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A 45-YEAR-OLD MAN WITH CHRONIC HEART FAILURE: CASE REPORT

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Abstract: Chronic heart failure is a disorder in which the heart muscle weakens and results in heart failure in pumping and supplying enough blood needed by the body. Factors that cause heart failure are very diverse, including smoking habits, diabetes mellitus, hypertension, hyperlipidemia, being overweight and stress. In this case, it was reported that a 45-year-old man came to the emergency room with complaints of shortness of breath since four days ago, aggravated by light activities such as walking to the bathroom, cold sweats, coughing at night, and swelling in both legs. The patient had the same complaint six months ago and was admitted to the hospital. However, the patient did not take the medication. History of hypertension recognized by the patient, but not controlled. Physical examination found Body Mass Index 27.7 (obese), blood pressure 178/104 mmHg, pulse rate 114 times/minute regular, respiratory rate 30 times/minute, Oxygen Saturation 92%. Jugular venous pressure increased 5+4 cm. Percussion of the heart reveals an enlarged heart border, auscultation of the heart reveals S3 Gallop, accompanied by heart murmurs. Lung examination revealed moist crackles in both lungs. Examination of the extremities revealed pitting edema in both legs. Electrocardiography examination found sinus tachycardia with and Couplet Ventricular Extra Systole. Chest X-ray examination showed cardiomegaly and early features of pulmonary edema. Echocardiography results showed global hypokinetic, decreased left ventricular and right ventricular systolic function, left ventricular ejection fraction 30.1%, left atrial and ventricular dilatation, concentric and eccentric left ventricular hypertrophy, moderate mitral regurgitation, moderate tricuspid regurgitation, severe pulmonary hypertension. Patients were given intravenous injections of Furosemide 40 mg every 8 hours, Ramipril tablets 1x10 mg, Digoxin tablets 1x0.25 mg, Nitrokaf Retard tablets 2 x2.5 mg, and Spironolactone tablets 1 x25 mg.

Keywords: chronic heart failure, shortness of breath, pitting oedema, VES couplet

Introduction

Congestive heart failure (CHF) is defined as a complex set of symptoms resulting from disturbances in the work of the heart, both structurally and functionally. The initial cause of congestive heart failure is the presence of disorders in the weakened walls of the heart muscle that affect the heart's failure to pump and adequate blood supply needed by the body.(Lilik & Budiono, 2021)

Data from the World Health Organization (WHO) in 2016 showed that in 2015 there were 23 million or about 54% of deaths due to heart failure or Congestive Heart Failure. (CHF). In Indonesia, data

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from Riskesdas in 2018 showed a prevalence of heart failure of 1.5%. According to the results of the study, the age most affected by heart failure were the elderly in the age group of 60-70 years (50%). Then followed by the 50-59 year-old group (37%), the 40-49 year old group (13%), and the lowest was the 30-39 year age group (3%).(Pangestu & Nusadewiarti, 2020)

Factors that cause heart failure include smoking habits, diabetes, hypertension, cholesterol, overweight, to stress. There are three other factors that cannot be avoided by humans: inheritance and family background, age and gender factors that are found in many cases of heart failure. In addition to hypertension, the causes of heart failure are abnormalities of the heart muscle, atherosclerosis, and inflammation of the myocardium.

Case Report

45-year-old Mr. B came to the Roemani Hospital Emergency Room on June 14, 2023 at 04.50 Indonesian Western Time with a complaint of shortness of breath since 4 days ago. Initially, the patient has already felt a sickness in the last month, but it felt disturbing 4 days ago and felt very shortness of breat in the early morning before going to the hospital. Sickness is felt constantly, heightened when the patient performs mild activities such as walking to the bathroom. Patients often wake up at night because of shortness of breath and cold sweat and must sleep in a sitting position. The pain in the left side of the chest is constantly felt. The pain feels sharp. Patient had a history of being hospitalized at Roemani Hospital in December 2022 with similar complaints but after that he was never controlled and did not take medicine regularly, if the medicine runs out he only buys the medicine himself at the pharmacy.

