CASE REPORT

Case Report : Prolonged Dyspneu In Patients With Mixed Type Pulmonary Hypertension

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ABSTRACT

Pulmonary Hypertension (PH) is a heterogeneous condition defined by a mean pulmonary artery pressure (mPAP) >20 mmHg at rest, confirmed via right heart catheterization. PH can be classified into several groups based on underlying mechanisms, and mixed type PH involves overlapping features from different categories. We report a case of a 49-year-old woman with a history of HIV infection and hyperthyroidism who presented with progressive shortness of breath. Transthoracic echocardiography suggested severe pulmonary hypertension, while further evaluation revealed a combination of pre-capillary and post-capillary components, consistent with mixed type PH. Contributing factors included HIV-associated pulmonary vasculopathy and hyperthyroidism-induced high-output cardiac failure. The patient was treated with intravenous Furosemide, Beraprost sodium, Spironolactone, Digoxin, Warfarin, and Omeprazole. During hospitalization, she developed distributive shock requiring norepinephrine infusion. This case highlights the importance of thorough diagnostic evaluation to identify multifactorial causes of PH, especially in patients with coexisting HIV infection and hyperthyroidism. Management strategies should be tailored to address the complex interplay of underlying conditions while considering drug interactions and local therapeutic resources.
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Keywords: Pulmonary Hypertension, HIV, Hyperthyroidism, Mixed-type pulmonary hypertension, High-output heart failure

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INTRODUCTION

Pulmonary hypertension (PH) is a heterogeneous disorder that can involve a variety of clinical conditions and complications in the cardiovascular and respiratory systems. PH arises as a consequence of many multiple etiologies, including external factors, internal factors and interactive factors. Epidemiologically, pulmonary hypertension is a fairly rare disease with an incidence rate of 1% of the total number of heart disease sufferers worldwide or 20-70 million people worldwide. However, the incidence of PH is still challenging with high morbidity and mortality rates. Non-specific complaints and the progressive development of PH will result in an increase in pulmonary vascular resistance which will then result in failure in lung and heart function. (1)

HIV is considered to be able to cause PH through the role of proteins and inflammatory mediators that HIV continuously triggers. Hyperthyroidism is a pathological condition characterized by excessive synthesis and secretion of thyroid. (2,3)

This case report was created with the aim of showing methods for diagnosing and providing treatment for patients with mixed type pulmonary hypertension in Maluku as a peripheral area which has various limitations from a medical perspective.

CASE REPORT

A 49-year-old woman was taken by her family to the emergency unit with complaints of shortness of breath since ± 1 week ago. Complaints accompanied by difficulty swallowing, chest discomfort, stomach feeling bloated, weakness and coughing. Blood pressure 110/80 mmHg, pulse 67 bpm, normal breathing rate, afebris, and malnutrition. Exophtalmus, jugular vein distension, fine wet rhonchi on both pulmonary surfaces, increased second heart sound.

Laboratory examination revealed abnormalities in the form of hypoalbuminemia (2.8 mg/dl), increased SGOT (36 ul/l), Free Triiodothyronine (T3) 6 pg/ml, Free Thyroxine (T4) 3.2 ng/dl, TSH 0.3 mIU/l, and HIV 3 methods (+). On plain chest x-ray examination, abnormalities were found in the form of cardiomegaly, bilateral pulmonary edema, and aortic dilatation. The EKG showed atrial fibrillation. Meanwhile, echocardiography showed pulmonary hypertension and heart valve as shown in Figure 1.

