Severe Respiratory Acidosis in Acute Exacerbation of Chronic Obstructive Pulmonary Disease, Is that Possible to Treat?

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DOI: 10.55497/majanestcricar.v42i3.395

ABSTRACT

Introduction: Chronic obstructive pulmonary disease (COPD), currently the third leading cause of death worldwide, is a common condition characterized by progressive airflow limitation and tissue destruction. Patients with COPD may present to the emergency room with severe acute exacerbations (AECOPD), which can be associated with acue respiratory failure—a life-threatening condition with a mortality rate approaching 50% in Indonesia—requiring rapid intervention and ICU admission. In this report, we present a severe respiratory acidosis in AECOPD case with successful emergency followed by ICU management and the frequent pitfalls.

Case illustration: In this report we present a-67 years old male, pedicab driver and smoker came to the ER with acute onset shortness of breath and decrease of consciousness with history of shortness of breath in last 10 years. In primary survey we found clear wheezing sound, tachypnea, intercostal retraction, decrease of peripheral oxygen saturation, tachycardia and verbal respond of consciousness. Blood gas analysis result interpreted severe respiratory acidosis with pH 6.90 and pCO_2 128.5 mmHg. Chest radiograph showed infiltrate that became the cause of exacerbation. Endotracheal intubation was performed due to decreased consciousness, persistent tachypnea and pCO2 over 100 mmHg. This patient was hospitalized for 12 days including 9 days in ICU followed by 3 days in regular ward.

Conclusion: The goal for AECOPD management is to minimize and prevent the negative effects of the exacerbation.

Keywords: AECOPD; intubation; mechanical ventilation; respiratory acidosis; respiratory failure

Asidosis Respiratorik Berat pada Penyakit Paru Obstruktif Kronis Eksaserbasi Akut (AECOPD)

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ABSTRAK

Pendahuluan: *Penyakit Paru Obstruktif Kronis (PPOK) yang saat ini menjadi penyebab kematian tertinggi ketiga di dunia adalah penyakit umum yang ditandai dengan keterbatasan aliran udara progresif disertai kerusakan jaringan yang dapat muncul di Instalasi Gawat Darurat (IGD) dengan kondisi gagal napas akut, suatu kondisi yang mengancam jiwa dengan angka mortalitas di Indonesia yang mencapai 50% yang membutuhkan penanganan segara dan perawatan di Ruang Terapi Intensif (RTI). Pada laporan kasus ini, kami menyajikan kasus asidosis respiratorik berat pada PPOK eksaserbasi akut dengan keberhasilan tatalaksana di IGD dan RTI serta kesulitan yang sering terjadi.*

Ilustrasi Kasus*: Dalam laporan ini kami menyajikan seorang laki-laki berusia 67 tahun, seorang tukang becak dan perokok aktif datang ke IGD dengan sesak napas akut dan penurunan kesadaran dengan riwayat sesak napas dalam 10 tahun terakhir. Pada pemeriksaan fisik didapatkan suara mengi yang jelas, takipnea, retraksi interkostal, penurunan saturasi oksigen perifer, takikardi dan respon kesadaran terhadap rangsangan verbal. Hasil analisa gas darah menunjukkan asidosis respiratorik berat dengan pH 6,90 dan pCO_2 128,5 mmHg. Foto polos dada menunjukkan adanya infiltrat yang menjadi penyebab eksaserbasi. Intubasi endotrakeal dilakukan karena penurunan kesadaran, takipnea persisten dan pCO_2 lebih dari 100 mmHg. Pasien ini dirawat di rumah sakit selama 12 hari termasuk 9 hari di RTI diikuti dengan 3 hari di bangsal biasa.*

Simpulan: *Tujuan dari manajemen PPOK eksaserbasi akut adalah untuk meminimalkan dan mencegah efek negatif dari eksaserbasi.*

Kata Kunci: *Asidosis respiratorik; gagal napas; intubasi; PPOK eksaserbasi akut; ventilasi mekanik*

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is recognized as a leading cause of death worldwide. Smoking and exposure to indoor air pollution (including from biomass burning), air pollution and occupational pollutants have been reported as major risk factors in most countries.[1] Smoking accounts for more than 70% of COPD cases in high-income countries. In developing countries, smoking accounts for 30-40% of COPD cases, and household air pollution is a major risk factor.[2]

