

Massive pleural effusion due to heart failure in an undetected diabetic hypertensive patient: A case report

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Case Report

ABSTRACT

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Pleural effusion is pathological accumulation of pleural fluid. The most common causes of pleural effusion are cancer, pneumonia, pulmonary embolism and heart failure. Diabetes mellitus and hypertension are two risk factors of heart failure. A 74-year-old male patient came to the emergency room feeling fatigued all over the body since seven days before, dyspnea and notable reduced appetite and weight loss. Initial vital examination revealed an increase in heart rate and blood pressure (BP=140/80 mmHg, 120 bpm). Oxygen saturation was normal and deteriorate from time to time (92.5%). Chest physical examination showed pleural effusion on the right lungs. Laboratory findings noted an elevated blood glucose and mild hypokalemia in patients. ECG result showed old myocardial infarct (OMI) anteroseptal and infrequent premature ventricular contraction (PVC). Chest X-Ray revealed a massive pleural effusion on the right lungs, a mediastinal shift and cardiomegaly. The patient were given IVFD RL, gliquidone, Lantus Bisoprolol, B-Complex, and also thoracentesis for the pleural effusion. The underlying pathology largely determines the clinical symptoms of pleural effusion. In this case, the most probable underlying cause is heart failure. The risk factors of heart failure, in this case, are undetected diabetes melitus, hypertension, OMI and arrhythmia. Increasing prevention and early detection of chronic illness such as diabetes melitus and hypertension is a very necessary step.

Efusi pleura merupakan akumulasi patologis cairan pleura. Penyebab paling umum dari efusi pleura adalah kanker, pneumonia, emboli paru dangagal jantung. Diabetes melitus dan hipertensi merupakan dua faktor risiko gagal jantung. Seorang pasien laki-laki 74 tahun datang ke ruang gawat darurat dengan perasaan lelah di seluruh tubuh sejak tujuh hari sebelumnya, terdapat dyspneu, nafsu makan berkurang dan penurunan berat badan. Pemeriksaan vital menunjukkan peningkatan denyut jantung dan tekanan darah (140/80 mmHg, 120 bpm). Saturasi oksigen normal dan menurun dari waktu ke waktu (92.5%). Pemeriksaan fisik dada menunjukkan efusi pleura pada paru kanan. Temuan laboratorium mencatat peningkatan glukosa darah dan hipokalemia ringan pada pasien. Hasil EKG menunjukkan OMI anteroseptal dan PVC Infrequent. Rontgen dada mengungkapkan efusi pleura masif pada paru kanan, pergeseran mediastinum, dan kardiomegali. pasien mendapatkan terapi IVFD RL, gliquidone, Lantus, Bisoprolol, B-Complex dan juga thorakosentesis untuk kondisi efusi pleura. Patologi yang mendasari sangat menentukan gejala klinis efusi pleura. Dalam hal ini, penyebab mendasar yang paling mungkin adalah gagal jantung. Faktor risiko gagal jantung dalam hal ini adalah diabetes melitus yang tidak terdeteksi, hipertensi, OMI dan aritmia.

Peningkatan pencegahan dan deteksi dini penyakit kronis seperti diabetes melitus dan hipertensi merupakan langkah yang sangat diperlukan.

INTRODUCTION

Pleural effusion is pathological accumulation of pleural fluid.^{1,6} Pleural effusion can be diagnosed using a simple chest radiograph.^{1,2,7} If the chest radiograph showed more than two-thirds of the hemithorax, the condition is called massive pleural effusion.⁷ The most common causes of pleural effusion are cancer, pneumonia, and pulmonary embolism and heart failure.^{1,6,8,9}

Heart failure (HF) is a complicated clinical condition induced by irregularities in the anatomy and physiology of the heart, affecting the capacity to supply blood according to the demands of the body.¹⁰ Heart failure contributes to 21% of massive pleural effusion cases.⁹ Diabetes mellitus and hypertension are two risk factors of heart failure.¹¹ The increasing number of both diseases also increases the prevalence of heart failure incidences.^{2,11} Unfortunately, most patients with it are likely late-diagnosed or even undetected until the complication emerges.²

CASE DESCRIPTION

A 74-Year-Old male patient came to the emergency room feeling fatigued all over the body since seven days before. The fatigue had

progressively worsened with no associated, aggravating, or relieving factors noted. The patient also complains of palpitations and dyspnea. There no symptoms such as pleuritic chest pain. He also noted a reduced in appetite and notable weight loss. He never felt the same symptoms before. He has never been diagnosed with other diseases.

Initial vital examination revealed an increase in heart rate (HR = 120 bpm) and blood pressure (BP = 140/80 mmHg). Respiratory rate and temperature were within the normal range. Oxygen saturation was normal and deteriorate from time to time (92.5 %). Chest physical examination showed the signs of volume gain at the right lung, reduced tactile vocal fremitus, dullness on percussion, shifting dullness, and diminished or absent breath sounds. No other clinical signs at the head-to-toe physical examination.

Complete blood counts showed everything within the normal range. Blood glucose level test indicated the presence of elevated blood sugar levels (286 mg/dL). There was mild hypokalemia on patient (K⁺ = 3.4 mmol/L). Electrocardiograph examination (Figure 1) showed abnormalities, which an old myocardial infarct (OMI) and infrequent premature ventricular contraction (PVC) anteroseptal.

