

# CASE REPORT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND LOWER RESPIRATORY TRACT INFECTION ASSOCIATED WITH RESPIRATORY DISTRESS

Amartya De\*1, Dipan Biswas2, Anusree Basu1, Rahul Mukhopadhyay3, Shraddha Basu1

\*1 BCDA College of Pharmacy & Technology, Campus1 Hridaypur, Kolkata, West Bengal, India.

\*2 R.G.Kar Medical College & Hospital, Kolkata , West Bengal, India.

\*3 BCDA College of Pharmacy & Technology, Campus 2, Madhyamgram, Kolkata, West Bengal, India.

**Abstract** – An 83 year-old- male admitted to the emergency department with severe cough and cold and respiratory distress for 12 days. Bilateral creps and Rhonchi were observed. The patient has co-morbidities such as Type II Diabetes Mellitus and hypertension. Lower respiratory tract infection and chronic obstructive pulmonary disease were revealed.

**Keywords** – Obstructive pulmonary disease, pulmonary

embolism, pneumothorax, Lower respiratory tract infection, Bronchilitis, non-invasive ventilation.

Received: October 25th, 2023, Revised: November 2nd, 2023, Accepted: November 25th, 2023,

Licensee Abhipublications Open.

This is an open access article licensed under the terms of the Creative Commons Attribution Non-Commercial License (<u>http://www.abhipublications.org/ijpe</u>) which permits unrestricted, non-commercial use, distribution and reproduction in any medium, provided the work is properly cited.

**Corresponding Author:** \* Dr. **Amartya De, Associate Professor, BCDA College of Pharmacy and Technology, Hridaypur, Kolkata, India.** E-mail: <u>amartyap.col30@redifmail.com</u>, Phone: +91-9051931652

Introduction - Acute lower respiratory infections include

pneumonia as well as infections affecting airways such as acute bronchitis and bronchiolitis, influenza, and whooping cough. They are a leading cause of death in children and adults across the world [1].

Chronic obstructive pulmonary disease describes the phenomenon of sudden worsening in airway function and respiratory symptoms in patients with COPD [1].

Such exacerbations can range from self-limited diseases to episodes of florid respiratory failure requiring mechanical ventilation. Bacterial infections are the most common causes of COPD, though, viral infections and environmental stresses cannot be overruled. Respiratory distress and COPD episodes can be triggered or complicated by other comorbidities such as heart diseases, Diabetes, Hypertension, and other lung diseases (e.g. Pulmonary embolism, pneumothorax) [2]. Its management includes bronchodilators, antibiotics, and corticosteroids in most patients. Oxygen, Physical therapy, mucolytics, and airway clearance devices are useful in selected patients.

# Case report :

An 83-year-old male was presented to the emergency department with a severe cough and cold and 12-day-long respiratory distress.

His past medical history is significant for COPD, hypertension, and type 2 Diabetes Mellitus. Initial physical examination revealed-

BP - 130/80 mm Hg

Pulse rate - 80 bpm

 $O_2\,saturation\,$  - 98% on room air

Chest examination revealed respiratory distress, wheezing sound, and bilateral creps were noted.

Several screening tests were done immediately:

# HRCT SCAN OF THORAX revealed

1) Small soft tissue density nodular lesion with surrounding ground

glass opacity with reticulation at the apical segment of the left lower lobe-

solitary pulmonary nodule.

2) Large peripherally located ground glass opacity with reticulations

and interlobular septal thickening in the posterior segment of the right

upper lobe.

3) Linear fibrotic change with borderline traction bronchiectasis in

apical segment of the left lower lobe.

# **ECHO-CARDIOGRAPHY** revealed

- 1) Good LV Systolic Function, LVEF 60%
- 2) Grade I Diastolic Dysfunction

- 3) Concentric LVH
- 4) Mild Mitral Regurgitation
- 5) Mild Tricuspid Regurgitation
- 6) Pulmonary Arterial Systolic Pressure 28mm of Hg
- 7) IVC 12mm (more than 50% respiratory variation)

#### WHOLE ABDOMENS USG revealed

- 1) Splenomegaly
- 2) Bilateral simple renal cortical cysts
- 3) Bilateral raised renal cortical echogenicity with maintained portico

medullary differentiation.

