

Case report: Cor pulmonale in COPD

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ABSTRACT

Cor Pulmonale is a condition of altered function and structure of the right ventricle of the heart caused by respiratory disease, mainly due to Chronic Obstructive Pulmonary Disease (COPD). This disease results in increased blood flow resistance in the pulmonary circulation and can lead to right heart failure. A 64-year-old man came to the emergency room with complaints of worsening shortness of breath accompanied by a cough with phlegm, leg edema, and a history of COPD. Physical and laboratory examination revealed cardiomegaly, aortic elongation, and right pleural effusion. The patient was diagnosed with COPD-induced cor pulmonale. Cor pulmonale is closely related to pulmonary hypertension caused by COPD. Chronic hypoxia and other factors such as chronic hypercapnia and anatomical disturbances in the pulmonary vasculature are the main causes of this condition. Diagnosis is made through clinical examination, echocardiography, and CT scan. Treatment involves pharmacological therapy that aims to reduce symptoms and improve respiratory function. Cor pulmonale is a serious condition often caused by COPD. Proper treatment and early diagnosis are essential to prevent further complications.

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INTRODUCTION

The human heart, as a vital organ in the circulatory system, is located inside the chest cavity in the mediastinal area, separated from the other mediastinal structures by the pericardium. The heart is shaped like an evergreen fruit, with dimensions of about 12 cm long, 8 cm wide, and 6 cm thick. The human heart can pump blood 100,000 times a day with a total volume of up to 7,571 liters of blood. Within the mediastinum, the heart is separated into two primary sections: one side accepts blood from the superior and inferior vena cava, while the other side receives oxygenated blood from the lungs through the pulmonary veins. Subsequently, the blood is propelled throughout the body via the aorta (Betts et al., 2017).

The heart is made up of three primary layers: the pericardium, myocardium, and endocardium. The pericardium serves as a protective outer layer to prevent friction, the myocardium is the robust muscle layer responsible for heart function, and the endocardium lines the heart cells and blood vessels from the inside (Betts et al., 2017). The heart is comprised of four

chambers, namely the right atrium, left atrium, right ventricle, and left ventricle, all of which are equipped with valves that control the movement of blood within the heart.

The heart's circulatory physiology is comprised of two primary circulations: the systemic and pulmonary circulations. Deoxygenated blood from the body is received by the right atrium and sent to the right ventricle, which then transports it to the lungs for oxygenation. The oxygenated blood is then delivered back to the left atrium and distributed to the body through the left ventricle (Sloane, 2004).

Cor pulmonale is a medical condition characterized by alterations in the function and composition of the right ventricle of the heart, typically caused by respiratory disorders that result in elevated resistance in the circulation of the lungs. Pulmonary hypertension, chronic hypoxia, hypercapnia, and anatomical disturbances in the pulmonary vasculature are some of the main causes of pulmonary cor. The condition can be chronic or acute, and is often difficult to diagnose clinically (Budev et al., 2003; Das et al., 2017; Voelkel et al., 2006; Weitzenblum & Chaouat, 2009).

The etiology of cor pulmonale can be categorized into obstructive lung disease, restrictive lung disease, and central respiratory insufficiency. Chronic obstructive pulmonary disease (COPD) is the main cause of cor pulmonale, including chronic obstructive bronchitis and emphysema, while restrictive lung diseases such as idiopathic pulmonary fibrosis can also be the cause. Obesity-hypoventilation syndrome is an example of respiratory insufficiency of central origin (Weitzenblum, 2003).

The incidence of cor pulmonale is difficult to ascertain, but is estimated to account for approximately 6-7% of total heart disease. In the UK, approximately 0.3% of the population over 45 years of age are at risk of cor pulmonale, with approximately 60,000 cases of pulmonary hypertension requiring long-term oxygen therapy (Leong et al., 2017).