Other complaints of the patient cough at night (+) cough unhealthy, fever (-), nausea (+) every want to eat, vomiting (-), defaecate within normal limits, urination within normal, left and right leg swelling, and heart feels beating quickly.

The patient's past medical history also complained of the same symptomps in December 2022 until he was hospitalized. The history of hypertension is recognized by the patient, but the patient has never been in control of the doctor and does not take medication regularly, for the average patient's blood pressure they do not know because they rarely measure it. A history of heart disease is also recognized by the patient; however, the patient also rarely goes to the doctor for control and rarely takes medication. Patient denied history of diabetes, asthma, allergies. The patient's drug use history, namely, digoxin, salbutamol and omeprazole. There was no family history of hypertension, heart disease, diabetes, allergies, and asthma.

Personal social and economic history, the patient has been an active smoker for a dozen years, and within a day the patient spent 1-2 packs of cigarettes but has stopped in December 2022. The patient has never consumed alcohol, the patient has also never been working out. At home the patient lives with his wife and sister. The patient uses BPJS to pay the treatment.

Examination of the general condition of the patient appeared numbness, mental consciousness, body mass index 27.7 (obesity 1), blood pressure 178/154 mmHg, Heart Rate 114x / minute regular, Respiratory Rate 30x / min, SpO2 which was initially 92% increased to 98% with NOC 5 lpm. Generalized status of the head, eyes, nose, ears, mouth within the normal limits, on examination of the

neck jugular venous pressure is increased 5+4; thorax cor inspection, palpation within the norm but percussion there abnormalities on the lower right edge of Intercostal Space (ICS) V line midclavicularis dextra, the lower left edge in ICS VI linea axillaris anterior sinistra, his waist is vertical, the upper edge at ICS II linea midclaviccularis esquerra, auscultation of the heart is S3 Gallop, the heart voice 1-2 regular, there is a heart murmur. On pulmonary inspection, palpation, percussion within normal limits, auscultation of the vesicular base sound but there are additional brute wet ronkhi sounds on ICS IV-VI dextra and sinistra. Abdominal inspection, auscultation, percussion, and palpation within normal limits. Examination of the extremities, there is a cold acral in the upper extremities dextra sinistra and the lower extremities of dextre sinistra, edema pitting on the lower ends of dectra esquerra, Capillary Refill Time more less than 2 seconds.

In the supporting examination, the laboratory results showed leukocytes 11880, MCHC 31.6, RDW 15.6, eosinophil 0.7, chloride 107, pH 7.533, PCO2 21.1, PO2 69.6, HCO3 18.1; for the EKG results in Figure 1, it gives the impression of sinus tachycardia with HR 111x/minute and there is a VES Couplet. Impression of the x-photo of the chest in Figure 2, there is cardiomegaly (LV, LA Susp RA) and an initial picture of pulmonary edema. The echocardiography results in Figure 3 show Global Hypokinetic I, decreased systolic FS LV + RV, LA + LV dilatation, concentric + eccentric LVH, Moderate MR, Moderate TR, Severe PH, Mild AR, LVEF 30.1 %

Patients were given Ringer Lactate infusion therapy, intravenous injection of Furosemide 40mg every 8 hours, Ramipril tablet 1x10mg, Digoxin tablet 1x0.25mg, Nitrokaf Retard tablet 2x2.5mg, Spironolactone tablet 1x25mg, Eperison tablet 3x50mg, and Mecobalamine capsul 2x500mcg.

The patient was hospitalized for 4 days until he was finally declared cured and allowed to go home. Patients are asked to come back for control 1 week later and must control every month and diligently take medication according to the dosage recommended by the doctor.