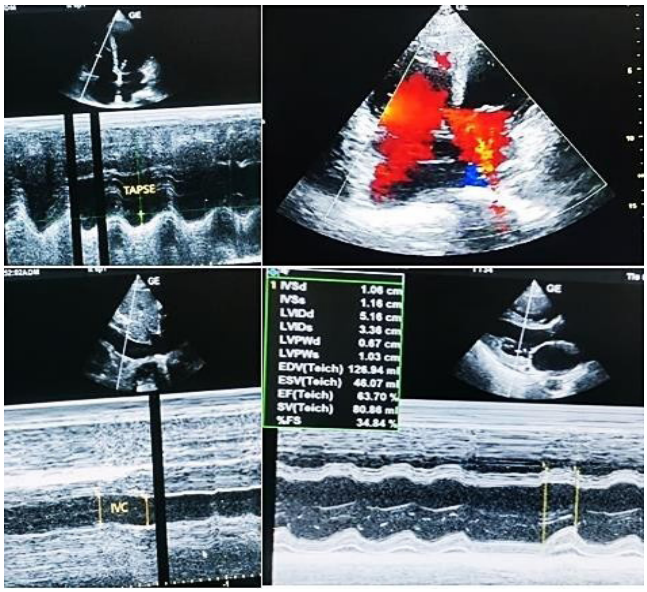

Figure 1: Results of the patient's transthoracic echocardiography as a diagnostic modality

The diagnosis of pulmonary hypertension was established based on findings from physical examination and supporting investigations. The patient was subsequently treated with pharmacological therapy, including intravenous Furosemide, Beraprost sodium, Spironolactone, Digoxin, Warfarin, and Omeprazole. After several days of hospitalization, the patient was discharged in a stable condition and free of shortness of breath.

Pulmonary hypertension is a condition that requires special attention and is still a global health problem. For this reason, a systematic understanding in diagnosing and providing comprehensive treatment for PH is deemed necessary.

DISCUSSION

Pulmonary hypertension is a disease with a poor prognosis that is generally caused by a number of disorders, including left heart, lung, and chronic thrombo-embolic disease. Based on etiology, PH can be grouped into several groups, namely pulmonary arterial hypertension (PAH), PH due to left heart failure, PH due to chronic lung disease, chronic thromboembolic PH (CTEPH), and PH due to other or multifactorial diseases. Initially, patients with PH generally present with main symptoms related to right ventricular dysfunction such as shortness of breath during activity, shortness of breath when bending (bendopnea), palpitations, hemoptysis, abdominal distension and pain, leg edema, and syncope. However, at a more advanced level symptoms can manifest in the form of chest pain, hoarseness (dysphonia), and other symptoms that can arise as the underlying etiology of PH. (1,2)

Physical findings can reliably reflect the severity of pulmonary hypertension and right heart failure. There are several findings that are considered significant in diagnosing PH. This includes monitoring vital signs, such as blood pressure which is often low but still tolerable, as well as an increase in resting heart rate that has no obvious cause. This can be a sensitive indicator of decompensated right ventricular (RV) failure. Oxygen saturation measured via pulse oximetry in PH patients can vary from normal to low. (1)

On physical examination of the abdomen, manifestations of right heart failure can be seen through signs such as hepatomegaly, ascites, and edema in the lower extremities. These three signs can occur due to significant tricuspid valve regurgitation. The presence of clubbing fingers, although not a common finding on physical examination, is considered not specific for establishing a diagnosis of PH. ECG examination of PH patients will show right ventricular hypertrophy, right atrial dilatation, and axis deviation to the right. Meanwhile, chest x-rays show abnormal results in 90% of PH cases. (2)

Based on the ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension 2022 , the probability of PH can be assessed using a combination of parameters such as tricuspid regurgitant velocity (TR Vmax), right ventricle size, interventricular septum function, fluctuation of the inferior vena cava (IVC) on the respiratory cycle, right atrial systolic area, flow velocity patterns systolic to early diastolic pulmonary regurgitant velocity (PVAcct), as well as the diameter of the Pulmonary Artery as seen in Tables I and Table II. (2)

Table I: Probability of PH based on echocardiography of symptomatic patients (2)

TR Vmax (m/s)	PH sign (minimum 2)	PH probability
≤ 2.8 / not measurable	There isn't any	Low
≤ 2.8 / not measurable	There is	Currently
2.9 – 3.4	There isn't any	
2.9 -3.4	There is	Tall
>3.4	There isn't any	

Table II: Echocardiographic signs suspicious for PH (2)