The development of cor pulmonale is strongly connected to pulmonary hypertension. Alveolar hypoxia, pulmonary vasoconstriction, and changes in pulmonary blood vessels all contribute to higher resistance in the lungs and increased blood pressure. The added strain on the right ventricle could result in the enlargement of the ventricle and eventual failure of the right side of the heart (V. Brashers, 2008; Leong et al., 2017).

Diagnosis of cor pulmonale includes clinical assessment and additional examinations such as echocardiography, CT scan, and MRI. Treatment for cor pulmonale aims to optimize gas exchange, reduce pulmonary hypertension, and treat the underlying disease and its complications. Management includes oxygen therapy, vasodilators, digitalis, diuretics, and anticoagulants, with an individualized approach required for each patient (Ariobimo & Nujum, 2023; Chen et al., 2016).

COPD is the primary reason for cor pulmonale, which is characterized by an ongoing restriction in airflow that cannot be completely reversed. Major contributing factors to COPD include smoking and exposure to hazardous environments. Early diagnosis of COPD is essential to prevent exacerbations and improve the patient's quality of life (American Thoracic Society, 1995; Barnes, 2000; Singh et al., 2019).

RESEARCH METHODOLOGY

A 64-year-old male patient presented to the emergency department reporting an increase in difficulty breathing, along with a productive cough and a past medical history of edema in the legs. The patient also suffered from nausea, vomiting, and a reduced appetite. The individual had previously experienced COPD (Chronic Obstructive Pulmonary Disease) and stroke, with no familial background of such illnesses. The patient used to smoke but stopped a year ago.

The patient arrived at the emergency department reporting difficulty breathing. Shortness of breath has been complained of but felt worse today, Shortness of breath is not affected by weather or dust. The patient's shortness of breath improves when resting. The patient also complained of coughing clear phlegm, sputum was difficult to expel. History of swollen legs is

complained of. The patient also complained of nausea vomiting 3 times SMRS and decreased appetite. The patient used to be a smoker but has quit since 1 year ago.

Impression

The patient has never been on any medication and is not currently on any medication. With regards to the patient's medical background, they had a history of experiencing a stroke and being diagnosed with Chronic Obstructive Pulmonary Disease (COPD), but no history of asthma, drug or food allergies. A history of diabetes mellitus (DM), congenital heart disease, and history of surgery were also denied. In the family history, there were no other family members who had similar complaints as the patient. The patient had a history of hypertension (HT), while a family history of tuberculosis (TB), asthma, and diabetes mellitus was denied. In terms of nutritional intake, the patient was known to eat only once a day and liked spicy food and street snacks.

Physical examination of the patient was conducted on January 18, 2023 in Arjuna Ward 1 of K.R.M.T Wongsonegoro Hospital at 2 pm. The patient's general status showed moderate pain with good consciousness (GCS 15: E4 V5 M6). Vital signs on July 6, 2023 at 10:00 am showed blood pressure 136/89 mmHg, heart rate 94 beats per minute, respiratory rate 28 beats per minute, body temperature 36.2°C, oxygen saturation 91% in room air and 97% with nasal cannula 5 lpm. Anthropometry showed height 162 cm, weight 50 kg, with underweight nutritional status (BMI 14.9 kg/m²).

System examination includes: normocephalic head without deformities or lumps, dark hair. The eyes show no signs of conjunctival anemia or scleral ictericity, with normal light reflexes. Ears and nose are normal with no secretions or septal deviation. The mouth appeared normal with no lip cyanosis or tongue abnormalities, and the pharynx showed no hyperemia. The neck appears with elevated JVP. Lung examination revealed normal vesicular breath sounds with coarse wet rales and no wheezing. Cardiac examination revealed a murmur at ICS IV linea midklavicularis sinistra. Abdomen appeared flat with epigastric tenderness, umbilicus, and positive muscular defans. Examination of the extremities and skin showed CRT less than 2 seconds, and the lymph nodes were not palpably enlarged.

