechanical devices to assist with ventilation or cardiac output. The occurrence of AF and ADHF together is common, yet there is scarce published research regarding this subject (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 3 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 warmprofile, 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).

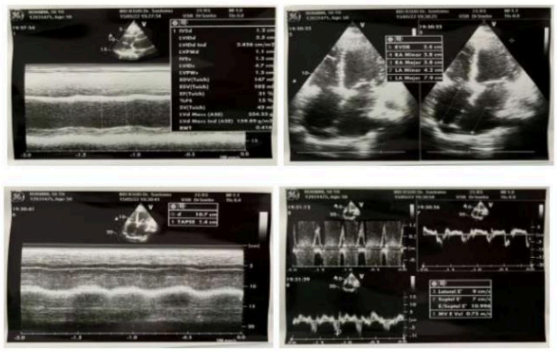
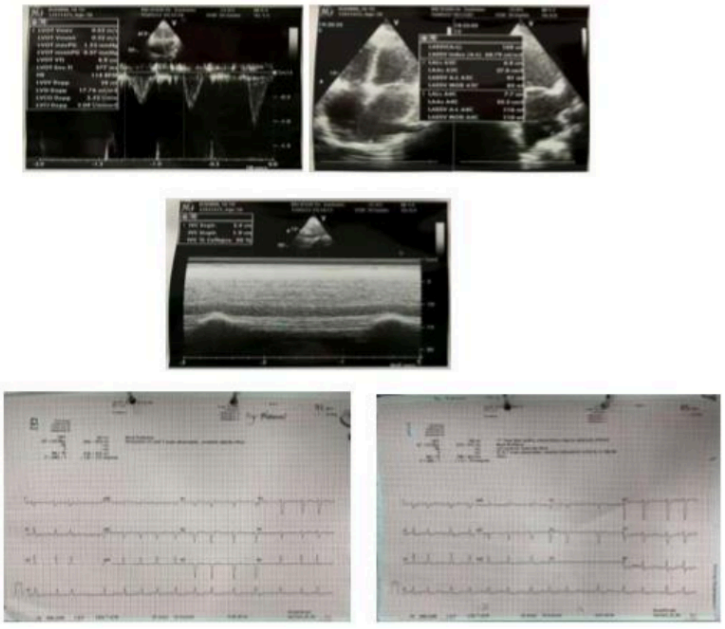
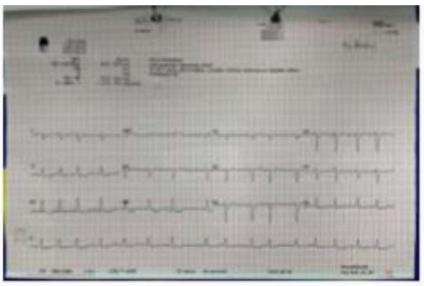


Figure 1. Transthoracic Echocardiogram



(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 temperature of 36.5°C, a HR of 130 beats per minute, a RR of 28 breaths per minute, and BP that was relatively normal at 120/89 mmHg. The patient had an SpO2 of 97%, a GCS of E4V5M6, MAP of 83, and a qSOFA score of 1. Laboratory data revealed hemoglobin at 13.5 g/dL, hematocrit at 41.8%, platelet count at 271,000 µ/L, BUN at 9 mg/dL, serum creatinine at 0.7 mg/dL, GFR at 88.6 mg/min, SGOT at 34 U/L, SGPT at 18 U/L, and serum albumin at 3.83 g/dL. The patient's coagulation factors showed an INR of 1.01, PTT of 14.3 seconds, and 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%, 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 etal., 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 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). Treatment starts offevolved with low doses (furosemide 20 to 40 mg, bumetanide 1 mg, torsemide 10 to 20mg). The dose may be doubled every 2-4 hours as much as the most encouraged 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 formorbidity 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 affected person also complained of palpitations, and the ECG results confirmed atrial fibrillation (AF), a normal supraventricular tachyarrhythmia, with uncoordinated atrial activation resulting in impaired atrial mechanical function. on the electrocardiogram, AF is characterised by using the absence of constant P waves, replaced by traumatic inflammation waves that change in amplitude, shape, and period. In normal atrioventricular node (AV) feature, AF is generally observed by an irregular and regularly rapid ventricular reaction (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 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 warfarintherapy 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 > 40 mg or rosuvastatin 20 mg to lower lipid levels. According to the ESC Guideline, high-intensity statin therapy can reduce LDL-C by > 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 preventionstrategies. It is crucial to understand the risks, treatment, and prevention of progression to stroke.

Acute Decompensated Heart Failure+Atrial Fibrilation: Case Report

ORIGINALITY REPORT

13%	8%	8%	%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	Joshua D. Niforatos, Michael R. Ehmann, Kamna S. Balhara, Jeremiah S. Hinson, Lukas Ramcharran, Katie Lobner, P. Logan Weygandt. "Management of atrial flutter and atrial fibrillation with rapid ventricular response in patients with acute decompensated heart failure: A systematic review", Academic Emergency Medicine, 2022 Publication	2%
2	ijconline.id Internet Source	2%
3	"Encyclopedia of Heart Diseases", Springer Science and Business Media LLC, 2011 Publication	2%
4	pulmonarychronicles.com Internet Source	2%
5	download.bibis.ir Internet Source	1%
6	academic.oup.com Internet Source	1%
7	archive.org Internet Source	1%
8	journals.innovareacademics.in Internet Source	1%
9	Miao Yu, Wenbin Wang, Wei Chen, Kexin Ye et al. "Diagnosis of Tropheryma whipplei	1%

pneumonia using targeted nanopore sequencing: a rare case report and literature review", Frontiers in Medicine, 2025
Publication

10	www.kcumb.edu Internet Source	1 %
11	Sonya G Gordon, Mark D Kittleson. "Drugs used in the management of heart disease and cardiac arrhythmias", Elsevier BV, 2008 Publication	<1 %
12	www.nice.org.uk Internet Source	<1 %
13	"Pathophysiology and Pharmacotherapy of Cardiovascular Disease", Springer Science and Business Media LLC, 2015 Publication	<1 %

Exclude quotes	On	Exclude matches	Off
Exclude bibliography	On		