Post Covid with Pleural Effusion: A Case Report

Ana Fauziyati[1](✉), Bagus Andi Pramono[2], and Untung Widodo[3]

[1] Department of Internal Medicine Faculty of Medicine, Universitas Islam Indonesia/Universitas Islam Indonesia Hospital, Yogyakarta, Indonesia
047110434@uii.ac.id

[2] Department of Cardiology, Universitas Islam Indonesia Hospital/Panembahan Senopati Hospital, Yogyakarta, Indonesia

[3] Department of Anaesthesiology and Intensive Care, Faculty of Medicine, Universitas Islam Indonesia/Universitas Islam Indonesia Hospital/Sardjito General Hospital, Yogyakarta, Indonesia

Abstract. The Covid-19 pandemic has lasted for 1,5 years. There are many patients who suffered from many conditions or symptoms post Covid-19. In this report, we share our experience of caring for a post Covid-19 who suffered from worsening symptoms of breathlessness. Clinicians should be aware of post Covid-19 syndrome, and try to find the underlying cause and solve the problems. A 65-year-old man came to Universitas Islam Indonesia Hospital with severe dyspnea 3 months after discharged from hospitalization for Covid-19 diagnosis. Clinical examination showed bilateral massive pleural effusion and was confirmed by chest x-ray. From echocardiography, we found a 42% decrease in ejection fraction of systolic function. After furosemide treatment, there was only marginal improvement. In order to reduce the pleural effusion, we conducted pleural effusion tapping, and we tapped 1200 ml serous discharge from the right pleura and 1100 ml from left pleura. From the laboratory examination we concluded that the discharge was transudation from reduced cardiac function. There was no evidence of bacterial infection nor mycobacterial tuberculosis infection. The patient had diabetes mellitus as comorbidity; however, the blood glucose remained stable and controlled with diet during hospitalization. Follow-up several months after discharge showed that maintenance treatment with diuretic, beta-blocker and Angiotensin II Receptor blocker showed good results and there was no recurrence of pleural effusion. Post Covid-19 symptoms can manifest as severe dyspnea from pleural effusion caused by reduced cardiac function. Several diagnostic tests should be performed to exclude other causes of pleural effusion. The symptom will improve with pleural tapping and administration of diuretic, beta-blocker and Angiotensin II receptor blocker.

Keywords: Post Covid-19 · Pleural Effusion · Congestive Heart Failure

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1 Introduction

Covid-19 pandemic has lasted for more than 1,5 years since December 2019. It has been spreading throughout the world and impacts more than 200 countries. Until October 2021, there are more than 241 million Covid-19 patients with more than 4.9 million deaths [1]. There are many factors that are associated with severe manifestations and mortality of Covid-19, namely: elderly age, male sex, previous chronic lung disease, smoking, previous cardiovascular disease, hypertension, diabetes mellitus, chronic kidney disease, chronic liver disease, cancer, disability, and immunodeficiency [2].

Covid-19 can induce acute and chronic conditions that correlates with deterioration of cardiac function, especially heart failure [3]. Many patients come to hospital with Post Covid-19 syndrome, particularly with symptoms of dyspnea. Clinicians should be aware of post Covid-19 syndrome and have to identify the underlying cause and solve the problems. This paper will describe a case of post Covid-19 patients that suffered from acute decompensated heart failure with early manifestation of pulmonary oedema and pleural effusion due to heart failure.

2 Method

3 Result and Discussion

3.1 History

A 65-year-old man came to Universitas Islam Indonesia Hospital with chief complaint dyspnea or shortness of breath. Three months before, he was diagnosed with Covid-19 and hospitalized at a hospital at Bantul region, Yogyakarta, Indonesia and after recovery, he was discharged from the hospital. For several weeks at home he was suffering from shortness of breath and returned to the hospital, but there was no improvement, so he came to Universitas Islam Indonesia Hospital. He had Diabetes Mellitus as comorbidity which was treated with antidiabetic drugs, but the medication had already been stopped due to hypoglycemia and is already stable with only diet restriction.