Table 1. Hematology laboratory (January 12, 2023)

| Examination | Results | Unit | Normal Value |
|-----------------------------------|--------------|--------|--------------|
| Random Blood Sugar | 107 | mg/dL | 70-110 |
| Calcium | 1.32 | mmol/L | 1.00-1.15 |
| Potassium | 5.00 | mmol/L | 3.50- 5.0 |
| Natrium | 139.0 | mmol/L | 135-147 |
| Triglycerides | 146 | mg/dL | <=150 |
| Total Cholesterol | 175 | mg/dL | <200 |
| Creatinine | 1,7 | Mg/dl | 0,6-1,1 |
| Urea | 55,8 | Mg/dl | 17-43,0 |
| Hemoglobin | 11.2 | g/dL | 11-15 |
| Hematocrit | 36.10 | % | 40-52 |
| Platelet count | 498 | /uL | 150-400 |
| PT control | 10.5 | second | |
| HIV 1 reagent | Non-reactive | | |
| Rapid antigen test for SARS-CoV-2 | Negative | | |

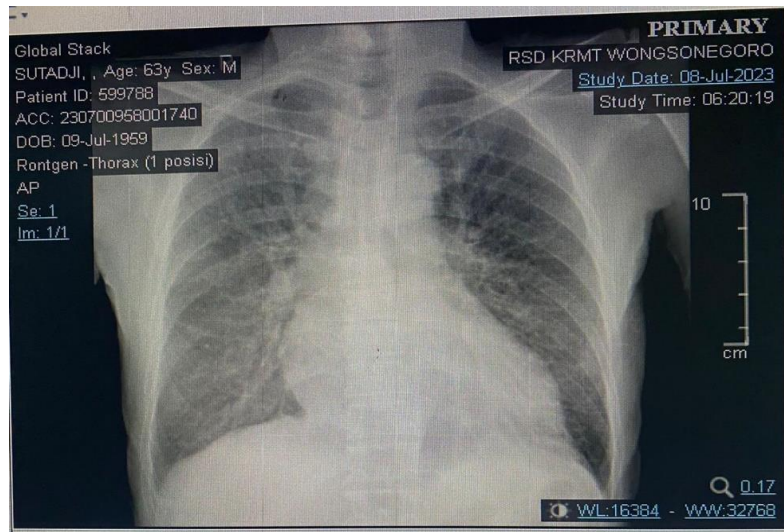


Figure 1. Chest X-ray (January 12, 2023)

The examination revealed lateral and caudal widening of the cardiac apex (laterocaudal widening of the cor apex) and elongation of the aorta. In the lungs, there was increased vascular patterning, with patches seen in both the right perihillary and paracardial areas. The diaphragm and left costophrenic sinus appear normal, but the right costophrenic sinus appears obtuse. Bones and soft tissues are in good condition. Based on these findings, the medical impression was left ventricular cardiomegaly (LVH), aortic elongation, bronchopneumonia, and pleural effusion on the right side.