The global prevalence of COPD is about 12% of the general population,currently ranked the 3rd leading cause of death worldwide, causing 3.23 million deaths in 2019.[2,3] The incidence of COPD is expected to increase in the next 40 years. By 2060, it is estimated that there will be more than 5.4 million deaths annually due to COPD.[3] Data by Indonesian Ministry of Health in 2019, the prevalence of COPD in Indonesia was 5.5% with the highest prevalence in people over 30 years of age. The prevalence continues to increase along with the increasing prevalence of smoking behavior in Indonesia.[4] An Indonesia prospective cohort study in 2021 reported that 25.5% acute respiratory failure (ARF) cases was caused by COPD. Around 44.7% ARF patients had hypercapnia respiratory failure. In hospital mortality rate ARF patients around 48.79%, with patients with COPD mortality rate around 23.4%.[5]

In this report, we present an acute exacerbation COPD (AECOPD) patient with severe respiratory acidosis, our initial emergency management continued with ICU management until outpatient care.

CASE ILLUSTRATION

A-67 years old male patient, came to the Emergency Room (ER) with shortness of breath and decreased consciousness since about an hour before admitted. This patient has history of shortness of breath in the last 10 years, history of hypertension, and smokes cigarette since he was 17 years old, estimated one pack for two days. This patient works as a pedicab driver and exposed to air pollution every day.

In primary survey, we found a clear wheezing sound during his prolonged expiration (early and late expiration) with respiratory rate 40 x/min, peripheral oxygen saturation 87% room air, blood pressure 160/90 mmHg, heart rate 130 x/min, glasgow coma scale (GCS) 345, and temperature 36.8 ºC. When the patient came to the ER with shortness of breath, we immediately administered non-rebreathing mask 15 liters per minute and gave nebulizer with anti-muscarinic agent. In a short period, this patient experienced decrease of consciousness (GCS 224) and hypoventilation. We immediate changed oxygen supplementation using Jackson Rees device and gave positive pressure ventilation to maintain good oxygenation then prepare for intubation.

Blood gas analysis (BGA) with Jackson Rees device, we obtained pH 6.90, pCO_2 128.5 mmHg, pO_2 258 mmHg, HCO3– 25.3 mmol/L, TCO_2 29 mmol/L, BE (base excess) –8 mmol/L and SaO_2 99% (Table 1). Laboratory examination showed HGB 14.72 g/dL, PLT 251 x103/dL, WBC 11.35 x103/dL, blood glucose 294mg/dL, SGOT 47.31 U/L, SGPT 25.46 U/L, serum lactate 2.64 mmol/L, and D-dimer 5340 ng/dL. BUN and serum creatinine within normal limit. This patient's chest x-ray showed infiltrate in right supra hilar region with hyperaeration of both lungs (Figure 1).

Working diagnoses included acute exacerbation of COPD with severe respiratory acidosis, septic condition due to pneumonia and hypercoagulation state. Due to decrease of consciousness, high respiration rate, and high pCO_2 result, endotracheal intubation was performed. Initial mode of mechanical ventilation was set to synchronized intermittent mandatory ventilation (SIMV) and we gave more longer expiration time to eliminate excess CO_2. Initial settings we used were SIMV mode with rate 24 x/min, tidal volume 370 ml, IE ratio 1:2.5, PEEP 5mmHg, and FiO_2 100%.

Pulmonologist gave this patient treatment involved double antibiotics therapy, mucolytic agent, corticosteroid, proton pump inhibitor for ulcers prophylaxis and nebulizer with short acting ß2-agonist combined with anti-muscarinic. Anticoagulant therapy was given by cardiologist. One day after intubation, BGA evaluation was examined and showed improvement in pH, pCO_2, BE, SaO_2 and serum lactate level (Table 1). This patient was admitted to ICU for 9 days and 3

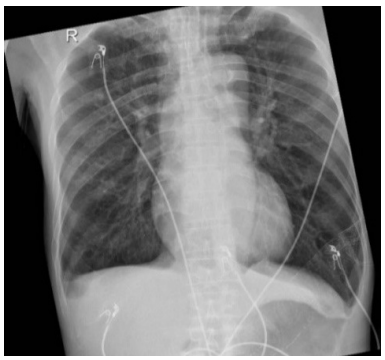

Figure 1. Chest X-Ray in ER

days in pulmonary ward, the following vital signs are listed in Table 2.