A: Ventricles	B: Pulmonary artery	C: IVC/right atrium
The ratio of the basal diameter of the right ventricle to the basal diameter of the ventricle is >1	PV/Acct <105 ms/ midsystolicnotching	IVC >21 mm with collapsibility <50% on deep inspiration /<20%on normal inspiration
Flattening of the interventricular septum (left ventricularеccentricity index >1.1 during systole and/or diastole	Early diastolic pulmonaryregurgitation velocity >2.2 m/s	Right atrial area (end systole) >18m ^{2}
	Pulmonary artery-diameter >25mm	

The images found on the patient's echocardiography examination are in accordance with the diagnostic criteria based on the table above, namely a pulmonary

artery diameter >25 mm, a right atrial area of 46.07 mm^2, and a basal right ventricle to left ventricle diameter ratio >1. Once the diagnosis of PH is confirmed, the severity of the disease needs to be evaluated to assess the risks and determine an appropriate treatment plan. Important evaluation instruments in assessing the severity of PH patients involve assessment of functional class, exercise ability, hemodynamic parameters in the lungs, acute vasoreactivity response, right ventricular performance, BNP (brain natriuretic peptide) concentration, endothelin-1 levels, uric acid, and troponin.(1,2) Non pharmacological treatment given to PAH patients is to reduce physical activity, reduce fluid intake, and diet. The pharmacological treatments available to treat PH are highly dependent on three PH pharmacodynamic pathways, namely the prostacyclin pathway, nitric oxide pathway, and endothelin pathway inhibitors. Although until now there is no drug specifically intended for the treatment of PH, the basic principles of PH management are to increase endurance, improve physical activity, and improve the patient's quality of life. Some of the drug classes used are anticoagulants, diuretics, digoxin, specific pulmonary vasodilator therapy, Calcium channel blockers (CCB), Endhothelin receptor antagonists (ERA), Phosphodiesterase type 5 inhibitors (PDE-5i), guanylate cyclase stimulators (sGC), Prostacyclin analogues and prostacyclin receptor agonists, as well as Nitric oxide (NO).(1,2) In this case, The patient was treated with intravenous Furosemide 20 mg every 8 hours, Beraprost sodium 20 µg three times daily, Spironolactone 25 mg once daily, Digoxin 0.25 mg once daily, Warfarin 2 mg once daily, and intravenous Omeprazole 20 mg every 12 hours. During hospitalization, she developed distributive shock requiring norepinephrine infusion.

Apart from this treatment, patients are also given symptomatic drugs to relieve symptoms and treat hyperthyroidism and HIV as causes of PH. Clinical treatment of this condition through the use of drugs that inhibit thyroid gland function, therapy with iodine radiation, or through surgery, has been proven effective in reducing pressure in the lung vessels based on the findings of several case reports.(3,4)

While treatment for PHpatients in HIV-infected individuals is generally similar to those who are not infected, the potential for drug interactions hinders the administration of sildenafil because its metabolite products produced through the metabolism of cytochrome P450 isoform 3A4 can interact with protease inhibitors. Given that HIV- infected individuals often have factors independently associated with PH, such as lung problems or drug use, finding a clear distinction between the impact of HIV infection and other risk factors is difficult. Therefore, treatment of identified secondary causes must be carried out decisively. Some studies suggest that HIV-infected individuals on antiretroviral therapy (ART) may experience improvements in function or hemodynamic parameters visible in Doppler echocardiography, but other studies imply that HAART has no significant effect on hemodynamic parameters. However, definitive conclusions are difficult to draw considering that information regarding the disease may not have been well documented before or after ART administration.(3-5)

CONCLUSION

Pulmonary hypertension is a multifactorial disease; therefore, history taking, physical examination, and supporting investigations must be performed carefully and systematically, as these factors are crucial in determining patient management and prognosis. HIV and hyperthyroidism are among the most common etiologies of pulmonary hypertension in developing regions. The use of prostacyclin analogs such as Beraprost, combined with Diuretics, Warfarin, and Digoxin that are available in peripheral areas, is considered to provide significant clinical improvement in PH patients with comorbid HIV and Hyperthyroidism.

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