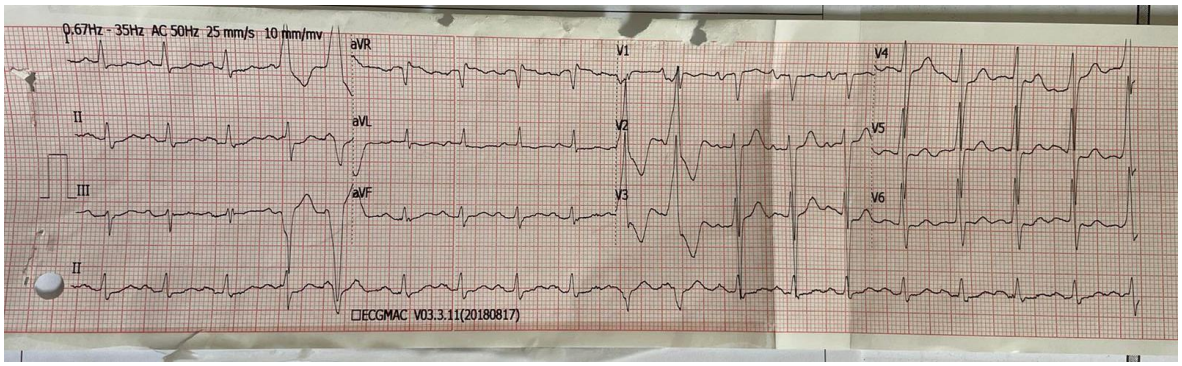

Image 1. Electrocardiogram result

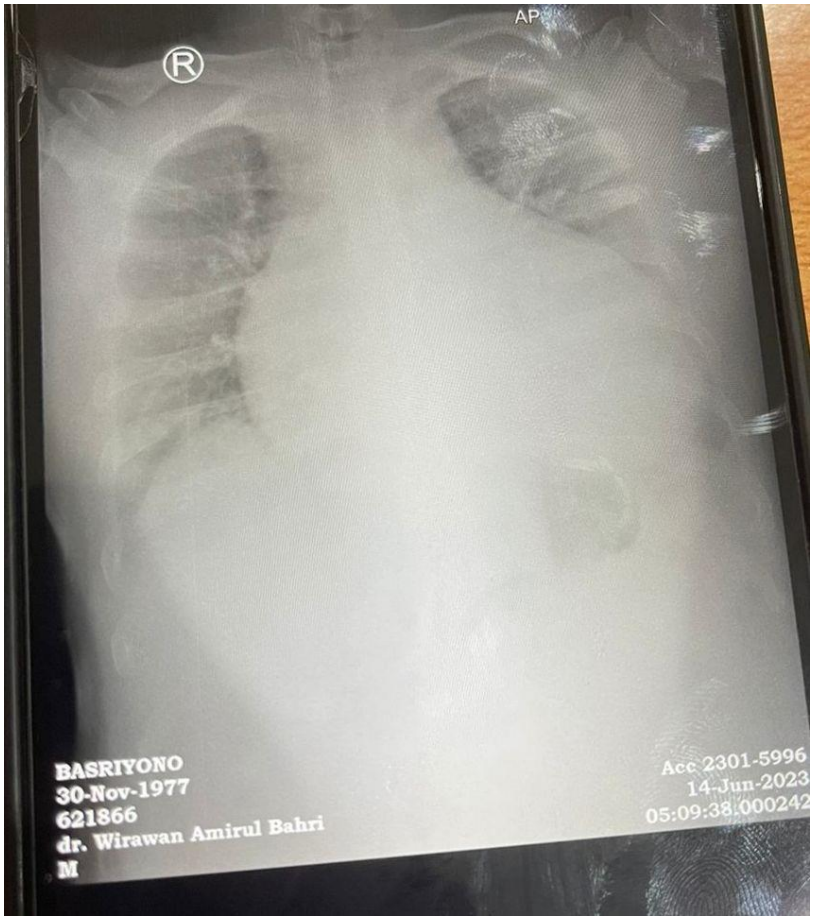

Image 2. Thorax X-Photo

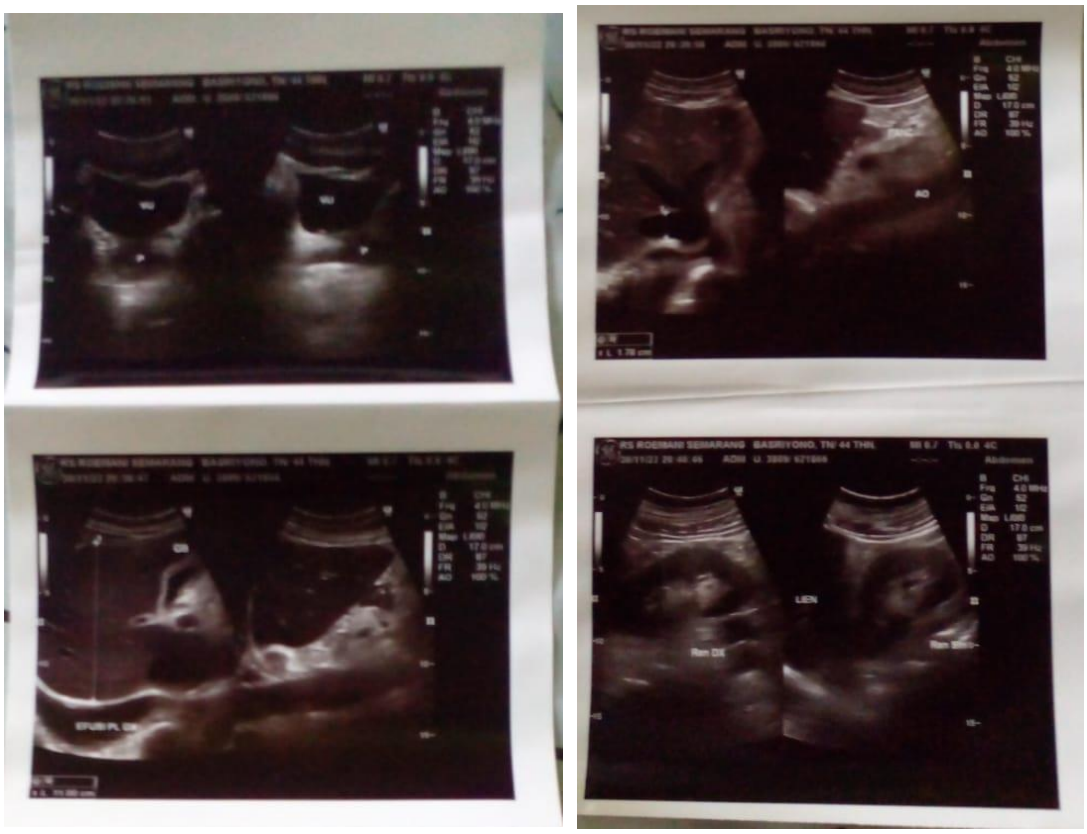

Image 3. Echocardiography result

Discussion

The diagnosis of Congestive Heart Failure in this patient is confirmed by anamnesis, physical examination and supporting examination.

In the anamnesis, the patient complained of shortness of breath since 4 days ago and worsened in the afternoon of the SMRS, left chest pain that felt sharp, often woke up at night due to shortness of breath, cold sweats, coughing at night, felt nauseous every time he ate, swollen right and left legs , the heart beats fast, the patient also experienced the same thing in December 2022 until he was hospitalized, the patient also has a history of high blood pressure and heart disease but is not routinely controlled and does not take medication regularly. The patient also has a smoking history dating back a dozen years and smokde 1-2 packs a day.