During treatment in ICU, patient's respiration was fully controlled for 5 days. Spontaneous breathing trial was started in the 6th day care, and this patient was extubated in the 8th day care and observed for one day and was moved into regular pulmonary ward in 9th day care. During hospitalization in regular pulmonary ward, this patient vital signs remained stable and was discharged by our pulmonologist after 12 days hospitalization.

Table 1. BGA results

Parameter	Normal Range	Pre-intubation in ER	Post-intubation in ER
pH	7.35 – 7.45	6.90	7.28
pCO_2 (mmHg)	38 – 42	128.5	51.3
pO_2 (mmHg)	85 – 100	258	298
HCO_3- (mmol/L)	22 – 26	25.3	24.2
TCO_2 (mmol/L)	19 – 25	29	26
BE (mmol/L)	(–2) – (+2)	-8	-3
SaO_2 (%)	95 – 100	99	100
Lactate	0 – 2	2.64	1.03

Table 2. Vital signs during hospitalization

Vital Signs	ICU									Pulmonary Ward		
	1	2	3	4	5	6	7	8	9	1	2	3
BP (mmHg)	134/97	100/80	117/90	119/90	100/70	107/90	150/83	91/62	91/62	144/87	137/88	153/94
HR (x/m)	81	92	93	92	90	92	83	91	89	100	119	110
RR (x/m)	20	26	20	20	22	22	20	21	21	24	24	22
T (°C)	37.4	36	36.1	36.1	36.5	36.8	37	36.5	36.6	37.2	37.8	36
SaO_2 (%)	100	100	100	99	99	99	99	99	99	94	95	98

DISCUSSION

COPD is a disease characterized by progressive airflow limitation and tissue destruction. It is associated with structural lung changes due to chronic inflammation from prolonged exposure to noxious particles or gases most commonly cigarette smoke. Chronic inflammation causes airway narrowing and decreased lung recoil. The disease often presents with symptoms of dyspnea, cough and sputum production.

Symptoms can vary from being asymptomatic to respiratory failure.[2]

In addition to smoking, a history of asthma, exposure to smoke and air pollution in the workplace can increase risk for COPD. Non-smoking related COPD accounts for about 25-45% of COPD cases. A history of respiratory infection is associated with decreased lung function, increased airway wall thickness and increased COPD exacerbations in adulthood. One of the most important non-smoking risk factors is biomass fuel exposure, which affects about 3 billion people worldwide, and the proportion is higher in developing countries, and impacts more than 80% of the population. This exposure rate is almost three times higher than that of smoking, and the rate of biomass fuel use is higher in developing countries. The most common etiology of respiratory failure in severe COPD patients is followed by pneumonia.[6]

AECOPD with respiratory failure can be diagnosed by history taking, physical examination and supporting examination. The history may include significant smoking exposure (usually 40 packs per year or more), chronic exposure to chemicals/dust from the workplace and patient or family history of respiratory disease. Symptoms may include more frequent coughing, increased sputum production and shortness of breath that interferes with activities. Physical examination focuses on the respiratory and cardiovascular systems. Decrease of consciousness, increased pulse and respiratory rate can be evaluated immediately to determine whether or not there is a possibility of respiratory failure.[7] In this report, patient's risk factors and symptoms were similar to the theory, such as pollution exposure, smoker and history of shortness of breath in the last 10 years.

Patients with AECOPD with respiratory failure should be immediately placed in the resuscitation room. Vital sign monitoring and blood gas analysis (BGA) must be done to determine the type and severity of respiratory failure.[8] The results of BGA may show respiratory acidosis and increase of lactate serum in severe condition. In COPD patients, elevated pCO_2 levels with respiratory acidosis are associated with worse outcomes, including the risk of ICU admission or death.[9] But that doesn't mean it can't be saved. Respiratory acidosis or primary hypercapnia occurs when CO_2 production exceeds lung elimination rates due to alveolar hypoventilation. Increased pCO_2, decreased pH and increased HCO_3– concentration are associated with the occurrence of respiratory acidosis. In this patient we found severe respiratory acidosis pH 6.90 and pCO_2 128.5 mmHg with pneumonia as the underlying cause.

