

# PROLONGED VENTILATOR IN ICU PUTRI HIJAU HOSPITAL WHAT SHOULD WE DO IN BPJS ERA

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## ABSTRACT

*Number of patients with chronic kidney disease (CKD) is increasing progressively worldwide, especially in developing countries. In Indonesia, the largest proportion of hemodialysis patients is due to hypertension and diabetes. Chronic kidney disease (CKD) is a progressive disease and causes variety of complications, which contribute to high mortality, morbidity and poor quality of life. In many cases, chronic kidney disease affects lung function directly or indirectly. Impaired renal filtration results in tissue edema that affects gas exchange processes and lead to life-threatening conditions. The clinical manifestations of patients with pulmonary edema are shortness of breath, tachypnea, cyanosis and use of accessory muscles of respirations and develop into acute respiratory distress syndrome (ARDS). The goals of treatment for pulmonary edema are to reduce symptoms, improve ventilation and adequate oxygenation, maintain cardiac output and perfusion to vital organs. Patients with pulmonary edema require mechanical ventilation and intensive care support. The duration of ventilator use in pulmonary edema varies from 1 to 40 days. Prolonged ventilator use is a challenge to hospital financing in BPJS Kesehatan era with current INA-CBGs tariff system.*

**Keyword:** Pulmonary Edema, ARDS, CKD, Kidney Failure.

## BACKGROUND

Number of patients with chronic kidney disease (CKD) is increasing progressively worldwide, especially in developing countries (Cuzzolino, Galassi, Pivari, Ciceri, & Conte, 2017). According to Hill et al, 2016, the global prevalence of CKD is 13.4% and considered as the largest economic burden on the health system (Hill et al., 2016). In Indonesia, the largest proportion of hemodialysis patients is due to hypertension and diabetes.

Treatment of kidney disease is the second largest financing from BPJS Kesehatan after heart disease (Kemenkes RI, 2017). Based

on BPJS Kesehatan data in 2014, there were 1.4 million cases of kidney failure at a cost of 2.2 trillion rupiah. As of the third quarter of 2015, there were 1.2 million cases of kidney failure funded by BPJS Kesehatan which amounted to 1.6 trillion rupiah (Susanto, 2016).

Chronic kidney disease (CKD) is a progressive disease and causes a variety of complications, which contribute to high mortality and morbidity and poor quality of life. Complications occur in almost all systems, such as cardiovascular, endocrine and metabolic, gastrointestinal, hematology, neurology, respiratory and musculoskeletal.

In the respiratory system, pulmonary edema and calcifications may occur (Bello et al., 2017; Harrison, 2018; Long, Koyfman, & Lee, 2017).

Pulmonary edema is an acute clinical condition characterized by symptoms of respiratory distress and tachypnea that are proportional to a decrease in PaO<sub>2</sub>. Physiological disturbances that cause hypoxaemia are ventilation-perfusion imbalances (ventilation-perfusion mismatch) (Perhimpunan Dokter Paru Indonesia (PDPI), 2019). The clinical manifestations of pulmonary edema are characterized by acute shortness of breath, tachypnea, cyanosis and use of accessory muscles of respiration (Assaad, Kratzert, Shelley, Friedman, & Perrino, 2018).

## CASE

Mr. Yondrizal, 44 years old, was referred to Putri Hijau Hospital Medan from Metta Medika Hospital Sibolga with loss of consciousness, shortness of breath and fever. The patient had history of diabetes mellitus, hypertension and stroke 6 years ago. On physical examination, the patient's GCS is 5, BP 156/102 mmHg, pulse 115 x/minute, temperature 39.1°C, RR 40 x/minute, SpO<sub>2</sub> 84% without oxygen and SpO<sub>2</sub> 95% with NRM 15 liters.

On inspection, he had tachypnea and intercostal retractions, dullness on percussion and additional crackles on auscultation of the left lung. From the laboratory examinations, the patient was anemic with hemoglobin level of 10 g/dL, leukocytosis with leukocyte level of 19.520 / $\mu$ L, uremia with urea level of 201 mg/dL, an increase in creatinine with a level of 10 mg/dL, hypoalbuminemia with albumin level of 2.4 g/dL, and hyperlipidemia with total cholesterol level of 247 mg/dL.