At the physical examination found IMT 27.7 into obesity category 1, blood pressure 178/154 mmHg, HR 114x/min, RR 30x / min, SpO2 which was initially 92% increased to 98% with NOC 5 lpm. Thorax cor percussion of the lower right boundary in ICS V linea midclavicularis dextra, the lower left boundaries of ICS VI linea axillaris anterior sinistra, the waistline of the heart is vertical, the upper boundars in the ICS II linea medclaviculularis esquerra, heart auscultation is S3 Gallop. In the auscultation of the pulmons there is a rough wet snoring in the ICS IV-VI dextra and sinistra. On the extremities found the presence of cold acrals in all extremities and edema pitting in the lower extremities dextra sinistra.

On the supporting examination found laboratory results of decreased leukocytes and eosinophils and increased chloride, the analysis of blood gases concluded the presence of uncompensated respiratory alkalosis. Sinus tachycardia ECG with HR 111x/min and VES Couplet. X-Photo Thorax found cardiomegaly (LV,LA Susp RA) and early picture of pulmonary edema. On echocardiography LVEF 30.1%.

The onset of heart failure begins with the presence of damage to the heart or myocardium. This will lead to a decrease in heart rate. When the heart rate is not enough to meet the metabolic needs, then the heart will provide a compensatory response mechanism to maintain the function of the heart so that it can pump the blood adequately. If the mechanism has been maximized and the normal heart rate remains unsatisfied, then after that there are symptoms of heart failure. There are three primary mechanisms that can be seen in the compensatory response, namely increased sympathetic adrenergic activity, increased initial load due to activation of the Renin Angiotensin Aldosterone System (RAAS), and ventricular hypertrophy. A decrease in momentary volume in heart failure will trigger a compensatory sympathetic response. This will stimulate the production of catecholamines from the adrenergic nerves of the heart and the adrenal medulla. The heart rate and contraction strength will increase to increase the heart rate. Furthermore, peripheral arterial vasoconstriction also occurs to stabilize blood pressure and redistribute blood volume to prioritize perfusion to vital organs such as the heart and brain.(Nurkhalis & Adista, 2020)

The activation of the renin system of angiotensin aldosterone will lead to sodium and water retention by the kidneys, increasing ventricular volume and fibrous stretching. This increase in initial load will increase myocardial contractility according to the Frank Starling mechanism. The last compensatory response to heart failure is myocardial hypertrophy or increased thickness of the heart muscle. Hypertrophy will increase the number of sarcomers in myocardial cells. Sarcomers can increase in parallel or serial depending on the type of hemodynamic load that results in heart failure.

Initially, this compensatory response of circulation has a beneficial effect. However, in the end, compensatory mechanisms can trigger symptoms and improve the work of the heart. The result of the above events is an increased load on the myocardium and a continuing prolongation of heart failure.

In this patient there is also high blood pressure that goes into Hypertension stage II, High blood pressure can increase the workload of the heart. When experiencing hypertension, the force of too strong blood flow can injure the walls of the arteries. The pressure can make small cracks that turn into scar tissue. As a result, cholesterol, fat, and other things easily accumulate inside the arteries. Untreated hypertension can make the blood vessels narrow and stiff. Over time, these things make it harder for blood to flow throughout the body and force the heart to work harder than usual. Excessive workloads cause the heart to enlarge, and its function becomes less efficient. If it gets worse, heart function will decline and cause heart failure.(Pratiwi, 2014)

The diagnosis of heart failure can be done with a physical examination and support. There are several types of symptoms found in patients with heart failure as shown in Table 1 below.(PERKI, 2020)

Table 1. Symptoms and Signs of Heart Failure

Symptoms	Signs
Tipikal : 1. shortness of breath 2. Orthopnea 3. paroxysmal nocturnal dyspnea 4. decreased activity tolerance 5. Tired quickly 6. swelling of the lower extremities	Specific : 1. increased jugular venous pressure 2. Hepatojugular reflux 3. S3 heart sound (gallops) 4. The apex of the heart is shifted laterally
Less typical: 1. coughing at night / early morning 2. wheezing 3. Weight gain > 2 kg/week 4. weight loss (advanced heart failure) 5. bloating / bloating, full quickly 6. decreased appetite 7. feeling confused (especially elderly patients) 8. depression 9. pounding 10. passed out	Less typical: 1. Peripheral edema 2. Pulmonary crepitus 3. Deafness in the lung bases on percussion 4. tachycardia 5. Irregular pulse 6. breath fast 7. Hepatomegaly 8. ascites 9. cachexia