Management of respiratory acidosis with respiratory failure is directed at the underlying cause while providing oxygenation and ventilation.[8] The initial step is to perform a primary survey including maintain airway patency, providing adequate ventilation and oxygen supplementation while stabilizing patients hemodynamic condition.[7] Oxygen therapy may be given with a target saturation 88% to 92%. A systematic review showed that AECOPD patients with saturation 88% – 92% on supplemental oxygen had higher survival rate compared to those with saturation 97% – 100%.[10] Another study showed that the mortality rate of AECOPD patients with 100% supplemental oxygen was 9% (11 out of 117) and 2% (2 out of 97) in the titrated oxygen group. Patients receiving high-flow oxygen are much more likely to develop acidosis or hypercapnia.[11] Oxygen therapy during AECOPD is potentially life-saving, but excessively administered oxygen often decreases survival rates.[12]

Ventilation stimulus in patients with COPD is compensated and manage by peripheral chemoreceptors which more sensitive to oxygen levels than central CO_2. Increased blood oxygen concentration after high oxygen supplementation may reduce respiratory rate and depth of breathing lead to hypoventilation and increase CO2 levels.[13,14] However, we made a mistake at primary survey intervention with giving high concentration oxygen using non-rebreathing mask 15 liters per minute and anti-muscarinic nebulizer that worsen patient's condition proven with decrease of consciousness and hypoventilation.

In AECOPD with acute respiratory acidosis, non-invasive ventilation (NIV) should be considered, especially when pH ≤ 7.25, pCO_2 > 45 mmHg, and respiratory rate > 20-24 x/min.[9] Patients with contraindications to NIV, NIV failure, and

severe respiratory distress. Intubation serves as the definitive airway. Invasive mechanical ventilation can be used as a last resort for acute respiratory failure in COPD. Indications for intubation in patients with AECOPD are similar to those for patients with life-threatening respiratory distress, oxygenation and ventilation failure.[1]

The decision to perform intubation is based on the clinical pathophysiology of the disease, airway assessment, and the likelihood of the condition worsening.[15] There is no absolute limit to the oxygen saturation or CO_2 value that can determine intubation. However, saturation that can not be maintained above 80%, respiration rate > 30 or CO_2 > 100 have a strong association for intubation.[16] Invasive mechanical ventilation is required in AECOPD patients with severe respiratory acidosis and results in good initial survival, but significant long-term morbidity.[17] Similar to this patient who required intubation to drain his excess CO_2 using mechanical ventilation.

After stabilizing the patient and giving adequate ventilation support, we treat the underlying cause of exacerbation. Most commonly used drugs for AECOPD are bronchodilators, corticosteroid and antibiotics. Inhalation of short-acting β2-agonists, with or without short-acting muscarinic antagonists, are recommended for the initial management of AECOPD. Intravenous corticosteroid injection for 7 days reduces the risk of recurrence, risk of treatment failure, and duration of hospitalization. Antibiotics are considered based on the patient's clinical picture and the presence of increased sputum purulence. Antibiotics can also reduce recovery time and duration of hospitalization.[18] This patient was given 3rd generation of cephalosporin, fluoroquinolone, corticosteroid, β2-agonists inhalation during hospitalization. After 12 days admitted, this patient was discharged in better condition.

CONCLUSION

AECOPD patients with severe respiratory acidosis is one of the emergencies that must be treated immediately by general practitioners in the emergency room. The initial step is to perform primary survey (airway, breathing and circulation) assessment as the primary emergency evaluation. Severe respiratory acidosis in AECOPD is possible to treat. Treatment of AECOPD with respiratory failure and severe respiratory acidosis is directed at the underlying cause while providing oxygenation and ventilation. Choosing the right treatment can help reduce patient mortality and morbidity, so knowledge and skills are needed in managing patients in the emergency room, shortening the length of stay in the ICU and hospital care.

CONFLICT OF INTEREST

Authors declare no conflict of interest in the writing of this article.

ACKNOWLEDGMENT

The authors would like to thank Bangil General Hospital for providing access to the data required in this study and other co-authors who provided valuable input and discussion during the case review process.

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