



A CASE STUDY OF A 54-YEAR-OLD MAN WITH NYHA CLASS III, STAGE 2 HYPERTENSION, AND TYPE II DIABETES MELLITUS

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ABSTRACT

Background: Heart failure is the condition when the heart cannot pump enough blood to fulfill the body's needs. This syndrome is dangerous and can lead to death. The most common risk factors are hypertension, diabetes mellitus, and metabolic syndrome. **Case Presentation:** A 54-year-old man complained of shortness of breath for the last one month, which worsened during light activities and decreased when he was in a semi-sitting position. The patient had a history of hypertension and type-II diabetes mellitus for the last five years. On physical examination, the patient's blood pressure was 140/100 mmHg, respiratory rate was 25 bpm. There was paroxysmal nocturnal dyspnea, orthopnea, rales on both basal lung fields, dyspnea on exertion, ankle edema, a pan systolic murmur heard in the apex, ascites and ankle edema. Blood laboratory examination showed HbA1c 6%, chest X-ray and ECG showed cardiomegaly. **Conclusion:** The patient was diagnosed with NYHA Class III Heart Failure with stage 2 hypertension and type-2 diabetes mellitus and was treated with diuretics, ACE inhibitors, beta-blockers, low salt diet, restriction of fluid intake, and oral antidiabetics.

Keywords: *Diabetes mellitus, heart failure, hypertension*

BACKGROUND

Heart failure, one of an emerging epidemic about 25 years ago.¹ Based on the data from World Health Organization (WHO) in 2019, there are 17.9 million people who died because of cardiovascular disease (CVD), which represents 31% of mortality of the world, and 85% caused by heart failure and stroke.² The most recent data regarding CVD risk factors based on AHA 2019 are hypertension (45.6%), obesity in adults (39.6%), Low-Density Lipoprotein Cholesterol ≥ 130 mg/dl (28.5%), smoking (15.5%), and diagnosed-Diabetes Mellitus (9.8%).³ The prevalence of heart failure in Indonesia is increasing each year. The data from Riskesdas in 2013 shows the prevalence of heart failure in Indonesia is 0.3% and has increased 1.5% in 2018.^{4,5}

Heart failure is a condition when the heart is no longer able to pump blood to the tissues in sufficient quantities to keep up the body's metabolic needs. This ability can only occur with high cardiac filling pressures or both.⁶ Clinical manifestations of heart failure are characterized by shortness of breath and feeling tired quickly, jugular veins distention, hepatomegaly, splenomegaly, ascites, and peripheral oedema caused by structural or functional abnormalities of the heart.⁷ If the cardiac output is not sufficient to fulfil the body's needs, the heart will perform a compensatory mechanism. These mechanisms include the Frank-Starling mechanism,

ventricular hypertrophy, and neurohormonal activation.⁸

There are various conditions or comorbidities associated with an increased tendency to develop structural heart disease. The main risk factors for HF are hypertension, diabetes mellitus, metabolic syndrome, and atherosclerosis.⁹ The incidence of HF in patients with type II diabetes mellitus is between 9% and 22%, which is 4 times higher than the general population. The incidence of HF is also increasing in patients with hypertension, a collection of abnormalities caused by hypertension including left ventricular hypertrophy (LVH), systolic and diastolic dysfunction, and symptomatic heart failure.¹⁰

CASE PRESENTATION

A 54-year-old man came to Dr. Kariadi Semarang Hospital in May 2021 with shortness of breath for one month ago. The symptoms had progressively worse, triggered by light activity such as walking around and relieving when the patient is at rest or in a semi-sitting position. Shortness of breath was not accompanied by wheezing, not triggered by weather, dust, and emotions. The patient was more comfortable sleeping using two pillows. The patient also reported waking up suddenly at night due to shortness of breath, easy fatigue, appetite decreased, and weight decreased. The patient also complained that his bilateral lower



extremities were swelling with an abdominal enlargement.

The patient's past medical histories were hypertension and type II diabetes mellitus. He has had hypertension since approximately five years ago. However, the patient does not take medication regularly. When the patient headaches, he only took the medicine he bought himself at the shop and rested. The patient also has had diabetes since approximately five years ago. The patient routinely took a blood-sugar-lowering drug taken every morning after breakfast.

The patient's family history included hypertension and type II diabetes mellitus in his mother. His father had a heart disease. The patient also had two brothers who died in young age. The patient was a previous smoker for 15 years and had stopped smoking 1 year ago.