3.2 Physical Examination, Laboratory Result and Imaging

The result of physical examination upon arrival at the hospital showed blood pressure 136/98 mm Hg, heart rate 130 x/minute, respiration rate 35 x/minute, temperature 37 °C, oxygen saturation: 99% using non rebreathing mask 12 L/minute. The patient had an increase in blood pressure, tachycardia, and severe dyspnea.

From thorax examination, we found symmetrical movement of chest wall from inspection, decreased tactile fremitus in the lower area from palpation, dullness in lower area of thorax from percussion, while from the auscultation we found decreased vesicular sound from ICS V to the lower lung field, and crackles in upper lung field. From cardiac examination, cardiomegaly was suspected. From the thorax examination, pleural effusion was suspected.

Electrocardiography showed sinus tachycardia, heart rate 107 x/m, left atrial hypertrophy, left axis deviation, old myocardial infarct at inferior and anterior (Fig. 1).

From the chest X-ray we found bilateral pleural effusion and early pulmonary congestion, but cardiac size could not be assessed (Fig. 2).

From blood examination, we found microcytic hypochromic anemia, which might be related to chronic disease. Leucocyte count, thrombocyte count and differential count of leucocytes were within normal limits (Table 1).

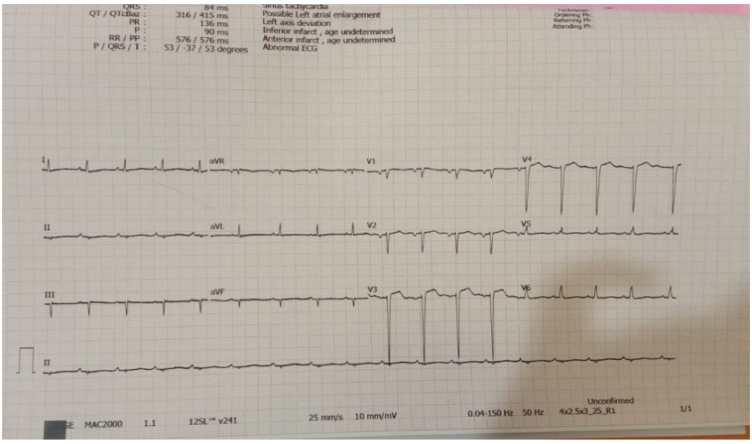

Fig. 1. *Electrocardiography*

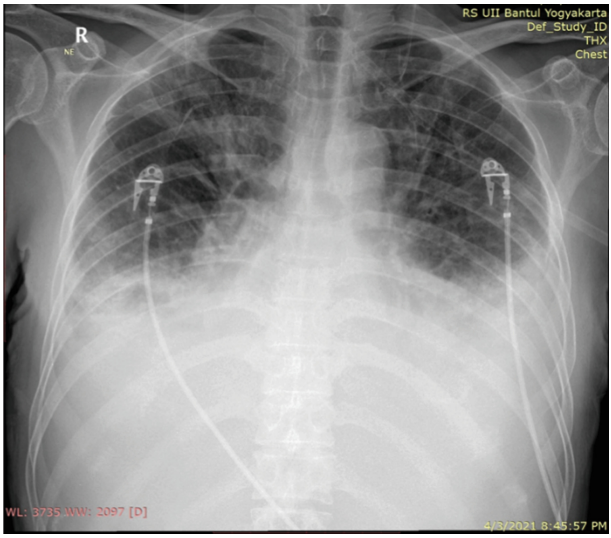

Fig. 2. *Chest X Ray at Arrival*

From chemical and serological examination, we found increased Troponin I, increased blood glucose and negative SARS CoV2 result from rapid antigen and PCR examination (Table 2).

From the electrolyte, liver and renal function examination we found slight hypokalemia and hyponatremia and slight increase in creatinine. Liver function was normal (Table 3).

