CRITICALLY ILL PATIENT WITH ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE DEVELOPING SEPSIS AND POTASSIUM DISORDER : A CASE REPORT IN SECONDARY HOSPITAL SETTING

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ABSTRACT

Sepsis and electrolyte disturbances are frequently experienced among acute exacerbation of chronic obstructive pulmonary disease (AECOPD) patients. It is characterized by organ failure brought on by unbalanced host response to infection. A 63-year-old male presented to the hospital with the complaint of breathlessness with the oxygen saturation of 76%. The leukocyte level increased to 19.000 and the Potassium level decreased to 2.88. The combination of antibiotics, Potassium supplement, oxygen support through NRM of 15 liters per minute and symptomatic treatment were administered to the patient. The NRM was then changed to HFNC due to his worsening condition and successfully decreased oxygen demand. The patient's condition has made a significant improvement after seven days of hospitalization. Patients with AECOPD are at higher risk of developing sepsis and electrolyte disturbances. Sepsis will change the biological system of the body due to different pathophysiology such as electrolyte imbalance and obviously engaged with the pathogenesis of ensuing regular intensifications. It is important for the clinicians to know the effective management for the patient such as the administration of fluid resuscitation, medicines, oxygen support so that the critical phase can be passed even at the limited facilities.

Keywords: Chronic Obstructive Pulmonary Disease; Exacerbation; Sepsis; Potassium; Critically Ill

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is characterized by persistent respiratory symptoms and airflow restriction that are brought on by abnormalities in the airways and/or alveoli, which are typically brought on by persistent exposure to noxious particles or gases.(1) According to data from World Health Organization (WHO), COPD killed 3.23 million people in 2019 and is the third highest cause of death globally.(2) Nearly 90% of COPD fatalities in people under the age of 70 arise in low and middle-income nations. Periods of acutely deteriorating respiratory symptoms in COPD, is commonly known as acute exacerbations.(3) Acute exacerbation of COPD (AECOPD) is a serious acute episode marked by a worsening of respiratory symptoms and followed by quickening loss of lung function. The quality of life is negatively impacted, and hospitalization and mortality rates are rising in consequence (4). The prevention of acute exacerbations and the improvement of COPD outcomes both rely on the early identification of the predisposing factors.(5)

Due to underlying comorbidities and reduced barrier function, sepsis have been linked to a greater risk of development in patients with AECOPD.(6,7) The life-threatening illness known as sepsis is characterized by organ failure brought on by an unbalanced host response to infection.(8,9) Increased heart rate, leukocytosis, fever, respiratory failure, and a high propensity to opportunistic infections are only a few of the clinical signs and symptoms of sepsis that

initiate with inflammation and develop to circulatory organ dysfunction.[10–12]

Electrolyte disturbances are also frequently experienced among COPD patients and are linked to poor prognosis in AECOPD.[13,14] Both patients who are hospitalized and those who attend the emergency room frequently develop serum potassium disorders. Due to decreased nutritional intake during an acute illness or increased renal loss, particularly in cases of concurrent decompensated heart failure, hypokalemia may be a side effect of AECOPD.[13] In a study conducted by Rashid et al., they discovered that patients with AECOPD had a significant prevalence of hyponatremia and hypokalemia, with 1 in 5 patients exhibit one of the electrolyte disorders.[14] The cause of hypokalemia in COPD may be long-term steroid medication, metabolic alkalosis, or respiratory acidosis.[13]

We present a case of the clinical management of a critically ill patient with acute exacerbation of chronic obstructive pulmonary disease who survived from sepsis and hypokalemia in a limited facility in a secondary hospital setting.

CASE PRESENTATION

A 63-year-old male presented to the emergency department with the complaint of shortness of breath. The breathlessness was felt during moderate activity, the patient often easily tired and fatigue on a daily basis. About 3 days before admission, the patient developed a fever with a temperature of 38 C at home and diarrhea, then he went to a secondary hospital for further management and was hospitalized for 3 days. After being discharged from the previous hospital, he began to have a shortness of breath. There were no complaints about nausea and vomiting, loss of smell or taste of the patient. Furthermore, he had no travel history to other cities or countries in the last 14 days. On initial physical examination, the patient was fully alert, the temperature was 38 C, blood pressure 149/99 mmHg, heart rate of 101 beats per minute, respiratory rate of 26 breaths per minute, and oxygen saturation of 76% on room air.