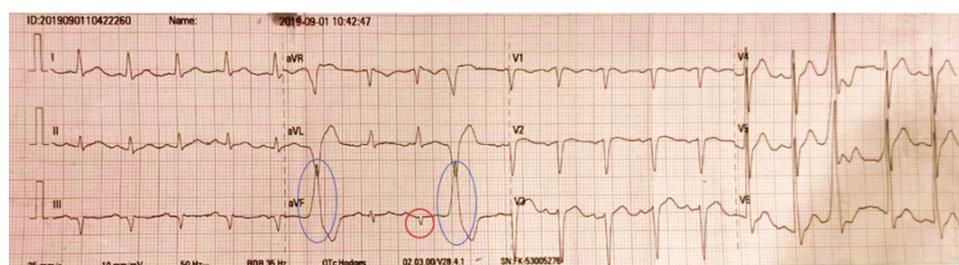


Figure 1. Electrocardiograph Result (ECG); Infrequent PVC (blue) and OMI (red) was found

Chest X-Ray examination (Figure 2) revealed a massive pleural effusion on the right lungs, mediastinal shift, and cardiomegaly. These findings corroborate up the physical examination of the chest, which suggests a pleural effusion. The amount of fluid to be

evident on anteroposterior film is more than 200 mL. Classically, a homogenous opacity is seen with obliteration of the costophrenic angle and a curved upper border, ie, the Ellis S-shaped curve. The interpretation of this showed right massive pleural effusion. Thoracocentesis was

done as a diagnostic assessment and therapeutic management for the massive pleural effusion. Intravenous fluid, ringer lactate, installed on

the patient. The patient was decided to require hospitalization for further observation.



Figure 2. Chest X-ray of the patient. The X-ray shows a massive pleural effusion on the right lungs, mediastinal shift and cardiomegaly.

Anti-hypertensive drugs, beta-blocker was given in the morning (Bisoprolol). Control sugar blood level also being done, because the patient has persistent high sugar blood level without anti-diabetic medication. Gliquidone (30 mg) was administered to the patient twice a day. On the third day, short-acting insulin was also be given to the patient to manage the hyperglycemia.

DISCUSSION

In a healthy adult, the pleural cavity contains low-level fluid, which serves as a lubricant for both of the pleural walls.³ This small amount fluid is produced by the vasculature of the parietal pleural surfaces and absorbed into lymphatic vessels. If one of these circulations becomes interrupted, fluid will accumulate in the pleural cavity, causing pleural effusion.^{2,4,6}

The underlying pathology largely determines the clinical symptoms of pleural effusion. In this case, the most probable underlying cause is heart failure.⁶ Heart failure can generate pleural effusion because in the certain condition there might be extremely high peripheral and pulmonary vasculature pressures and will lead to unnecessary transudation of fluid toward the

pleural cavity and causing pleural effusion.³ The diagnosis of heart failure, in this case, was made based on Framingham Criteria. It is a diagnostic tool that has a fairly high sensitivity value for assessing heart failure. This criterion consists of major and minor criteria. The diagnosis of heart failure can be made if there are at least two major criteria (or one major accompanied by two minor criteria).^{12,13}

According to the Framingham criteria, this patient has a decompensated heart since he has two major criteria (cardiomegaly and rales) and three minor criteria (dyspnea, pleural effusion, and tachycardia). His diagnosis is also supported by other symptoms which might emerge in a patient with heart failure such as generalized fatigue and noted weight loss. Moreover, Electrocardiograph examination result of the patient showed the presence of OMI and PVC which can be a risk factor for heart failure.^{12,13}

Other risk factors of heart failure, in this case, is undetected chronic disease such as diabetes melitus and hypertension.^{8,13} Diabetes and heart failure are inextricably linked; people with diabetes are more likely to develop heart failure and vice versa.^{8,13,14} People with diabetes

melitus are prone to develop heart failure because of impairment of the heart managing glucose and free fatty acids and also because the impact of metabolic derangements on the cardiovascular system. The two reasons above lead to anatomical and physiological changes of the heart and cause cardiac dysfunction.¹⁴

In addition to diabetes, this patient also has undetected stage 2 hypertension. Hypertension is a substantial risk factor for many cardiovascular diseases, including heart failure. Chronic hypertension develops anatomical and functional alterations in the heart, eventually leading to HF, which increases mortality and morbidity.^{15,16} The left ventricle undergoes hypertrophic structural remodeling in response to higher pressure demands. The myocardium becomes increasingly fibrotic in response to the neurohormonal stimulation that occurs with persistent hypertension. Both structural alterations result in a functional decrease in ventricular compliance and lead to decompensated heart.^{15–17}

CONCLUSION

Diabetes mellitus and hypertension are two chronic diseases that have an increasing incidence every year. Both diseases can also increase the incidence or worsen other diseases. As in the patient in this case, the presence of diabetes mellitus and undetected hypertension led to heart failure with a persistent pleural effusion presentation. For this reason, increasing prevention and early detection of these two diseases is a very necessary step.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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