3.3 Diagnosis

Diagnosis of the patient were: Post Covid-19 with bilateral pleural effusion, Congestive Heart Failure functional class III et causa Ischemic Heart Disease, Diabetes Mellitus

Table 1. *Blood Examination*

Item	Level	Normal Value	Interpretation
Hemoglobin	12.0g%	13.2–17.3	Low
Erythrocyte	5.07 millions/ul	4.4–5.99	Normal
Hematocrite	35.5%	40–52	Low
Thrombocyte	352 thousand/uL	150–450	Normal
Leucocyte	9.77 thousand/uL	3.8–10.6	Normal
Neutrophyl	68.2%	28–78	Normal
Lumphocyte	24.7%	25–40	Normal
Monocyte	6%	2–8	Normal
Eosinophyl	1.0%	2–4	Low
Basophyl	0.1%	0–1	Low
NLR	2.76	1–3	Normal
MCV	70.0 fL	80–100	Low
MCH	23.7 pg	26–34	Low

Table 2. *Chemical dan Serologic Examination*

Item	Level	Normal Value	Interpretation
Troponin I	169 ng/mL	≥100(+)	Increase
Antigen SARS-CoV2	Negative	Negative	Normal
PCR SARS-CoV2	Negative	Negative	Normal
Blood gulose random	159 mg/dL	75–140	Slight increase
Fasting blood glucose	141 mg/dL	75–100	Slight increase
Post prandial blood glucose	143 mg/dL	<140	Slight increase

Table 3. *Electrolyte, Liver and Renal Function Examination*

Item	Level	Normal Value	Interpretation
Natrium	133 mmol/L	135–147	Slight decrease
Kalium	3.0 mmol/L	3.5–5.0	Slight decrease
Chloride	98 mmol/L	95–105	Normal
Aspartate transderase	23 U/L	<35	Normal
Alanine transferase	13 U/L	<31	Normal
Ureum	25 mg/dL	17–50	Normal
Creatinine	1.53 mg/dL	0.75–1.35	Slight increase

Type II, microcytic hypochromic anemia of chronic disease, electrolyte imbalance and Acute Kidney Injury.

3.4 Treatment

Treatment that was given to the patient were: oxygenation with NRM 12 L/minute, 1900 cal diet for diabetic patient, NaCl 0.9% infusion 16 drops/minute, Furosemide injection 20 mg/8 h, Spironolactone 1 × 12,5 mg, Nebilet (Nebivolol) 2 × 5 mg, Valsartan 2 × 80 mg, Aspar K 3 × 300 mg, Aspilet (Aspirin) 1 × 80 mg and Meropenem injection 500 mg/8 h.

3.5 Follow Up

After 3 days of treatment with Furosemide, Valsartan, Nebivolol, Aspilet and antibiotics, there was only marginal improvement of the dyspnea, therefore we conducted pleural effusion tapping at the fourth and sixth day of hospitalization. From the left pleura, we tapped 1100 ml of serous liquid, and from the right pleura we tapped 1200 ml of serous liquid. After the treatment, the patient felt significant improvement in the dyspnea symptom (Figs. 3 and 4).

The patient also underwent an echocardiography examination. From the echocardiography examination we found: Left Ventricle dilatation, Ejection Fraction (EF) 42%, Tricuspid Annular Plane Systolic Excursion (TAPSE) 14 mm indicating decreased contractility of right ventricle (normal > 18 mm), Diastolic dysfunction of Left Ventricle pseudo-normal type, regional wall motion abnormality (RWMA) (+), mild Mitral Regurgitation and mild Tricuspid Regurgitation.