The physical *examination* revealed the general state looked moderately ill, *compos mentis*, blood pressure 140/100 mmHg, pulses 90x/min regular, respiratory rate 25x/minute, temperature 36.8°C The conjunctiva was not pale; there was no discharge from the nose or ears, the JVP on the normal value, and no masses in his neck. The right and left hemithorax movements were symmetrical when static and dynamic. On palpation, the right and left stem fremitus were normal. On percussion, there were resonant sounds throughout the lung fields. There were vesicular base sounds on lung auscultation, fine crackles on the right and left lungs below the fifth intra-costal space, and no wheezing. On heart palpation, ictus cordis was palpated at the sixth intra costal space 2 cm outward to the left midclavicular line, epigastric pulsations, sternal lift, and parasternal pulses found. Heart percussion; the upper border of the heart at the second intra-costal space on the left parasternal line, the right heart border at the fifth intra-costal space on the right parasternal line, left heart border at the sixth intra costal space 2 cm outward to the left midclavicular line with a flat cardiac waist. Cardiac auscultation revealed regular first and second heart sounds, a grade 3/6 pan systolic murmur heard in the apex, and no gallops. On abdominal examination, found normal bowel sounds, tympanic, shifting dullness. On extremities examination, pitting edema in both lower extremities was observed.

The laboratory test had shown the following results: hemoglobin 16.8 g/dL, hematocrit 49.7%,

erythrocytes 5.49 million/mm³, leukocytes 6.900/mm³, platelets 186.000/mm³, urea 40 mg/dl, creatinine 0.8 mg/dl, blood glucose 90 mg/dL, HbA1c 6.6%, albumin 3.9 mg/dL, AST 25 U/L, ALT 16 U/L, sodium 132 mmol/L, potassium 4.3 mmol/L, and chloride 102 mmol/L. Chest X-ray examination showed pan-cardiomegaly, calcification of the aortic arch, and cephalization. An ECG had shown a sinus rhythm, heart rate 83x/min, left axis deviation with Left Ventricular Hypertrophy (LVH), Left Atrial Enlargement (LAE), Right Atrial Enlargement (RAE), Poor R Wave Progression (PRWP), and inferior infarction.

Based on the history, physical examination, and laboratory investigations, the patient's diagnosis was heart failure AHA stage C with stage 2 hypertension and type-2 diabetes mellitus. The patient was given Ringer Lactate solutions 8 drops per minute intravenously as maintenance fluids, bisoprolol 2.5 mg/24 hours orally, ramipril 10 mg/24 hours orally, aspirin 80 mg/24 hours orally, furosemide 20 mg/2 mL/24 hours IV, and glimepiride 2 mg/24 hours orally. The patient was given the education to bedrest, limit fluid intake per day so that the heart's workload is not heavy, diet low in salt (1.5 grams/day or ± 1 teaspoon per day), and limit excessive sugar consumption. After five days, the physical examination showed that the respiratory rate turned normal. The pitting edema and the ascites were reduced. The patient went home after the laboratory results are stable, was informed to taking medicine regularly and control every one month.

DISCUSSION

According to ACCF/AHA 2013, heart failure (HF) is a complex clinical syndrome based on the heart's structural or functional disorder that results in impaired ventricular filling and pumps blood throughout the body's tissues adequately. Diagnosis of HF is confirmed based on history, physical examination, and laboratory investigations.¹¹ The Framingham criteria for diagnosing heart failure required the concomitant presence of 2 major or 1 major and 2 minor criteria. The major criteria include paroxysmal nocturnal dyspnea or orthopnea, distended neck veins, rales, cardiomegaly, acute pulmonary edema, an S₃ gallop, elevated jugular venous pressure, and a positive hepatojugular reflux. Minor criteria included ankle edema, night cough,



dyspnea on exertion, hepatomegaly, pleural effusion, and tachycardia ≥ 120 beats per minute.^{7,12,13}

