Asthma of Cardiac Origin in a 66-Year Old Male

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ABSTRACT

Heart failure is a disease with high morbidity and mortality. Wheezing, suggesting cardiac asthma, is one of the signs of heart failure. Treatment of cardiac asthma is different from bronchial asthma. An accurate history and physical examination may lead to appropriate diagnosis and treatment.

Keywords: Acute heart failure, cardiac asthma, wheezing

ABSTRACT

Gagal jantung adalah penyakit dengan morbiditas dan mortalitas tinggi. Mengi, yang menandakan asma kardial, adalah salah satu tanda gagal jantung. Tatalaksana asma kardial berbeda dari asma bronkial. Anamnesis dan pemeriksaan fisik yang tepat, didukung pemeriksaan penunjang, dapat membimbing klinisi untuk diagnosis dan tatalaksana yang tepat. **Kevin Wibawa. Asma Kardial pada Laki-laki Usia 66 tahun**

Keywords: Asma kardial, gagal jantung akut, mengi

Introduction

Asthma is one of the most prevalent diseases found in the world; it is estimated that around 20 million individuals in the world have asthma.[1] Asthma is usually treated by a general physician in primary care. However, asthma signs do not always come from the respiratory tract.[2]

Cardiac asthma is a condition marked by wheezing, coughing, and orthopnea due to congestive heart failure.[2] This term was first introduced by James Hope in 1832.[2] Based on National Health Interview Survey, National Center for Health Statistics, CDC, the prevalence of cardiac asthma in individuals more than 65 years old is 7.5%.[2]

Wheezing is one of the signs of heart failure (HF).[3] Cardiac asthma with wheezing is one sign that may alert the physician to the possibility of HF.[4] Since the pathophysiology is different, it is important to distinguish between bronchial and cardiac asthma. Generally, the conventional treatment for HF can be used to treat cardiac asthma; loop diuretic, beta blocker, and nitrate can be given to alleviate symptoms.[3] Patient must be encouraged to routinely check for HF and associated comorbidity.[2,4]

Pathophysiology of Cardiac Asthma

Impaired left ventricular (LV) function in heart failure results in symptoms and signs of HF. Elevated LV diastolic pressure increases the pressure in the pulmonary veins.[4] As a consequence, the bronchial veins and pulmonary tissues are congested. The congested bronchial veins increase airway thickness that results in wheezing.[2,7,8]

The human body has several compensatory mechanisms toward HF. At the initial stages, the Frank-Starling mechanism, neurohormonal change, and ventricular remodelling serve as an adaptive mechanism toward decreasing cardiac output (CO). However, the compensatory mechanism transforms into a maladaptive mechanism as HF progress.[7,8] LV remodelling is a response toward increasing preload. The remodelling process serves as a response to reduce wall tension and to preserve contraction. As heart failure worsen, the hypertrophic ventricle pumps lower stroke volume.[7,8] Residual blood in the left ventricle increases end systolic volume (ESV), end diastolic volume (EDP), and left ventricular end diastolic pressure (LVEDP); this increased left ventricle pressure is transmitted into the left atrium (LA), and LA undergoes a structural change to compensate the increasing pressure.[9]

In response to the increasing pressure in LA, the pulmonary vein (PV) compensate by increasing its pressure to prevent pulmonary edema. However, as the pressure in the left ventricle and LA continues to increase, the change in PV cannot prevent the pulmonary edema.[4] The capillary pressure begins to rise, followed by fluid transudation and accumulation, causing pulmonary congestion.[8] The increasing PV pressure can be transmitted to bronchial veins. Bronchial veins become congested and reduce the bronchial diameter. Both the pulmonary and bronchial veins congestion contribute to the development of wheezing.[2,9]

Treatment

The treatment of cardiac asthma is aimed to improve cardiac contractility and to alleviate symptoms.[2,4] Bronchodilator use for cardiac asthma has small evidence and is usually used to determine the coexistence of bronchial asthma and heart disease.[2] Treatment modalities for cardiac asthma

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involve the treatment for acute heart failure (AHF), such as diuretics, nitrates, inotropic agents, angiotensin converting enzyme (ACE) inhibitors, and angiotensin receptor blockers (ARB).[2,3,9]

Diuretics can be given as an initial treatment to reduce fluid congestion. The most common is furosemide, a loop diuretic. Furosemide acts in dense ascending loop of Henle. Furosemide is a powerful diuretic since it can excrete 20-25% sodium. Intravenous preparation is preferred for rapid onset of action in congested patients.[7,10] For naïve-furosemide patients, an initial doses of 20-40 mg IV boluses can be given. Patients with a history of diuretic use may need a higher dose. If the congestion persists, doses of 5-40 mg/h IV infusion may be given.[3,10]