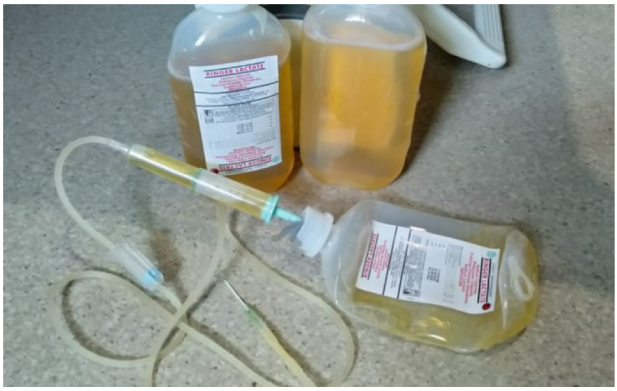

Fig. 3. *Result of Left Side Pleural Tapping*

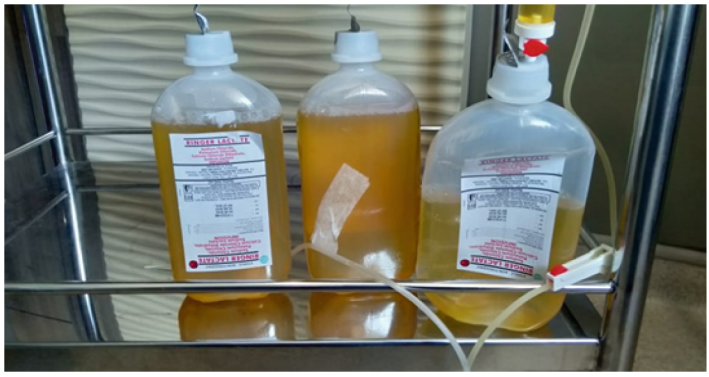

Fig. 4. *Result of Right Side Pleural Tapping*

The examination indicated that there were ischemic heart disease with decrease of systolic and diastolic function or cardiomyopathy.

We conducted pleural effusion liquid analysis, Ziehl Nielsen examination of sputum to find acid resistant mycobacteria and gene expert (rapid molecular examination) to exclude Tuberculosis infection. The result of pleural effusion analysis showed that the liquid was transudate and there was no evidence of Tuberculosis infection. Ziehl Nielsen examination of sputum was negative and gene expert showed no Mycobacterium tuberculosis gene. The ADA test result was 11.9, indicating there was no tuberculosis infection as the cause of bilateral pleural effusion in this patient (Tables 4 and 5).

Table 4. *Chemical Pleural Effusion Analysis*

Item	Level	Normal Value	Interpretation
Color	Yellow	No color	Abnormal
Turbidity	Clear	Clear	Normal
Specific grafity	1.015	<1.018 transudate >1.018 exudate	Transudate
Rivalta test	Negative	Transudate < 3 Exudate > 3	Transudate
Protein	1.6 g/gL	<200 transudate >200 exudate	Transudate
LDH	106 IU/L	<plasma glucose: transudate ≥plasma glucose exudate	Transudate
Glucose	200 mg/dL		Exudate

Table 5. *Microscopic and Serologic Test of Pleural Effusion*

Item	Level	Normal Value	Interpretation
Leucocyte	500 /uL	<1000	Normal
Erythrocyte	8.000	<10.000	Normal
Polynuclear	24%	<25%	Normal
Mononuclear	76%	<75%	Slight increase
ADA test	11.9	<40 normal >40 tuberculosis infection	Normal
Conclusion			Transudate
			Not tuberculosis infection

3.6 Discharge Planning

Patient was discharged after 6 days of hospitalization with the following treatments: Furosemide, Nebivolol, Valsartan, Spironolactone, AsparK, and Cefixime. Follow-up after 5 days post-discharge showed improvement in the shortness of breath symptom.

3.7 Follow-Up at the Outpatient Clinic

Follow-up at the outpatient clinic showed significant improvement in the shortness of breath symptom. The congestive heart failure remained stable and there was no evidence of pleural effusion development. Evaluation of the chest x-ray showed only minimal left pleural effusion (Fig. 5).

