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Acute Exacerbation of COPD with Type II Respiratory Failure and Its Management – Case Report

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ABSTRACT

A 75 years old female presented to the emergency department with acute onset of shortness of breath on exertion with respiratory distress for two days along with disorientation and hematuria. Acute exacerbation of COPD with type-II respiratory failure was revealed. Acute exacerbation of COPD with Type II respiratory failure is a serious medical condition that requires prompt diagnosis and management. It occurs when an acute event, such as infection or environmental pollutants, causes the airways to become inflamed and narrowed resulting in increased breathlessness. Treatment typically involves oxygen therapy, bronchodilators to open up the airways, antibiotics if there is an infection present and steroids for inflammation reduction. In addition lifestyle modifications such as quitting smoking will help reduce symptoms long-term. Early recognition of this condition can be life-saving so it important that health care providers are aware of its signs and symptoms in order to provide appropriate treatment promptly.

Keywords: Obstructive pulmonary disease; Pulmonary embolism; Pneumothorax; Non-invasive ventilation

INTRODUCTION

The occurrence of a rapid worsening of respiratory symptoms and airway function in COPD patients is known as an acute exacerbation.[1] From self-limiting illnesses to periods of severe respiratory failure necessitating mechanical ventilation, these exacerbations can occur. Although viral infections and environmental stressors cannot be discounted, bacterial infections are the most frequent causes of AECOPD. Other comorbidities including heart disease and other lung disorders might cause or exacerbate AECOPD episodes (e.g. Pulmonary embolism, pneumothorax).[2] Most individuals who have it are treated with bronchodilators, antibiotics, and corticosteroids. In certain individuals, oxygen, physical therapy, mucolytics, and airway clearing devices are helpful.

When the respiratory system is unable to adequately expel carbon dioxide from the body, type-II respiratory failure, also known as hypercapnic respiratory failure, develops.[3]

CASE REPORT

On October 29, 2022, at 10:15 p.m., a 75-year-old woman arrived at the emergency room complaining of severe shortness of breath with exercise. About two days ago, symptoms including respiratory discomfort, confusion, and hematuria started to appear.

She has a considerable prior medical history for type 2 diabetes mellitus, COPD, hypertension, and ischemic heart disease. Initial physical testing uncovered:

BP -130/80 mm Hg

Pulse rate - 80 bpm

O_2 saturation - 98% on room air

During a chest exam, it was discovered that there was respiratory discomfort, wheezing, and bilateral creep. Moreover,

pH – 7.43

Pco_2 – 61.6

Po_2 – 93

HCO_3 – 39.9

To stabilize her, she was advised-

Sucrose rich diet

Inj Pan 40(Pantoprazole 40mg)-IV	Once daily before meal
Inj Zofer 4(Ondansetron 4mg) – IV	Thrice daily
Inj Pipzo 4.5(Piperacilin 4000mg + Tazobactum 500mg) - IV	Thrice daily
Inj CortS 100(Hydrocortisone 100mg) – IV	Thrice daily
Inj Lasix 20(Furosemide 20mg) – IV	Thrice daily
Nebulizer Levolin (Levosalbutamol)	6 hrly
Nebulizer Foracort (Formoterol+Budesonide)	12 hrly
Tab Montek- LC (Levocetirizine+Montelukast)	Once daily
Tab Mucinac 600(Acetylcysteine 600mg)	Once daily
Tab Concor 1.25(Bisoprolol 1.25mg)	Once daily
Tab Admenta 5(Memantine 5mg)	
Tab Aldactone 25(Spironolactone 25mg)	
Syr. Duphalac 15ml(Lactulose)	Once daily at bedtime

BiPap (iPAP/ePAP) 16/6 (mm Hg) Oxygen 4L/min

Total fluid intake 800-1000ml/day

CBC was advised to establish whether an infectious or anemic source was present.

CRP test, CPK, and CPK-MB tests were also performed.

ECG and chest X-ray were done.