Vasodilators such as nitrates, ACE Inhibitors, and ARBS can be given to reduce the preload and/or afterload. Vasodilators such as nitrates act primarily in veins, while ACE Inhibitors and ARBs act in both veins and arteries.[7] Nitrates in oral forms, such as nitroglycerine and isosorbide dinitrate, can be given initially. Nitrates have a rapid onset of action. Intravenous nitroglycerine (starting at 10-20 µg/min, up to 200 µg/min) or isosorbide dinitrate (starting at 1 mg/h, up to 10 mg/h) can be given according to clinical response and judgement.[3]

Beta-blockers (BBs) are not usually used for the treatment of AHF. The negative inotropic effect of BBs is contraindicated to patients with systolic dysfunction.[3,7] However, as maintenance therapy, many RCTs showed BBs reduce mortality and morbidity in patients with HFrEF. But, the benefit of BBs in AHF is still uncertain. Some AHF patients have BBs as a routine medical treatment; in this subset of patients, it is still uncertain whether to stop or to continue the BBs treatment.[3] A study from Meuwese et al showed BBs must be lowered or stopped temporarily in patients with cardiogenic shock and/or severe decompensation because BBs may worsen the haemodynamic.[11] Another study from Jondeau G, *et al*, showed a continuation of BBs in ADHF patients did not delay or reduce the clinical improvement. Therefore, there is a possibility to continue BBs treatment in some AHF patients.[12]

Table 1. The New York Heart Association (NYHA) classification[6]

Class	Symptoms
I	HF does not cause limitations to physical activity; ordinary physical activity does not cause symptoms
II	HF cause slight limitations to physical activity; the patients are comfortable at rest, but ordinary physical activity results in HF symptoms
III	HF cause marked limitations to physical activity; the patients are comfortable at rest, but less than ordinary physical activity results in HF symptoms
IV	HF patients are unable to carry on any physical activity without HF symptoms or have symptoms when at rest

Table 2. Symptoms of heart failure[3]

Symptoms	
Typical	Atypical
Breathless	Nocturnal cough
Orthopnea	Wheezing
Paroxysmal nocturnal dyspnea	Bloated feeling
Reduced exercise tolerance	Loss of appetite
Fatigue, tiredness, increased time to recover after exercise	Confusion (especially in elderly)
Ankle swelling	Depression

Table 3. Signs of heart failure[3]

Signs	
More specific	Less specific
■ Elevated jugilar venous pressure ■ Hepatojugular reflux ■ Third heart sound (gallop rhythm) ■ Laterally displaced apical impulse	■ Weight gain (>2kg/week) ■ Weight loss (in advanvance HF) ■ Tissue wasting (cachexia) ■ Cardiac murmur ■ Peripheral oedema (ankle, sacral, scrotal) ■ Pulmonary crepitations ■ Reduced air entry and dullness to percussion at lung bases (pleural effusion) ■ Tachycardia ■ Irregular pulse ■ Tachypnoea ■ Cheyne Stokes respiration ■ Hepatomegaly ■ Ascites ■ Cold extremities ■ Oliguria ■ Narrow pulse pressure

Case

A 66-year-old male was admitted to the emergency room (ER) with worsening shortness of breath since yesterday. The patient had experienced shortness of breath for 14 days; the symptom worsened and became troublesome for 1 day. The shortness of breath was felt during mild physical activity and resolved during rest. He had experienced the same symptom when heart disease was first diagnosed by a cardiologist. However, the patient did not remember the type of heart disease. The patient also complained of frequent urination at night during the last 1 month.

The patient had a history of hypertension, diabetes mellitus, and heart disease. He denied any history of bronchial asthma. The patient stopped smoking 20 years ago. He did not engage in routine physical activity. His routine medications are glimepiride 3 mg o.d, bisoprolol 5 mg o.d, isosorbide dinitrate 5 mg b.i.d, digoxin 0.25 mg o.d, ivabradine 5 mg b.i.d, and antacid 400 mg t.i.d. Patient compliance was poor even though he did not experience any side effects. The routinely consumed drugs were isosorbide dinitrate and glimepiride. Bisoprolol is consumed when he felt a headache. The rest of the medications was never consumed. His last visit to a cardiologist was 4 months ago. COVID-19 pandemic and insurance problem made him reluctant to come to the hospital.

At initial presentation, he was obese with body weight 78 kg, body height 169 cm, body mass index of 27.31 kg/m^2, and appeared dyspneic. His vital signs were: fully awake, blood pressure 160/90 mmHg, heart rate 100 bpm, respiratory rate 27 x/min; body temperature 36.3 °C, and oxygen saturation 90% at room air. Other findings included slightly increased jugular vein pressure, normal cardiac sounds, no cardiac murmur, cardiomegaly, wheezing all over the lung field, warm extremities, and

capillary refill time <2 seconds. ECG showed sinus rhythm and incomplete left bundle branch block. The patient declined any further laboratory and imaging tests.