From the radiological examination of CT-Scan without contrast at Metta Medika Hospital Sibolga, it was found extensive infarcts in gray and white matter at frontal, parietal and occipital lobes, and right basal ganglia. On chest x-ray examination in Putri Hijau Hospital Medan, the broncho-vascular pattern was increase accompanied by blurring vascular and presence of infiltrate haziness in the middle field of the left lung that supports the appearance of pulmonary edema. So, the patient was consulted to the pulmonary department to get advice and therapy. The patient was diagnosed with cerebral stroke, chronic kidney disease, diabetes mellitus, hypertension and pulmonary edema.

Management and therapy given were intubation and installation ventilator as an airway access, regular hemodialysis twice a week, IVFD RL 20 drops/min, moxifloxacin 400 mg for 24 hours IV, meropenem 1 gr for 8 hour, citicoline 500 mg for 12 hours IV, phenitoin 100 mg for 8 hours IV, furosemide IV, omeprazole 40 mg for 12 hours IV, paracetamol 1000 mg for 8 hours IV, novorapid 8-8-8 SC, levemir 6 units a day SC, clopidogrel 75 mg a day PO, atorvastatin 40 mg a day PO, amlodipine 10 mg a day PO, candesartan 16 mg a day PO, N-acetylsisteine 200 mg three times a day PO, bicnat three times a day PO dan vip albumin three times a day PO. The prognosis for this patient is dubia ad malam.

## DISCUSSION

According to The International Kidney Disease: Improving Global Outcomes (KDIGO), chronic kidney disease is kidney damage in the form of structural or functional abnormalities accompanied by a decrease in the glomerular filtration rate for more than 3 months. Chronic kidney disease is based on the degree of disease, namely

grades 1-5. This classification is made on the basis of the glomerular filtration rate (GFR), which is calculated using the Cockcroft

Gault formula as follows:

$$GFR = \frac{(140 - age) \times weight}{72 \times \text{creatinine plasma (mg/dl)}}$$

Based on the above formula, the patient's GFR value is 9.3 ml/min/1.73m<sup>2</sup> that indicating end stage renal disease (ESDR) or renal failure and require renal replacement therapy such as hemodialysis. Hemodialysis aims to replace the function of kidneys in excretory function (removing metabolic wastes in the body, such as urea, creatinine, and others), improve the patient's quality of life and prevent complications due to uremia that can cause morbidity and mortality (Kerr, Bray, & Medcalf, 2014; Suhardjono, 2010).

Recent studies have shown a link between kidney and organ dysfunction that are far apart. In many cases, chronic kidney disease affects lung function directly or indirectly (Azarkish et al., 2013; M Malek & Maleki, 2018). Impaired renal filtration causes the trans-capillary pressure to increase and cause tissue edema. Edema causes serious effects on the lungs because pulmonary edema affects gas exchange and lead to life-threatening conditions (Maryam Malek, Hassanshahi, Fartootzadeh, Azizi, & Shahidani, 2018).

Pulmonary edema is defined as the accumulation of fluid or protein in the alveoli, resulting from a change process of Starling forces. The causes of pulmonary edema are divided into cardiogenic and non-cardiogenic processes. Non-cardiogenic pulmonary edema is caused by acute respiratory distress syndrome (ARDS), pulmonary embolism, opioid overdose, and this case end stage renal disease or renal failure. Fluid accumulation in the lungs due

to renal failure is the result of water transport regulation to epithelial cells such as ENaC, sodium-potassium ATPase and aquaporin-5 in the lung. This transporter is responsible for the absorption of sodium from the alveoli into the epithelial cells where fluid follows. In addition, the accumulation of inflammatory mediators such as IL-6, IL-8, IL-1 $\beta$  and macrophages initiates the pathological process that causes pulmonary edema and ARDS (Farha & Munguti, 2020; Sakka, 2013).