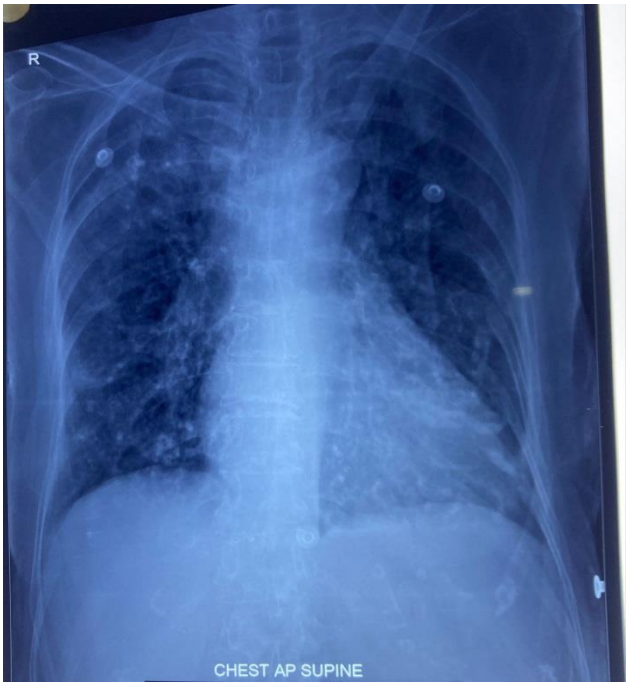


Figure: Hyperinflated lungs consistent with COPD

Intake-output chart of urine was advised to monitor.

CBC revealed - WBC 8500 cells/mcL

Hb 7.3 gm/dL

CRP revealed - 18 mg/dL

CPK revealed - 108 mcg/dL

CPK-Mb revealed – 19 IU/L

On the second day of admission,

Her vitals were found to be -

BP – 110/70 mm Hg

Pulse rate – 82bpm

O_2 saturation – 97%

Urine I/O - 600/400 ml

Her stool OBT was advised.

2 Unit PRBC transfusion(A+) was advised once per day as her Hb was much less than the normal range (11.6-15 gm/dL for female)

Physician included

P. Enema

Inj. Lasix 19 (Furosemide 19mg) Three times a day

Omnacortil (Prednisolone)

Inj. Lizomak 600 (Linezolid 600mg) – IV Twice daily

O_2 1Lt/min

Chest Physiotherapy

On the very next day, she was again examined. Her vitals revealed-

BP – 120/70mm Hg

Pulse rate – 86 bpm

O_2 saturation – 96% with BiPAP+1Lt O_2/min

Urine I/O – 1550/900ml

Her ABG assessment revealed -

pH of blood – 7.5

pO_2 – 56

pCO_2 – 48

O_2 Saturation – 85.8% on room air

HCO_3 – 44

Along with other medications and physiotherapy, the physician included

BiPap – 4 hours on / 2 hours off and overnight and the physician suggested for another opinion.

The 2nd physician diagnosed Bronchiectasis with infective exacerbation.

The patient was immediately advised for -

NIV3hrs on/2hrs off

with IPAP/EPAP – 16/6mmHg + overnight

at the rate of 3Lt O_2 per min

Glycohale (Glycopyrrolate) Twice daily

Glevo 500 (Levofloxacin 500mcg) IV Once daily

Two days later, her assessment revealed -

BP – 140/80

Pulse rate – 75 bpm

O_2 saturation – 98% (2Lt O_2 /min)

Urine I/O – 2200/2100 ml

The physician advised to follow a Pulmonologist along with physiotherapy.

On the very next day,

BP – 130/80 mmHg
Pulse rate – 72 bpm
O_2 Saturation – 94% (2 Lt O_2/min)
Urine I/O – 2000/1300 ml

She was advised to be administered -

T. Vertin 16 (Betahistine 16mg)	Thrice daily
Cap. Pan D (Domperidone + Pantoprazole)	Once daily before meal
Neb. Levolin (Levosalbutamol)	Thrice daily
Neb. Foracort (Formoterol + Budesonide)	Twice daily
Tab AB phylline N (Acebrophylline + Acetylcysteine)	Twice daily after meal

Inj. Pan was advised to be withdrawn.

BiPap was advised to be turned off and her ABG assessment was advised to be done at 6 am.