Chest examination revealed muscle retraction between the ribs and rhonchi on the both lungs through auscultation. A chest X-ray on day 1 of hospital admission demonstrated hyperinflated lungs in the both lung fields. The results of nasopharyngeal swab using RT-PCR method showed negative SARS-CoV-2. The blood test at the hospital admission showed that the leukocyte level increased to 16.740, neutrophile count of 12,95, increased C-Reactive Protein to 114 and decreased Kalium level of 2.88. Blood gas analysis using 15 liter/minute oxygen via a non-rebreathing mask (NRM) showed pH 7.31, partial pressure of carbon dioxide/pCO2 of 25/7 mmHg, partial pressure of oxygen/pO2 51.3 mmHg, bicarbonate/HCO3 level of 12.6 mmol/L, base excess/BE level of -12 mEq/L, oxygen saturation of 84.1 % and the impression of a metabolic acidosis. The patient was also consulted by the cardiologist due to his preexisting hypertensive heart disease and received therapy of Valsartan 80 mg daily. Furthermore, the patient was diagnosed with critically ill acute exacerbation of chronic obstructive pulmonary disease (AECOPD), Hypertensive Heart Disease and Hypokalemia. Comprehensive management was given to the patient, starting from the administration of weight-adjusted fluids infusion as initial resuscitation in patients with Ringer Lactate and NRM oxygenation of 15 liters per minute. He was treated with Levofloxacin 750 mg daily, Furosemide every 8 hours, Aminophylline 480 mg intravenously every 8 hours, Dexamethasone every 12 hours, Omeprazole 40 mg daily, Ceftazidime 1 gram every 12 hours, Potassium supplement of 600 mg daily, Acetylcysteine every 8 hours, Nitroglycerin 2.5 mg every 12 hours, Valsartan 80 mg daily, Salbutamol 2 mg every 8 hours, Ipratropium Bromide and Budesonide nebulizer solution every 8 hours. The patient's condition was monitored and the evaluation of laboratory examination was performed, On the third day of hospitalization,

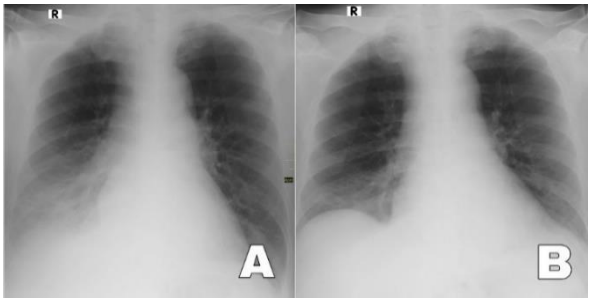

Figure 1. Chest X-ray of the patient were obtained on the first day of hospitalization (A) and the last day of hospitalization (B)

the leukocyte increased to 19.000. The antibiotics combination of Levofloxacin and Ceftazidime were still administered to the patient and the NRM was changed to high flow nasal cannula (HFNC) due to his worsening condition with flow of 40 lpm and FiO2 78%. HFNC was started as respiratory support therapy with quick decrease of breathlessness and stopped after 5 days. The blood gas analysis evaluation was performed again and showed a resolved result of pH 7.5, partial pressure of carbon dioxide/pCO2 of 26 mmHg, partial pressure of oxygen/pO2 of 104.8 mmHg, bicarbonate/HCO3 level of 20 mmol/L, base excess/BE level of -1.8 mEq/L, oxygen saturation of 98.4 %. The potassium level started to increase to 3.35. Chest X ray was done again and demonstrated normalized imaging on the last day of hospitalization. He was discharged from hospital after seven days of treatment. Spirometry examination of the patient after discharge demonstrated Forced Vital Capacity of 42%, Forced Expiratory Volume of 48% and Forced Expiratory Volume in First Second/ Forced Vital Capacity ratio of 113%. It showed a significant improvement, so the LABA treatment was ended in this patient. The patient's symptoms were resolved and made full clinical recovery.