The patient brought his routine medications and denied any intravenous treatment. Oxygen 2 lpm, isosorbide dinitrate 5 mg, and bisoprolol 5 mg were administered. The haemodynamic were observed for 30 minutes. His shortness of breath was improved and the wheezing was resolved. The patient and his caregiver was educated before discharged from ER to consume his daily medications, to visit a cardiologist as soon as possible, and to measure and to record patient body weight and abdominal circumference everyday

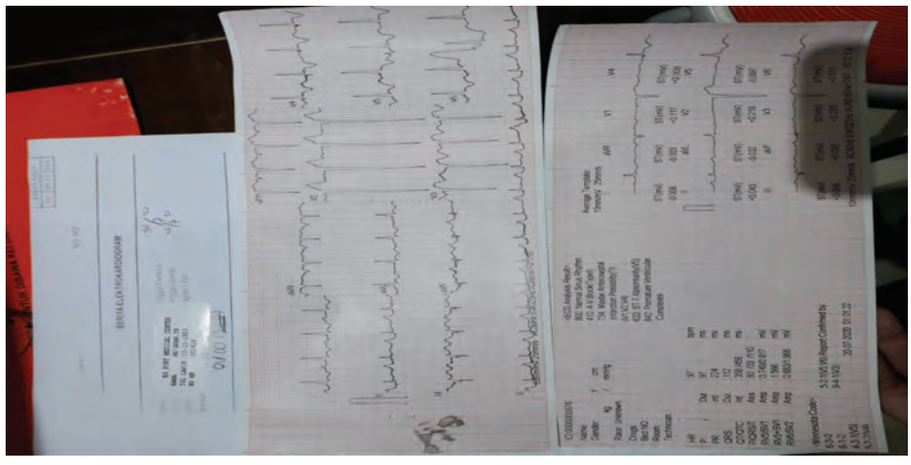

Figure. Electrocardiograph at admission

Discussion

The patient was diagnosed with acute heart failure and the acute precipitating factor is his uncontrolled hypertension and possibly diabetes mellitus complicated with poor compliance.[3] A positive history of heart disease and no history of bronchial asthma can help differentiate cardiac asthma from bronchial asthma. Based on NYHA classification, the patient was at NYHA III.[6] Wheezing all over the lung fields suggests the presence of pulmonary and bronchial vein congestion.[8] Cardiomegaly in this patient increases the likelihood of cardiac asthma compared with bronchial asthma.

It is very important to differentiate between cardiac asthma and bronchial asthma. β-agonist treatment in bronchial asthma, for example salbutamol, may have detrimental effects to the heart since β-agonist increases heart rate and may induce hypertension or arrhythmia.[2] Wheezing in cardiac asthma improves with the administration of diuretic and/or nitrate.

According to Forrester Classification,[3] the patient is categorized as warm and wet. Therefore, therapy at ER is aimed to reduce and relieve the congestion.[3] Vasodilators isosorbide dinitrate was administered. Nitrates have a rapid onset of action to dilate and increase the vein's capacity to accommodate more blood and reduce the returning blood to the left ventricle; nitrates reduce the left ventricular diastolic pressure, reduce lung capillary hydrostatic pressure, and reduce the pulmonary and bronchial vein congestion.[7] Beta blockers (BBs) with β1 selectivity have negative chronotropic and inotropic effects by blocking the adrenergic activity, as shown in bisoprolol.[7] In addition, bisoprolol also decrease renin release from juxtaglomerular cells because juxtaglomerular cells also have β1 receptor.[13] By lowering the heart rate, BBs improve LVEF and symptoms of heart failure (HF). Bisoprolol at 5 mg dose was administered because the patient was not in cardiogenic shock or severe decompensation and did not have any side effects.[11]

Besides alleviating symptoms, the acute precipitating factors and the etiology of HF must be treated, and comorbidities must be controlled.[3] In this patient, poor compliance triggers the symptoms; however, the etiology of HF is still unknown. Hypertension may be the etiology since it increases the afterload and precipitates ventricular remodelling in the long-term.[7] Diabetes mellitus can cause structural and functional abnormalities that result in cardiac dysfunction and ultimately heart failure.[14]

The patient and the caregiver must be educated on the possibilities of side effects from medications. BBs can cause dizziness or lightheadedness and may need dose adjustments.[10]

There are several limitations in this case report. The patient and the caregiver did not bring the result of previous laboratory tests and imaging tests. An imaging test using transthoracal echocardiography is essential for patients with heart failure. LVEF measurement aids in determining the appropriate and proper long-term treatment. Maintenance therapy with diuretics, ACE inhibitors or ARBs, and BBs reduce mortality and morbidity in HFrEF patients. However, the benefits of the aforementioned therapy in HFpEF are lacking.

Besides ECG, no additional laboratory and imaging tests were performed at ER; the diagnosis of acute heart failure is based on history taking and physical examination; other subclinical comorbidities, such as mild anemia, thyroid disease, and valve disease, remain unknown.

Conclusion

Distinguishing bronchial and cardiac asthma is important since the therapy is different. Careful and thorough history taking and physical examination may distinguish cardiac asthma from bronchial asthma. Initial therapy can reduce congestion. However, long term therapy must be started to reduce morbidity and mortality. Patient and caregiver must be educated to improve compliance.

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