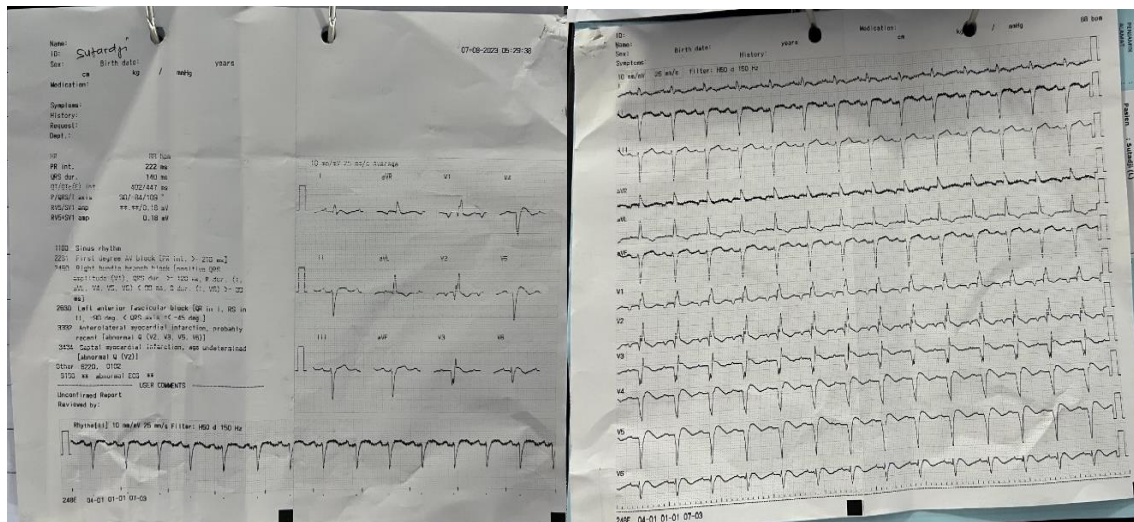


Figure 2. Electrocardiogram (ECG) results (January 12, 2023)

Interpretation:

AV Block

RBB

The patient was diagnosed with cor pulmonale due to COPD (Chronic Obstructive Pulmonary Disease). Regarding pharmacological treatment, the patient was given several therapies, namely ranitidine injection twice a day, ondansetron injection twice a day, mecobalamin injection once a day, and methylprednisolone injection with a dose of 2 x 62.5 mg. In addition, the

patient also received nebulization therapy with combivent and flixotide every 8 hours. Based on the assessment of the patient's condition, the prognosis *ad vitam* (life expectancy), *ad functionam* (body function), and *ad sanationam* (disease recovery) all showed good results or "*ad bonam*."

RESULTS AND DISCUSSIONS

The human heart is situated in the chest cavity, positioned between the lungs in an area called the mediastinum (Saminan, 2019)(Hasrianto et al., 2023)(Firdaus, 2024). It displays the heart's placement in the chest cavity. The heart in the mediastinum is isolated from other mediastinal structures by the pericardium, also known as the pericardial sac, and is contained in a chamber called the pericardial cavity (Betts et al., 2017)(Mahendra et al., 2023)(Adelia et al., 2023)(Mafruchati, n.d.). Physical and laboratory examination revealed cardiomegaly, aortic elongation, bronchopneumonia, and pleural effusion on the right side. The first assessment indicated cor pulmonale caused by COPD, the primary reason for chronic respiratory failure and cor pulmonale, responsible for 80-90% of cases.

Cor pulmonale is a condition characterized by alterations in the function and formation of the right ventricle of the heart, primarily caused by respiratory illnesses that lead to heightened resistance in the pulmonary circulation. The most common association between cardiac and pulmonary dysfunction in cor pulmonale is pulmonary hypertension (Budev et al., 2003; Weitzenblum & Chaouat, 2009). In this patient, COPD most likely caused pulmonary hypertension, which caused the right ventricle to have an increased workload or afterload, resulting in cardiomegaly and right heart failure. The findings of cardiomegaly and pleural effusion on the right side as well as the radiologic examination results showing increased vascular patterning and bronchopneumonia support the diagnosis of cor pulmonale. Cor pulmonale typically requires mean pulmonary artery pressure to be over 20 mmHg. Complete right ventricular failure will happen when the average pressure in the pulmonary artery reaches 40 mmHg. Chronic hypoxia is thought to often cause constriction of pulmonary arterioles due to the increased action of physiologic mechanisms that occur to maintain the balance of ventilation and perfusion in the lungs (Weitzenblum & Chaouat, 2009).

Other mechanisms that could potentially increase pulmonary arterial pressure leading to cor pulmonale include chronic hypercapnia and respiratory acidosis leading to pulmonary vasoconstriction. Furthermore, anatomical disruptions can occur in the pulmonary vascular system due to primary pulmonary illnesses like pulmonary fibrosis, pulmonary thromboembolic disease, and emphysema (Voelkel et al., 2006).