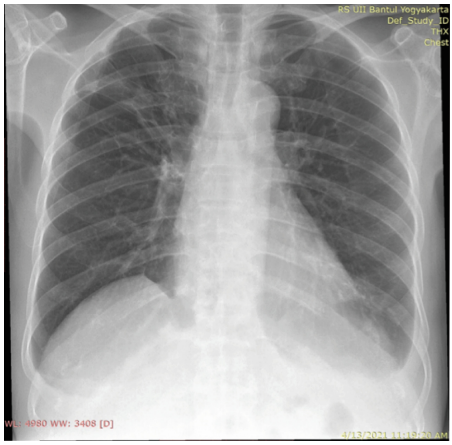

Fig. 5. *Chest X-Ray Evaluation Showed Minimal Left Pleural Effusion and Cardiomegaly*

4 Discussion

Our patient suffered from post Covid-19 syndrome with shortness of breath due to bilateral pleural effusion and early pulmonary congestion. This sign and symptom might be caused by acute chronic decompensated heart failure induced by Covid-19. Three months previously, he was diagnosed with Covid-19 and hospitalized at a regional hospital. He had diabetes mellitus as comorbidity which increases the risk of developing cardiovascular disease such as ischemic heart disease and higher risk of developing cardiovascular events during acute and chronic recovery phase of Covid-19.

Pleural effusion might be induced by another disease, such as tuberculosis infection. Therefore, we conducted several examinations to exclude the possibility of tuberculosis infection. Pleural analysis showed that the serous liquid was transudate which was likely caused by congestive heart failure. Echocardiography showed left ventricle dilatation with decreased ejection fraction and hypo-contractility of right ventricle, which might indicate ischemic heart disease or Covid-19-induced cardiomyopathy.

Conventional treatment with diuretic, Angiotensin II Receptor Blocker, beta-blocker, anti-Aldosterone (Spironolactone), and pleural effusion tapping resulted in significant symptomatic improvement. Therapy continuation at maintenance dose post-discharge further reduces the symptoms of congestive heart failure and prevents the redevelopment of pleural effusion [4].

Besides respiratory manifestation, COVID-19 is also characterized by cardiovascular involvement, which includes deterioration of pre-existing conditions and inflammation-facilitated acute events. Cardiovascular manifestations include ischemic/inflammatory heart disease, ventricular arrhythmias, conduction disturbances, pulmonary thrombotic events and systemic activation of the coagulation cascade leading to disseminated intravascular coagulation [3].

COVID-19 infection has both intermediate and long-term consequences for the cardiovascular system. In acute infection, troponin elevation is more commonly a consequence of indirect cardiac injury from critical illness and multi-organ dysfunction than direct viral damage to the heart. In resolved infection, special attention should be paid to athletes at-risk for exercise-induced arrhythmias, as well as survivors with residual cardiopulmonary symptoms. Long Covid-19 Syndrome occurred in 60–80% of Covid-19 patients who survived. Several symptoms that developed in long Covid-19 syndrome are chronic fatigue, dyspnea, chest pain, and dysautonomia. Some patients had orthostatic tachycardia that will improve with beta-blocker or Ivabradine treatment [5].

We also treated the patient with ARB i.e. Valsartan, because the use of ARB will not cause deterioration of the Covid-19 condition and is recommended by guideline for treatment of acute heart failure from European Society of Cardiologist. A study confirmed previous observations suggesting that underlying cardiovascular disease is associated with an increased risk of in-hospital death among patients hospitalized with Covid-19. The result of the study did not confirm previous concerns regarding a potential harmful association of ACE inhibitors or ARBs with in-hospital death in this clinical context [6].

Our patient had a decreased ejection fraction (42%). This is in line with data which showed that patients with Covid-19 have higher risk for developing heart failure with preserved ejection fraction (HFpEF) during acute and chronic recovery phase of Covid-19. The mechanism that underlies myocardial injury in Covid-19 patients is either direct injury to cardiac myocytes and systemic inflammation related to Covid-19 [7].

Cardiovascular disease risk factors (hypertension, diabetes mellitus and ischemic heart disease) are prevalent in Saudi patients infected with COVID-19. Framingham Risk Score could be a useful tool to identify Cardiovascular risk factors among COVID-19 patients and predict a complicated course [8].