Clinical manifestations of patients with pulmonary edema are dyspnea, tachypnea and hypoxia. In patients with non-cardiogenic pulmonary edema, symptoms of infection such as fever, cough with phlegm and shortness of breath should be assessed carefully because they can progress to acute respiratory distress syndrome (ARDS). In addition, bilateral extremity edema may be seen. On auscultation, it was found coarse crackles heard during inspiration (R. Malek & Soufi, 2022).

On chest x-ray examination was found alveolar filling patterns. At stage 1 (compensated), a diffuse infiltrate was seen. At stage 2 (perihilar edema), the appearance of interstitial pulmonary edema in the form of Kerley B lines, indistinct vessels and peribronchial cuffing. In stage 3 (alveolar edema), shows a picture of air space opacification in the form of batwing appearance (Assaad et al., 2018).

In principle, pulmonary edema therapy is based on the etiology and pathophysiology. The goals of treatment are to reduce symptoms, improve ventilation and adequate oxygenation, maintain cardiac output and perfusion to vital organs (Purvey & Allen, 2017). In general, management of pulmonary edema with CKD includes basic disease therapy and supportive therapy such as mechanical ventilation, adequate

nutrition, lowering pulmonary artery pressure with diuretics, ultrafiltration or hemodialysis, administration of antioxidants and fluid restrictions (Sakka, 2013).

In pulmonary edema patients with CKD, capillary-alveolar barrier is disrupted due to systemic inflammation, uremia and increased oxidative stress leading to fluid accumulation in the lungs. This condition cannot be significantly improved by simply treating uremia and controlling fluid balance with dialysis because there is role of inflammatory mediators or increased levels of cytokines such as IL-6 and/or IL-8 (Susanto, 2016). In general, after hemodialysis is conducted, there is decrease in urea levels and improvement in the lung function. In this case, there was no significant improvement was found after regular hemodialysis twice a week.

Fluid overload and uremia may be treated with dialysis, but progressive lung and kidney damage leading to mortality in patients with both diseases does not improve significantly with this therapy (Basu & Wheeler, 2011).

According to Roguin et al, 15% of pulmonary edema patients require mechanical ventilation with a mortality of 55% (Roguin et al., 2000). Patients with long term intubation and use of ventilator in ICU at risk of developing lung injury and nosocomial infection that called as ventilator-associated pneumonia (VAP) due to bacterial colonization of the respiratory tract due to the absence of cough reflex and mucus secretion in mechanically ventilated patients (Suhardjono, 2010). the duration of ventilator use in pulmonary edema varies from 1 to 40 days (Roguin et al., 2000).

In Indonesia, the health financing system is implemented by Badan Penyelenggara Jaminan Sosial (BPJS) Kesehatan in accordance with the implementation of The

National Health Insurance / Jaminan Kesehatan Nasional (JKN) which is regulated by Presidential Regulation Number 111 of 2013. The applicable rates are in accordance with The Indonesia Case Base Groups (INA-CBGs) which has is regulated by Health Ministry Regulation Number 69 of 2013 (Suhartoyo, 2018).

INA-CBGs is a system where the amount of claim payment by BPJS Kesehatan at an advanced health facility is a codification system of the final diagnosis and actions or procedures that become service outputs referring to The International Code Disease Ten (ICD 10) and International Code Disease Nine (ICD 9) Clinical Modification (CM) that compiled by World Health Organization (WHO). The INA-CBGs tariff is a package tariff that includes all components of hospital resources used in both medical and non-medical services (Suhartoyo, 2018). So prolonged ventilator use is a challenge to hospital financing in the current BPJS Kesehatan era.

## CONCLUSION

Pulmonary edema is a frequent complication in patients with renal failure that contributes to an increased mortality rate. The relationship of lung damage to renal failure is a consequence of complex biological processes that lead to dysregulation of inflammatory mediators. A better understanding of this relationship is expected to be a gateway to both therapeutic and preventive strategies for reducing high mortality rate in pulmonary edema due to renal failure.

Patients with pulmonary edema require mechanical ventilation and intensive care support. Prolonged ventilator use for patients with pulmonary edema is a challenge to hospital financing in the current BPJS Kesehatan era.

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