In chronic respiratory conditions, pulmonary hypertension results from elevated pulmonary vascular resistance (PVR), with normal cardiac output and pulmonary capillary pressure; This type of pulmonary hypertension is known as precapillary. Alveolar hypoxia is the predominant factor among many that contribute to increased PVR in chronic respiratory diseases (Fishman, 1976), in COPD and obesity-hypoventilation syndrome, as well as kyphoscoliosis. Two distinct ways that alveolar hypoxia works should be taken into account: sudden hypoxia leads to narrowing of the pulmonary blood vessels, while prolonged hypoxia results in alterations to the structure of the pulmonary vascular system (pulmonary vascular remodeling) (Leong et al., 2017).

Findings on CT scan and echocardiography supported this diagnosis by showing changes in the right ventricle and pulmonary artery pressure. In terms of management, the patient was given pharmacological therapy including ranitidine, ondansetron, mecobalamin, methylprednisolone, as well as nebulization with combivent and flixotide. These therapies aimed to reduce symptoms, manage inflammation, and improve respiratory function, which is in line with the principles of cor pulmonale treatment that aim to address the underlying cause and associated symptoms.

Cor pulmonale can also develop from elevated blood thickness caused by lung disease and its complications like secondary polycythemia. It is generally a progressive chronic process but can

also occur acutely in some cases due to cases of acute pulmonary hypertension, often following pulmonary embolism (Das et al., 2017).

This case showed clinical signs consistent with cor pulmonale, including shortness of breath, cough with phlegm, and edema of the lower extremities. Physical and laboratory examinations, such as elevated blood pressure and decreased oxygen saturation, supported the diagnosis of cor pulmonale. Thorax and abdominal X-rays showing cardiomegaly of the left ventricle, aortic elongation, and pleural effusion on the right side were also consistent with the pathophysiologic findings of cor pulmonale, where pulmonary hypertension plays a role in increasing right ventricular afterload.

Clinical signs on the body do not provide a detailed description of the disease and some of them, such as signs related to increased jugular venous pressure, may not be visible in many COPD patients because of chest hyperinflation. The differentiation of acute and chronic lung disease depends on the patient's medical background and physical assessment (V. L. Brashers, 2008). Increased morbidity and mortality are linked to right ventricular failure (Alajaji et al., 2016). Right heart failure characterized by peripheral edema may occur. The timeframe from the beginning of HP to the development of right heart failure is uncertain and can differ depending on the patient, organs, elevated arterial pressure, and hepatomegaly. Ankle edema is a sign of RHF that indicates poor prognosis in patients with coronary artery disease, but is not specific and may be caused by other things. Peripheral edema indicates hyperaldosteronism due to impaired renal function. This condition is caused by hypercapnic acid and hypoxemia. Tricuspid regurgitation is a late symptom. It is due to right ventricular enlargement. The strength of the second heart sound's respiratory aspect is only noticeable in patients with severe HP (Verma, 2016).