This patient had underlying disease diabetes mellitus. Covid-19 patients with risk factors such as hypertension, diabetes mellitus and cardiovascular disease have a greater risk of severe Covid-19 and mortality. SARS-CoV2 entered human tissue by binding to the ACE-2 receptor. Hyperinflammation may be the mechanism that mediated cardiovascular disease in patients with severe manifestation of Covid-19 [9].

Myocardial injury could be seen from elevated cardiac enzymes, such as Troponin I, which also increased in this patient. Respiratory illness is the dominant clinical manifestation of COVID-19; cardiovascular involvement occurs much less commonly. Acute cardiac injury, defined as significant elevation of cardiac troponin, is the most commonly reported cardiac abnormality in COVID-19. It occurs in approximately 8–12% of all patients. Direct myocardial injury due to viral involvement of cardiomyocytes and the effect of systemic inflammation appear to be the most common mechanism responsible for cardiac injury [10].

Coronavirus disease 2019 can cause pneumonia and inflammation in other systems, especially cardiovascular systems. Many Covid-19 patients have cardiovascular disease and risk factors. Covid-19 is associated with high inflammation that can cause vascular inflammation, myocarditis, and cardiac arrhythmias. Factors associated with mortality are: elderly age, male sex, and presence of comorbidities such as diabetes mellitus,

hypertension, cardiovascular disease and cerebrovascular disease [11]. In this patient we found the risk factors elderly age, male sex, and diabetes mellitus.

An umbrella review of meta-analysis showed that risk factors associated with mortality of Covid-19 are: renal disease, diabetes mellitus, hypertension, smoking history, cerebrovascular disease, cardiovascular disease, liver disease, and obesity. Cardiovascular complications that are often identified in Covid-19 patients are: acute heart failure, myocardial infarction, deep vein thrombosis, myocardial injury, angina, arrhythmias, pulmonary embolism and venous thromboembolism [12]. In this patient we found acute heart failure.

A meta-analysis of 77317 hospitalized patients from 21 studies showed that cardiovascular complications are frequent among Covid-19 patients and might contribute to adverse clinical events and mortality. The pre-existing cardiovascular comorbidities and risk factors, age and cardiovascular complication during hospitalization correlated with mortality of Covid-19 [13].

Many of Covid-19 patients developed heart disease during hospitalization (7% of all Covid-19 patients and 22% from critical ill patients). Previous congestive heart failure in Covid-19 patients will result in a worse outcome of hospitalization compared to other patients who did not have any heart disease history. Covid-19 may increase the risk of developing heart failure in the future. Covid-19 might decrease heart function so there will be an increase of heart failure symptoms (acute decompensated heart failure) [14].

Cardiovascular disease and its risk factors (diabetes mellitus and hypertension) are closely related with mortality of Covid-19 patients in all ages. Young patients with diabetes, hypertension, and cardiovascular disease had higher risk for mortality from Covid-19 compared to older patients without such risk factors [15]. Fortunately, our patient's condition improved until discharge and during follow-up several months afterwards.

5 Conclusion

We presented a case report of Post Covid-19 patient with bilateral pleural effusion and congestive heart failure with underlying Diabetes Mellitus type 2. Symptoms of shortness of breath were relieved through oxygenation, administration of diuretics, beta-blocker, Valsartan, Spironolactone and pleural tapping. Covid-19 could directly attack the heart, which might decrease systolic and diastolic function of the ventricle or worsen previous heart disease. Clinicians should undertake several diagnostic tests to exclude other causes of pleural effusion in post Covid-19 patients.

Acknowledgment. The patient had given approval to be written in this case report with anonymity. The authors were the team of doctors who cared for this patient from hospitalization until follow-up at the outpatient clinic.

Author's Contribution. Ana Fauziyati was the leader in the doctor's team who cared for this patient in the ward. Bagus Andi Pramono was the cardiologist consultant in the management of this patient. Untung Widodo was the leader of the patient management when the patient was cared in intensive care unit at Universitas Islam Indonesia Hospital.

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