DISCUSSION

Acute exacerbation of COPD (AECOPD) patients are more likely than the general population to develop lung infections, which increases the risk of sepsis.[15,16] Exacerbations do not necessarily coincide with acute infections, despite the fact that the respiratory tract infection (bacterial or viral) appears to be the most common cause of acute exacerbations in COPD patients.[16,17] Patients who experience COPD exacerbations usually lack the ecological indicators of infection, such elevated bacterial load and poor community diversity connected with other measures of host inflammation, like alveolar neutrophilia and elevated catecholamine concentrations. Be that as it may, basic ailments, for example, sepsis will change the biological system of the body's microbiota due to different pathophysiology such as electrolyte imbalance and is obviously engaged with the pathogenesis of ensuing regular intensifications.[6,8] Besides, sepsis-prompted organ brokenness, like renal capability impairment and muscle wasting, may likewise add to long-term disability and increase the death rate in patients with COPD.(10)

In this patient, the patient developed the suspected case of sepsis, when the leucocyte increased to 19.000 and his condition worsened. Antibiotic combinations of Levofloxacin and Ceftazidime were administered to this patient. The efficacy of Levofloxacin has been shown in a number of illnesses, including pneumonia, acute exacerbation of chronic bronchitis, skin infection, and soft tissue infection. Currently, it is believed that Levofloxacin can be used to treat bacteremia/sepsis [18]. Ceftazidime has a wide antibacterial action against most clinically relevant nosocomial gram-negative bacillary infections as well as numerous gram-positive bacteria.[18] Ceftazidime has broad antibacterial activity against many gram-positive and most clinically significant nosocomial gram-negative bacillary pathogens. Ceftazidime has been used as an empiric therapy for sepsis. Sepsis should not be disregarded while choosing a treatment plan for individuals with COPD because of how adversely it affects those patients. Statins, theophylline, macrolides, and vaccines are just a few of the treatments and methods that have been proven to reduce the risk of sepsis; they may also be used to treat COPD. Systemic corticosteroid was administered (Dexamethasone) to the patient

during the hospitalization. Systemic corticosteroids have been shown to hasten the improvement of symptoms, gas exchange, and airflow, as well as to lower the failure rate of treatment. Aminophylline, a methylxanthines drug, is also administered to the patient to improve muco-ciliary clearance, respiratory muscle activity, and central respiratory drive.[16]

The patient was nebulized with the mixture of salbutamol, ipratropium along with oxygen. The evidence shows that both inhaled beta-adrenergic agonist (Salbutamol) and anticholinergic agent (Ipratropium Bromide) can improve airflow during acute exacerbation of COPD. N-Acetylcysteine (NAC) is also given orally to the patient with the frequency of 3 times a day. According to a prior study, NAC, an antioxidant and precursor to glutathione, has been used to boost the pulmonary defense mechanism in COPD patients in order to protect the lungs against harmful chemicals. As a result, this lowers the chance of exacerbations and ameliorates symptoms. The group D therapy had been given to this patient. Group D therapy was given for patients with more severe symptoms, especially driven by greater dyspnea and/or exercise limitation. Combination of long-acting beta agonist and inhaled corticosteroid were given to reduce the exacerbation. The patient in this case still had persistent breathlessness on LABA/ICS treatment, so the long-acting muscarinic antagonist (LAMA) treatment was added to escalate to triple therapy.[4,5]

Spirometry examination of the patient demonstrated Forced Vital Capacity of 42%, Forced Expiratory Volume of 48% and Forced Expiratory Volume in First Second/ Forced Vital Capacity ratio of 113%. It showed a significant improvement, so the LABA treatment was ended in this patient. The laboratory examination of this patient revealed the decreased level of Potassium into 2.88. Potassium chloride supplement of 600 mg was given to the patient due to the correction of electrolyte imbalance. Patients with AECOPD are at a higher risk of developing hypokalemia or hyponatremia.[18–21] Patients with COPD were shown to have a high death rate for acute respiratory failure related to hypokalemia. In order to prevent negative effects, electrolyte levels in those individuals should be frequently evaluated and rectified as soon as feasible. Hypokalemia was reported in 34 (76.6%) COPD patients in the earlier study by Haroon, along with hyponatremia in 42 (70%) and mixed electrolyte abnormalities in 34 (56%) individuals.[22]