Identifying right ventricular hypertrophy through electrocardiography is very accurate, however, it lacks in sensitivity. A regular ECG does not rule out the potential presence of hypertension, particularly in individuals with COPD. Likewise, radiographic signs of pulmonary hypertension, such as right pulmonary artery enlargement, are less sensitive, and radiographic images of right ventricular enlargement are late (and consistent) signs. Currently, the diagnosis of hypertension is usually made by echocardiography. Continuous wave Doppler echocardiography, utilizing Bernoulli's equation, enables the estimation of the transtricuspid pressure gradient based on the peak beam velocity of tricuspid regurgitation. With a right atrial pressure of 5 mm Hg, the right ventricular systolic pressure can be determined by adding the transtricuspid pressure gradient, resulting in the equivalent pulmonary artery systolic pressure. One can also estimate the pulmonary artery diastolic pressure by combining the right atrial pressure with the end-diastolic pressure gradient between the pulmonary artery and the right ventricle. Pulsed-wave Doppler echocardiography, which also assesses flow speed, enables an indirect calculation of pulmonary artery systolic pressure. Yet, echocardiography is challenging to conduct in numerous COPD patients due to hyperinflation, resulting in unsuccessful measurements in 60-80% of cases. In COPD patients, there is often not a consistent confirmation of a strong correlation between pulmonary artery pressure calculated from echocardiographic data and pressure measured invasively, resulting in an average estimation error for pulmonary artery pressure of around 10 mm Hg (Verma, 2016). Echocardiography plays a crucial role in evaluating both the structure and function of the right ventricle in various types of pulmonary hypertension (PH) (Widiastari et al., 2022). The TAPSE method is widely used and established for longitudinally evaluating right ventricular function. The typical range for TAPSE values is between 2.3 and 2.7 cm. A tricuspid annular plane systolic excursion (TAPSE) below 1.8 cm reliably forecasts a decrease in stroke volume index as measured invasively. TAPSE also correlates significantly with other markers of right ventricular impairment and is linked to higher rates of hospitalization and mortality in patients with pulmonary hypertension. Pericardial effusion may develop in individuals with advanced HP and serves as a negative prognostic factor. Doppler echocardiography is a non-invasive tool that is more frequently used in clinical practice to detect HP in patients with active

tuberculosis. Higher pulmonary artery pressure detected via Doppler echocardiography is significantly associated with mortality rates in new cases of pulmonary tuberculosis.

CONCLUSION

Individuals with a background of Chronic Obstructive Pulmonary Disease (COPD) experience cor pulmonale, which is identified by pulmonary hypertension, cardiomegaly, and right ventricular failure. This condition was supported by clinical and radiological findings such as right ventricular enlargement and pleural effusion. The patient received pharmacological treatment to reduce symptoms and improve respiratory function. The patient's prognosis is considered good, but proper management is essential to prevent further complications.