- 4) Thickened UB wall
- 5) Significant post-void residual urine volume

#### HAEMATOLOGICAL EXAMINATION revealed

Hb	=	<b>8.2</b> g/dl
Total RBC count	=	3.25 million/cumm
Total WBC count	= <b>11,900</b> /cumm	
Neutrophil	=	85%
Lymphocyte	=	12%
Monocyte	=	2%
Eosinophil	=	1%
Basophil	=	0%
Total platelet count	=	2.35 lakhs/cumm
ESR	=	12mm at 1 <sup>st</sup> hour
PCV	=	26.7%
Mean corpuscular volume	=	82.1 Fl
Mean corpuscular haemoglobin	=	<b>25.2</b> pg
Mean corpuscular Hb conc.	=	<b>30.7</b> g/dl

International Journal Of Pharmacy and Engineering (IJPE)

RDW	=	15.7%
BIOCHEMICAL EXAMINA	TION (	OF BLOOD revealed
HbA1C	=	9.7%
Serum Urea	=	<b>52</b> mg/dl
Serum Creatinine	=	1.03mg/dl
Serum Sodium	=	<b>135.0</b> mEq/L
Serum Potassium	=	4.1mEq/L
LIPID PROFILE		
Serum Cholesterol	=	83mg/dl
Serum HDL Cholesterol	=	50mg/dl
Serum LDL Cholesterol	=	21mg/dl
Serum VLDL Cholesterol	=	12mg/dl
Serum Triglycerides	=	60mg/dl
LIVER FUNCTION TEST		
Serum Bilirubin(Total)	=	0.8mg/dl
Serum Conjugated Bilirubin	=	0.3mg/dl
Serum unconjugated bilirubin	=	0.5mg/dl
Serum total protein	=	6.4gm/dl
Serum albumin	=	3.8gm/dl
Serum globulin	=	2.6gm/dl
Serum Alb : Glb	=	1.5 : 1
Serum SGPT(ALT)	=	23U/L
Serum SGOT(AST)	=	25U/L
Serum GGT	=	15U/L
Alkaline Phosphatase	=	114U/L

#### SEROLOGICAL EXAMINATION OF BLOOD

C-REACTIVE PROTEIN	=	<b>79.43</b> mg/L
		U

## PATHOLOGICAL EXAMINATION OF URINE revealed

Color	:	Pale yellow
Transparency	:	Clear
Sediment	:	Absent
Specific Gravity	:	1.005
pH	:	6.0
Albumin	:	Absent
Sugar	:	Present
Bile salt	:	Absent
Bile pigments	:	Absent
Blood	:	Absent
Ketone Bodies	:	Absent
Pus cells	:	1-2 /hpf
Epithelial cells	:	0-1 /hpf
RBC	:	Absent
Casts	:	Not found
Crystals	:	Not found

#### MICROBIOLOGICAL CULTURE OF URINE

Culture shows no growth within 48 hrs of incubation at 37°C He was prescribed the following medicines along with a diabetic diet: Tablet Telsar20 mgOnce daily Tablet Feronia XT Once daily Tablet Ferinext Once daily Tablet Rosuvas 10 mgAt bedtime Tablet Geripod 8 mgAt bedtime

International Journal Of Pharmacy and Engineering (IJPE)

Tablet Doxolin M	Once daily
Tablet Pantodac DSR	Once daily
Tablet Istavel D	Before
	Breakfast

Tablet Dimicron XR 60 mgBeforemeal

Tablet Reclide MR 30 mgBeforedinner

On the second day of the admission, several tests were performed again:

# HAEMATOLOGICAL EXAMINATION revealed

Hb	=	<b>9.1</b> g/dl
Total RBC count	=	3.41 million/cumm
Total WBC count	=	10,800 /cumm
Neutrophil	=	<b>87</b> %
Lymphocyte	=	10%
Monocyte	=	2%
Eosinophil	=	1%
Basophil	=	0%
Total platelet count	=	2.30 lakhs/cumm
ESR	=	<b>50</b> mm at 1 <sup>st</sup> hour
PCV	=	28.5%
Mean corpuscular volume	=	83.5 fl
Mean corpuscular haemoglobin	=	<b>26.7</b> pg
Mean corpuscular Hb conc.	=	31.9 g/dl
RDW	=	16.1%

# MICROBIOLOGICAL EXAMINATION OF SPUTUM revealed

Few pus cells and gram-positive cocci in chain form were found in the smear.

On the very next day, he was examined for tuberculosis and his A.F.B stain test was negative.

He was kept under the above prescribed medications and his condition was improving day by day. One day later, he was examined again:

### HAEMATOLOGICAL examination revealed

Hb	=	<b>9.4</b> g/dl
Total RBC count	=	<b>3.67</b> million/cumm
Total WBC count	=10,300 /	cumm
Neutrophil	=	<b>78</b> %
Lymphocyte	=	<b>19</b> %
Monocyte	=	2%
Eosinophil	=	1%
Basophil	=	0%
Total platelet count	=	2.10 lakhs/cumm
ESR	=	<b>25</b> mm at 1 <sup>st</sup> hour
PCV	=	30.2%
Mean corpuscular volume	=	<b>82.2</b> fl
Mean corpuscular haemoglobin	=	<b>25.6</b> pg
Mean corpuscular Hb conc.	=	<b>31.1</b> g/dl
RDW	=	<b>15.9</b> %

## **BIOCHEMICAL EXAMINATION OF BLOOD revealed**

Serum Urea	=	45mg/dl	
Serum Creatinine	=	0.81mg/dl	
Serum Sodium	=	140.0mEq/L	
Serum Potassium	=	4.0mEq/L	

## SEROLOGICAL EXAMINATION OF BLOOD

C-REACTIVE PROTEIN = 10.9mg/L

Two days later, he was examined again before approving his discharge:

After keeping him under observation for one day, he was discharged upon showing stable parameters:

The physicians advised following medications at the time of discharge and he was asked for review after 7

days:

Nebulisation with Duolin

Nebulisation with Foracort		Twice daily
Tab Telday 20	1 tab	Once daily
Tab Ferium XT	1 tab	Once daily
Tab Ferinext 5	1 tab	Once daily

International Journal Of Pharmacy and Engineering (IJPE)

Tab Rosuvas 10	1 tab	Once daily at bedtime	
Tab Geripod 8	1 tab	Once daily at bedtime	
<b>T</b> 1 1 1 1 2			
Tab Montek-LC	1 tab	At bedtime	
Capsule Doxt SL(100)		1 cap twice daily	
Syrup Lupituss	10ml	Thrice daily	
The following were advised to do if urgency arises:			
Tab Sitaclass M (50/500)	1 tabBefore breakfast		
& Before dinner			
Injection Lantus 18 units	subcutaneously	at 8 P.M.	
Tab Pan - 40	1 tab	Before breakfast	
Tab Predmet 16	1 tab	Once a day	
		post meal	
		for 5 days	

He was also advised to examine his blood sugar level (both fasting and P.P.) and lipid profile after 7 days.

## **Discussion:** Lower respiratory tract infections(LRTIs) are among the

most common reasons for consulting in primary care and physicians usually treat LRTI empirically based on clinical assessment without microbiological testing for the causal pathogen. LRTI is a broad terminology that includes acute bronchitis, pneumonia, acute exacerbations of chronic obstructive pulmonary disease, and acute exacerbation of bronchiectasis.

LRTI is characterized in many different ways. Acute infections that affect airways include acute bronchitis, bronchiolitis, and influenza, whereas acute infections that affect the alveolar sacs can include pneumonia.

COPD is an umbrella term for a range of progressive lung diseases. Chronic bronchitis and emphysema can both result in COPD[3].