Thorax photo examinations often indicate cardiomegaly (cardiotorax ratio (CTR) > 50%), especially if heart failure is already chronic. Cardiomegaly can be caused by dilated left or right ventricles, LVH, or sometimes by pericardial effusion. The degree of cardiomegaly is not related to the function of the left ventricle. Electrocardiography shows several major abnormalities of the patient's well-being (80-90%), including Q wave, ST-T changes, LV hypertrophy, conduction disorders, arrhythmia. Echocardiography should be performed in all patients with suspected clinical heart failure. Dimensions of the heart chamber, ventricular function (systolic diastolic), and abnormalities of wall movement can be assessed, and heart valve disease can be reversed.

According to the American Heart Association (AHA), heart failure is classified according to the symptoms and abnormalities of the heart structure, i.e., Stage A: High risk of cardiovascular failure, but without any anomalies in the structure or symptoms of cardiac failure. Stage B: There are abnormalities in the structure of the heart but no signs or symptoms of heart failure. Stage C: There is an abnormality in the structure of the heart accompanied by previous symptoms of heart failure or is still ongoing. Based on the above classification, this patient is included in the stage C group, which is patients with heart failure with the presence of abnormalities in the structure of the heart accompanied by symptoms of cardiac failure that continue to this day. The aim of implementation in patients with heart failure accompanied by the presence of a decrease in the ejection fraction at stage C is to control symptoms, educate patients, prevent hospitalization and prevent mortality.(Pangestu & Nusadewiarti, 2020)

In this case, medication is given captoril 3 x 25 mg, furosemide 1 x 40 mg, and bisoprolol 1 x 5 mg. In addition, non-medicamentous therapy in the form of education and lifestyle modification should

also be given, which aims to prevent further complications of the patient's disease. The administration of captopril as an ACE inhibitor in these patients was based on recommendations from the American Heart Association (AHA) with level of evidence (LOE) A. Captopril as an ACE inhibitor is useful in inhibiting the Angiotensin Converting Enzyme so that Angio II as a vasoconstrictor cannot be formed. This causes an increase in blood pressure not to occur and the afterload increases. A dose of 3 x 25 mg is given based on the AHA recommendation, i.e., the correct dose used in patients with heart failure is 2-3 x 25mg/day.

Another treatment given to this patient is furosemide 1 x 40 mg. Furosemide is a diuretic loop drug that works on the henla ansa. The administration of diuretics in patients with heart failure is aimed at lowering the preload. The recommended dose of AHA is 0.5-1 mg/kg. Furosemide is a diuretic that can cause side effects of hypokalemia. Given the side effects, it is advisable to give furosemide along with the administration of spironolactone that plays a role in the retention of blood potassium or can be given an external substitution of potash such as KSR tablets.

Another treatment is Bisoprolol. Bisoprolol is a type of selective heart blockers. These drugs are safer than non-selective beta-blockers, such as propanolol. In the treatment of heart failure, this drug has a role in increasing the afterload, so it can reduce the workload of the heart.(Pratiwi, 2014)

Conclusion

The diagnosis of this patient is congestive heart failure/CHF with hypertension grade II where from Framingham criteria there are 4 major criteria and 3 minor criteria in the patient. CHF occurs due to a disruption in the weakened walls of the heart muscle that affects the heart’s failure to pump, and adequate blood supply needed by the body. The risk factors for heart failure in patients are smoking habits as well as uncontrolled hypertension. With good and continued medikamentosa and non-medicamentosa therapy, heart failure and hypertension are expected to be well controlled so that further complications to other target organs can be prevented.

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