This patient had major symptoms in the form of paroxysmal nocturnal dyspnoea, orthopnoea, rales below the fifth intra-costal space on the right and left lungs, and the cardiac margin was widening, indicating cardiomegaly. Orthopnoea unmasks congestion when dependent fluid is redistributed from the abdominal reservoirs and venous circulation when lying recumbent. This loading of the right ventricle will correspondingly increase left-sided and pulmonary venous pressures. Reduced pulmonary compliance and pulmonary oedema subsequently develop, giving rise to the symptoms of breathlessness when lying down.^{12,14} Paroxysmal nocturnal dyspnoea occurs several hours after the patient lies down to sleep, resulting from increased pressure in the bronchial arteries. The bronchial resistance increases and the air is difficult to pass. The horizontal position during sleep also tends to increase pulmonary congestion by increasing right ventricular filling pressure and decreasing vital capacity.^{12,14,15} Minor symptoms in this patient were dyspnoea on exertion and ankle oedema. Signs of ankle oedema result from fluid retention and volume overload.^{12,14} Fatigue is triggered by inadequate blood perfusion leading to decreased oxidative capacity because affecting the respiratory and peripheral muscles. These signs and symptoms meet the Framingham criteria for the diagnosis of HF, followed by the presence of risk factors, history of hypertension for five years, and diabetes mellitus in the past five years. The clinical manifestations of chronic heart failure that last a long time will be cardiomegaly and secondary mitral/tricuspid regurgitation.^{12,16}

According to the New York Heart Association criteria for HF, heart failure can be classified into several functional classes according to structural abnormalities of the heart (according to the AHA) or based on symptoms related to functional capacity (according to the NYHA). This classification is used extensively in clinical practice and research to determine a patient's eligibility for appropriate health care. Shortness of breath is the most common symptom of left-sided heart failure. It can occur during activity (NYHA II or III) or, in more severe cases, at rest (NYHA IV). This patient met the criteria for AHA stage C, where there were signs and symptoms. There was evidence of underlying cardiac structure, namely based on

physical examination found cardiomegaly, pansystolic murmur, OMI, and based on echocardiography. Meanwhile, this patient's NYHA functional capacity classification includes Class III, where the patient can still perform light activities and has no complaints at rest.^{3,17}

The patient had a history of type II diabetes mellitus. Insulin resistance is a significant risk factor in the development of HF. The presence of diabetes mellitus significantly increases the development of HF in patients without structural heart disease and has a negative influence on the outcome of HF patients. There is evidence indicating that diabetes is a major risk factor for heart failure with ischemic disease. A common abnormality is left ventricular (LV) diastolic dysfunction, possibly due to enlargement of the LV myocardium and vascular rigidity.^{10,18,19}

High levels of glucose in the blood result in the glycosylation of proteins in the basement membrane of blood vessels. As a result, highly reactive AGEs (Advanced Glycation End Products) are deposited and cause inflammation. Then it can cause damage to the walls of blood vessels. Protein deposition will increase, and the walls of blood vessels will continue to thicken (forming hyaline atherosclerosis). As a result, the lumen of the blood vessels narrows and allows ischemia, leading to damage to related organs.^{20,21}

Severe or repeated blockage of heart tissue from a myocardial infarction can seriously impair the heart's ability to circulate blood. Its main consequence is congestive heart failure, a condition characterized by abnormalities of myocardial function and neuro-hormonal regulation, resulting in fatigue, fluid retention, and reduced longevity. Vascular stiffness arising from diabetes mellitus also has a major influence on the development of heart failure. Pulse wave velocity is known as an indicator of target organ damage in patients with diabetes. Higher peripheral pulse pressure is associated with a greater risk of cerebrovascular disease (CVD) in the general population and high-risk patients with LV dysfunction.^{18,19,22}

An increase in blood pressure accompanied by a cardiovascular risk exacerbates each other. Hypertension was identified as a significant precursor to left ventricular hypertrophy (LVH). The principle of structural adaptation of the heart to increased pressure loads is LVH, which essentially causes an increase in the thickening of the walls of



the cardiac chambers. The thicker the heart muscle, the smaller the heart cavity. Inadequate relaxation and the small size of the heart cavity will reduce ventricular filling, leading to decreased cardiac output. Ventricular relaxation occurs during diastole so that when a hypertrophic heart cannot relax, cardiac hypertrophy causes diastolic dysfunction. If the heart cannot perform optimal relaxation, then the forward blood flow will be reduced, and there will be a reversal of the blood flow (backup of flow). Diastolic dysfunction due to hypertension can show signs and symptoms of heart failure, although the ejection fraction is still within normal limits. The development of LVH is associated with progressive degenerative changes in hypertrophied cardiac myocytes and abnormal accumulation of collagen in the interstitial spaces.²³⁻²⁵