References

- Adelia, N. G., Kep, M., Zul'irfan, N. M., Kep, M., Roni, N. Y., Kep, M., Kharisna, N. D., Kep, M., Azhar, N. B., & Kep, M. (2023). *PATOFISIOLOGI SISTEM PERNAPASAN*. CV Pena Persada.
- Alajaji, W., Baydoun, A., Al-Kindi, S. G., Henry, L., Hanna, M. A., & Oliveira, G. H. (2016). Digoxin therapy for cor pulmonale: a systematic review. *International Journal of Cardiology*, 223, 320-324. <https://doi.org/10.1016/j.ijcard.2016.08.018>
- American Thoracic Society. (1995). Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*, 152, 73-78.
- Ariobimo, B. N., & Nujum, N. (2023). Kor Pulmonal pada Tuberkulosis Paru: Sebuah Laporan Kasus. *Jurnal Kedokteran Meditek*, 29(3), 301-307. <https://doi.org/10.1136%2Fheart.89.2.225>
- Barnes, P. (2000). *Chronic obstructive pulmonary disease*. N Engl J Med.
- Betts, J. G., Colledge, T. J., Desaix, P., Johnson, J. E., Wise, J. A., Womble, M., & Young, K. A. (2017). The Cardiovascular System: The Heart. In *Anatomy & Physiology*. Rice University.
- Brashers, V. (2008). *Aplikasi klinis patofisiologi. Edisi ke-2*. Jakarta: Penerbit EGC.
- Brashers, V. L. (2008). *Aplikasi Klinis Patofisiologi: Pemeriksaan & Manajemen edisi 2*, diterjemahkan oleh Kuncara, H. Y. Penerbit Buku Kedokteran EGC.
- Budev, M. M., Arroliga, A. C., Wiedemann, H. P., & Matthay, R. A. (2003). Cor pulmonale: an overview. *Seminars in Respiratory and Critical Care Medicine*, 24(03), 233-244. <https://doi.org/10.1055/s-2003-41105>
- Chen, Y., Liu, C., Lu, W., Li, M., Hadadi, C., Wang, E. W., Yang, K., Lai, N., Huang, J., & Li, S. (2016). Clinical characteristics and risk factors of pulmonary hypertension associated with chronic respiratory diseases: a retrospective study. *Journal of Thoracic Disease*, 8(3), 350.
- Das, S. K., Choupoo, N. S., Saikia, P., & Lahkar, A. (2017). Incidence proportion of acute cor pulmonale in patients with acute respiratory distress syndrome subjected to lung protective ventilation: a systematic review and meta-analysis. *Indian Journal of Critical Care Medicine: Peer-Reviewed, Official Publication of Indian Society of Critical Care Medicine*, 21(6), 364.
- Firdaus, N. I. (2024). BAB 8 Sistem Kardiovaskular. *BUNGA RAMPAI ANATOMI FISILOGI TUBUH MANUSIA*, 85.
- Fishman, A. (1976). *Chronic cor pulmonale*. *Am Rev Respir Dis* 1978;114:775-94. ▶ A state of the art report on chronic cor pulmonale by one of the major experts in this field.
- Hasrianto, R. T., Jeniyanthy, N. P. R., & Mahendrayana, I. M. A. (2023). Pengaruh Variasi Window Width Dan Window Level Pada Lung Window Terhadap Kualitas Citra CT Scan Thorax Dengan Klinis Tumor Paru Di Rumah Sakit TK II Pelamonia Makassar. *Compromise Journal: Community Professional Service Journal*, 1(4), 1-10.
- Leong, D., Dave, R. H., & Kocheril, A. G. (2017). Cor Pulmonale: overview of cor pulmonale management. *Accessed Dec*, 15, 1-17.
- Mafruchati, M. (n.d.). *Mikroba Patogen Pada Sistem Organ dan Bakteri Penyebab Mastitis Sebagai Dasar Kajian Pada Embrio Hewan*. Zifatama Jawara.
- Mahendra, D., Hakim, R. I., Wulandari, P., Miftahuddin, D., Gelung, A., & Aini, U. N. (2023). *EFEKTIF DIAGNOSIS JANTUNG Peran-Sistole, Diastole dan Detak Jantung*. Airlangga University Press.
- Saminan, S. (2019). Efek Kelebihan Berat Badan terhadap Pernafasan. *Jurnal Kedokteran Nanggroe Medika*, 2(4), 27-33.
- Singh, D., Agusti, A., Anzueto, A., Barnes, P. J., Bourbeau, J., Celli, B. R., Criner, G. J., Frith, P., Halpin, D. M.

- G., & Han, M. (2019). Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2019. *European Respiratory Journal*, 53(5).
- Sloane, E. (2004). *Anatomi dan Fisiologi Untuk Pemula*. Jakarta: Penerbit Buku Kedokteran EGC.
- Verma, A. K. (2016). Tuberculosis and pulmonary hypertension: Commentary. *Lung India*, 33(2), 232-233.
- Voelkel, N. F., Quaipe, R. A., Leinwand, L. A., Barst, R. J., McGoon, M. D., Meldrum, D. R., Dupuis, J., Long, C. S., Rubin, L. J., & Smart, F. W. (2006). Right ventricular function and failure: report of a National Heart, Lung, and Blood Institute working group on cellular and molecular mechanisms of right heart failure. *Circulation*, 114(17), 1883-1891. <https://doi.org/10.1161/CIRCULATIONAHA.106.632208>
- Weitzenblum, E. (2003). Chronic cor pulmonale. *Heart*, 89(2), 225-230.
- Weitzenblum, E., & Chaouat, A. (2009). Cor pulmonale. *Chronic Respiratory Disease*, 6(3), 177-185. <https://doi.org/10.1177/1479972309104664>
- Widiastari, E. F., Graha, W. A., & Pardede, M. (2022). Lung Bullae Due to Septic Pulmonary Embolism in a 4-Year-Old Child: A Case Report. *Pharmacology, Medical Reports, Orthopedic, and Illness Details (COMORBID)*, 1(3). <https://doi.org/10.55047/comorbid.v1i3.355>