In the United States, COPD affects more than 15 million adults and many more do not know they have it. COPD is a major cause of disability and it is the fourth leading cause of death in the United States according to the Centres for Disease Control and Prevention.

Acute respiratory distress syndrome is a life-threatening lung injury that allows fluid to leak into the lungs. Breathing becomes difficult and oxygen cannot get into the body. Most people who get ARDS are already at the hospital for trauma or illness. During the manifestation of ARDS,[4] an increase in the number of breaths per minute is seen, bluish color is seen around the mouth, on the inside of the lips, and peripherals. Grunting sounds, Nose flaring, Retractions, Sweating, and Wheezing sounds are observed. The person may spontaneously lean forward while sitting to help take deeper breaths.

Basal crackles are crackles apparently originating in or near the base of the lung. Bibasal crackles, also called bilateral basal crackles, are heard at the bases of both the left and right lungs. Bilateral crackles are associated with Ronchi that resemble gurgling or bubbling sounds typically heard during both inhalation and exhalation.[5,6]

The treatment of all these can be done by using

Antibiotics

Corticosteroids

Physiotherapy

NIV(BiPap)

Chest physiotherapy as well as walking programs in patients with AECOPD benefit patients requiring assistance with sputum clearance and improve arterial blood gases, lung infection, and dyspnoea.

NIV or non-invasive ventilation refers to the provision of ventilator support through the patient's upper airway using a mask or similar device. It is considered in patients with an acute exacerbation of COPD[7]. NIV works by creating a positive airway pressure – The pressure outside the lungs being greater than the pressure inside the lungs. It causes air to be forced into the lungs i.e. down the pressure gradient, lessening the respiratory effort and reducing the work of breathing. NIV is of two types – Non-invasive positive pressure

i.e. NIPPV and Negative pressure

ventilation i.e. NPV

BiPap(Biphasic positive airway pressure) is a Non-invasive positive pressure treatment[8][9]In this case, the inspiratory positive airway pressure (iPAP) is higher than the expiratory positive pressure (ePAP).

Study has found that increasing frequency and severity of LRTIs prior to COPD diagnosis are associated with an increased rate of future exacerbations of COPD and increased risk of mortality after COPD diagnosis[9][10].

**Conclusion:** In a nutshell, this was a case of respiratory distress, LRTI and rhonchi along with bronchiectasis that manifested in a 83 year-old-male as a result of COPD. It needed investigation with an X-ray of the chest managed with several medicines and NIV techniques supported by physiotherapy and nebulizer.

**Acknowledgement:** We are grateful to Dr. DipanBiswas for giving us an opportunity to do research on this case and make a case report accordingly.

**Declaration:** Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Conflict of interest: The author declares no conflict of interest.

### **References:**

- 1. Ball P. Epidemiology and treatment of chronic bronchitis and COPD. Chest 1995; 107:45S-52S.
- Celli BR, Barnes Pj. Respiratory distress. Eur Respir J 2009;29:1228-1240
- Haddad M, Sharma S. StatPearls Publishing: Jul18,2022 Physiology, Lung.
- Papi A, Bellettato CM, Braccioni. Infections and airway inflammation in chronic obstructive pulmonary disease severe exacerbation. Am I Respir Crit Care Med 2006;173:1114-1121.
- Vallabhajosyula S, Kashani K. Acute respiratory failure in the USA, 2000-2014. Ann intensive care.
  2019 Aug 28;10(1):97
- Pingleton SK. Complications of acute respiratory failure. Med Clin North Am. 1983 May;67(3):711-20
- Nava S, Hill N. Non-invasive ventilation in acute respiratory failure. Lancet 2009;374(9685):248-9
- 8. Nagashima O, Suzuki Y, Iwase A, et al.. Acute hemorrhage in a giant bulla. *Intern Med* 2012;51:2672169
- 9.Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: 2023 report
- 10. Maltais F, Decramer M, Casaburi R, et al. An official American Thoracic Society/European Respiratory Society statement: update on limb muscle dysfunction in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2014 May 1;189(9):e15-62.