Management of HF patients is divided into non-pharmacological and pharmacological therapy. Dietary sodium restriction is generally recommended in patients with HF and is supported by various guidelines. The AHA recommends sodium restriction to 1500 mg/day seems appropriate in most patients with stage C and D HF.^{11,26}

ACE inhibitors function to suppress neurohormonal activation and in heart failure caused by left ventricular systolic dysfunction. ACE inhibitors can reduce the risk of death and reduce the hospitalization of HF patients. The benefits of ACE inhibitors have been seen in patients with mild, moderate, to severe HF and in patients with coronary artery disease. Treatment with an ACE inhibitor should be started with a low initial dose, followed by a gradual increase in the dose to the optimal dose.^{27,28}

The use of beta-blockers (e.g., bisoprolol) has been shown to reduce mortality and recommended in patients with HF. Long-term therapy with beta-blockers reduces HF symptoms and improves hemodynamic parameters. Like ACE inhibitors, beta-blockers reduce the risk of death and hospitalization. The benefit of beta-blockers was seen in patients with or without DM. The ESC and AHA guidelines recommend concurrent use of ace inhibitors with beta-blockers for all patients with systolic HF with reduced EF to prevent symptomatic HF, improve left ventricular remodeling, and reduce the risk of hospitalization and premature death.^{11,29,30}

Diuretics inhibit sodium reabsorption. Loop

diuretics are the most preferred diuretic agents for use in most HF patients. Controlled trials demonstrated the ability of diuretics to increase sodium excretion and decrease signs of fluid retention in patients with HF. Diuretics are the only drugs used in the treatment of HF that can adequately control fluid retention in HF. Aldosterone receptor antagonists (e.g., spironolactone) are also recommended in patients with NYHA class II-IV HF and who have a left ventricular ejection fraction. LVEF 30%).³¹⁻³³

Many HF patients with diabetes mellitus require insulin either as monotherapy or in combination with other glycaemic agents to achieve adequate blood glucose control. According to Perkeni, the primary therapy for diabetes mellitus is still starting from modifying a healthy lifestyle, losing weight, adjusting diet (according to ideal body weight), and physical activity at least 3-5x/week for 30-45 minutes. For type 2 diabetes, as long as HbA1c is still <7%, healthy lifestyle is the primary therapy, followed by oral antidiabetic drugs monotherapy if necessary (HbA1c <7.5%). If the HbA1c has not reached 7% or HbA1c > 7.5% within three months, the therapy can be increased to double oral antidiabetic drugs (whose mechanism of action is different). If HbA1c has not reached 7% within three months, then therapy can be increased to triple oral antidiabetic drugs. If HbA1c still has not reached 7% or HbA1c > 9% within three months, insulin can be given.^{21,34} This patient was given glimepiride as oral antidiabetic drug monotherapy since his HbA1c 6.6%.

The development of HF is a common complication in patients with diabetes, and the coexistence of diabetes and HF signifies increased morbidity and mortality. It is essential to understand the balance that exists in pharmacological treatment for both conditions. Current guidelines recommend individualizing hyperglycemic therapy to treat type 2 diabetes based on patient needs, comorbid conditions, and potential side effects of medications. These recommendations may be essential in patients with diabetes and HF. Further efforts are needed to confirm the optimal treatment of glycemia in patients with diabetes and HF.^{18,35}

CONCLUSION

A 54-year-old man came to Dr. Kariadi hospital with shortness of breath, which worsened during light activities and decreased when he was in



a semi-sitting position, had a history of diabetes mellitus and hypertension. The patient met the Framingham criteria. Blood laboratory examination showed HbA1c 6%, chest X-ray and ECG showed an enlarged heart. The patient's diagnosis was NYHA class III heart failure. The patient received antiplatelet, ACE inhibitors, beta-blockers, diuretic, low salt diet, restriction of fluid intake, and oral antidiabetics.

Heart failure is a clinical syndrome that results from any disorder that impairs the ability of the heart to supply the body's blood requirement. Hypertension, diabetes mellitus, and metabolic syndrome are the most common risk factors for this clinical syndrome. The author would like to state that early and accurate diagnosis is crucial so that the treatment may start immediately to prevent severe complications. Therapy for risk factors can slow the progression of heart failure. The patients are recommended to take medicines routinely, do exercises regularly, change in diet and lifestyle.

ACKNOWLEDGMENTS

The authors would like to give a special thanks to the Internal Department, Faculty of Medicine, Diponegoro University.

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