The next day, Examinations revealed -

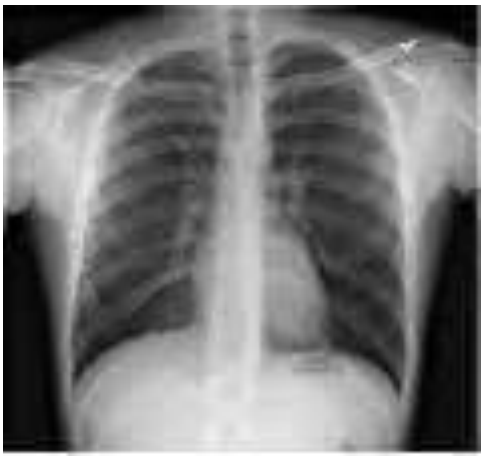
BP- 120/80 mmHg
Pulse rate – 82 bpm
O_2 Saturation – 97% (2 Lt O_2 /min)
Urine I/O – 2100/1050ml

She was advised to shift to the ward.

At 2 pm in the ward, her vitals were-

Bp – 120/80 mmHg
Pulse rate - 79 bpm
O_2 Saturation – 96% (2 Lt O_2/min)
Urine I/O – 2000/1050 ml

Clinically, the patient became well with no recurrence of hypercapnia. A further X-ray taken a few months later showed continued progress. Before her discharge, a chest X-ray is attached below.



DISCUSSION

A COPD exacerbation can present clinically in a number of different ways. Similar symptoms and signs to those at baseline may be present in patients with AECOPD, although they may be more severe.

When congestive heart failure coexists, orthopnea and paroxysmal nocturnal dyspnea, which are often not present at baseline, may become obvious. Increased expiratory wheezes might be detected during physical exams. If the airway blockage is severe, wheezing or breath sounds may become less frequent.[4]

The cough may get worse and be more laborious if you have AECOPD. Sputum's volume might rise, and the colour could shift from pale to yellow or green. Hemoptysis, which might include streaks or flecks of blood mixed with purulent sputum, is extremely prevalent during AECOPD.

There are two different forms of respiratory failure: Type I and Type II.

Type II or hypercapnic respiratory failure is defined as an increase in arterial carbon dioxide (CO2) (PaCO)>45 mmHg with a pH of 7.35 brought on by pulmonary pump failure and/or increased CO production.

Type II respiratory failure is often referred to as ventilatory failure.[6] Hypoxemia-related damage to important organs is one of its challenges.

CNS depression brought on by elevated amounts of carbon dioxide breathing acidosis

An increase in common respiratory symptoms such coughing up sputum, malaise, exhaustion, and dyspnea is seen in cases with bronchiectasis exacerbation. The treatment of all these can be done by using

Antibiotics

Corticosteroids

Physiotherapy

NIV(BiPap)

Patients with AECOPD who require assistance with sputum clearing benefit from chest physical therapy, and walking programmes can reduce arterial blood gases, lung infection, and dyspnea.

With NIV, or non-invasive ventilation, the patient's upper airway is used to deliver ventilator assistance via a mask or other similar equipment. Patients having a recent COPD exacerbation are taken into consideration.[7] Positive airway pressure, or having the pressure outside the lungs be greater than the pressure inside the lungs, is how NIV works. By forcing air down the pressure gradient and into the lungs, it lessens the respiratory effort and the labour required to breathe. Two kinds of NIV exist: positive pressure that is not intrusive i.e. NIPPV and Negative pressure ventilation i.e. NPV

A non-invasive positive pressure therapy called BiPap (Biphasic Positive Airway Pressure) is available[8]. The inspiratory positive airway pressure (iPAP) in this instance is greater than the expiratory positive pressure (ePAP).

CONCLUSION

This case included a 75-year-old female who developed bronchiectasis, type II respiratory failure, and shortness of breath as a result of an acute COPD exacerbation. It required inquiry, including a chest X-ray, management with multiple medications, and the NIV approach supplemented by physical therapy.

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DECLARATION

The patient's written informed consent was acquired before this case report and any related photographs were published.

CONFLICT OF INTEREST

The author says there are no competing interests.

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