The patient was not intubated during the hospitalization period. Due to the decreased oxygen saturation in this patient, oxygen support was changed from NRM to high flow nasal cannula (HFNC). According to the previous study, HFNC does not control case mortality but considerably lowers intubation procedure and subsequent invasive mechanical ventilation. The complexity of acute lower respiratory symptoms, in which the underlying lungs do not hold typical features of acute respiratory distress syndrome, and the finding that mortality is not affected by HFNC are in line with a previous trial that showed reduced intubation hypoxemic rates in the most patients.[4]

Table 1. Laboratory Findings

Parameter	Day 1	Day 3	Day 5	Normal value
Hb (g/dL)	13.5	13.9	12.9	13.2 – 17.5
Ht (%)	41.5	44	41	40 – 52
Erythrocyte (.$10^{3/}$uL)	4.56	4.86	4.39	4.4 – 5.9
Leukocyte (.$10^{3/}$uL)	16.74*	19*	15.44*	3.8 – 10.6
Eosinophil (%)	0		0	2 – 4
Basophil (%L)	0		0	0 – 1
Band (%L)	2		1	3 – 5
Segmented (%)	77		94	50 – 70
Lymphocyte (%L)	13		1	25 – 40
Monocyte (%)	8		4	2 – 8
Thrombocyte (.$10^{3/}$uL)	450	352	332	150 – 450
MCV (fL)	91		93	80 – 100
MCH (pg)	29,5		29	26 – 34
MCHC (g/dL)	32,5		31	32 – 36
NLR	5,78		94	< 3.13
CRP	114			< 10
Sodium (mmol/L)	141.38	145.07		135 – 147
Potassium (mmol/L)	2.88*	3.35		3.5 – 5
Chloride (mmol/L)	101.32	96.82	0.95*	95 – 105
Calcium (mg/dL)	8.38	8.28		8.8 – 10.3
pH	7.31	7.50		7.33 – 7.45
PO2 (mmHg)	51.3	104.8		75 – 100
PCO2 (mmHg)	25.7	26.1		32 – 45
HCO3 (mmol/L)	12.6	20		21 – 28
BE (mmol/L)	-12	-1.8		-2 – (+3)
O2 saturation (%)	84.1	98.4		95 – 98
RT-PCR (Nasopharyngeal Swab)	Negative			Negative

Abbreviations: Hb=hemoglobin, Ht=hematocrit, MCV=mean corpuscular volume, MCH=mean corpuscular hemoglobin, MCHC=mean corpuscular hemoglobin concentration, NLR=neutrophil (NEU)-to-lymphocyte (LYM) ratio, CRP=C-Reactive Protein, PO2= partial pressure of oxygen, PCO2=partial pressure of carbon dioxide, HCO3=bicarbonate, BE=base excess, RT-PCR=real time polymerase chain reaction, g/dL=grams per deciliter, %=percent, µL=microns per liter, fL=femtoliters, pg=picograms, %L=percent liter), mg/dL=milligrams per deciliter, mmol/L=milimoles per liter.

CONCLUSIONS

Although the patient had passed the critical phase and almost failed to breathe, with prompt and appropriate patient management, the absence of comorbidities even with old age, became the key to successful treatment in this patient. The steps taken in order to pass the critical phase starting from the administration of weight-adjusted fluids infusion as initial resuscitation in patients with Ringer Lactate and NRM oxygenation of 15 liters per minute. He was treated with Levofloxacin 750 mg daily, Furosemide every 8 hours,

Aminophylline 480 mg intravenously every 8 hours, Dexamethasone every 12 hours, Omeprazole 40 mg daily, Ceftazidime 1 gram every 12 hours, Potassium supplement of 600 mg daily, Acetylcysteine every 8 hours, Nitroglycerin 2.5 mg every 12 hours, Valsartan 80 mg daily, Salbutamol 2 mg every age, 8 hours, Ipratropium Bromide and Budesonide nebulizer solution every 8 hours and oxygen support including HFNC. It is important for the clinicians to know the effective management for the patient, so that the critical phase can be passed even at the limited facilities.

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CONFLICT OF INTEREST

According to the author, this study has no conflicts of interest.

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