Emphysema: An Overview

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Abstract— Emphysema can be defined as loss of lung capacity, chronic enlargement of the airways to the posterior bronchioles, and destruction of alveolar walls. It can be classified under the common name of chronic obstructive pulmonary disease (COPD). The Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD) has defined COPD as "a common, preventable and incurable disease caused by persistent respiratory symptoms and airway obstruction and or alveolar abnormalities often caused by exposure to harmful substances, particles or particles". .

People with COPD having the harder problem in breathing, which can lead to shortness of breath and or fatigue. At the onset of the disease, people with COPD may feel short of breath when exercising. As the disease progresses, it may be difficult to exhale (inhale) or inhale (inhale). A person with COPD may have obstructive bronchiolitis (bron-kee-oh-lite-is), emphysema, or a combination of both. The value of these conditions varies from person to person. [1]

Asthma is another disease that causes airway obstruction, sometimes making it difficult to breathe, but asthma is not included in the definition of COPD. Some people have a combination of COPD and asthma. Chronic bronchitis is no longer considered a form of COPD, although health care professionals still use the term to describe a productive cough patient for three months in two consecutive years.

Emphysema is primarily a diagnosis of disease that affects the airways away from the final bronchiole. It is characterized by an abnormal continuous expansion of the airways of the lungs by the destruction of their walls without fibrosis and destruction of the lung parenchyma by loss of strength.

Index Terms— Chronic-bronchitis, chronic bronchitis, lungs, respire, COPD, CT, Chest X-Ray.

I. ETIOLOGY

Emphysema is caused by chronic and important exposure to harmful gases, of which smoking is the most common cause, with 80% to 90% of patients being classified as smokers, with 10-15% of smokers forming COPD. For smokers, however, the symptoms also depend on smoke exposure, years of exposure, and early lung function, which usually start after at least 20 packs of cigarette exposure a year.

Biomass oils and other pollutants, such as sulphur dioxide and particles, are known to be a major cause in developing countries, with devastating effects on women and children. A rare genetic disease inherited from autosomal, alpha 1 antitrypsin, can also lead to liver failure. However, it only accounts for 1% to 2% of COPD cases. It is a risk factor and can manifest with pan-acinar bibasilar emphysema early in life. [2]

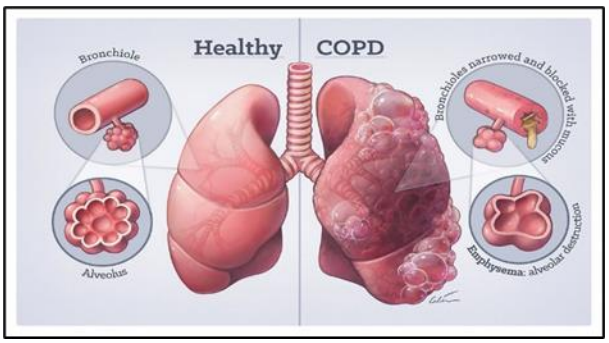
Different etiological factors are 2d-hand smoke, lung infections and allergies. Additionally, a new-born's low beginning weight makes it greater prone to developing COPD later in lifestyles. [3]

II. PATHOPHYSIOLOGY

The clinical manifestations of emphysema are caused by damage to the distal airways to the terminal bronchiole, including the respiratory bronchioles, alveolar sacs, alveolar ducts, and alveoli, collectively known as acini. There is a rare abnormal expansion of air space and the destruction of its walls due to the action of proteins. This causes alveolar and capillary surface degradation which reduces gas exchange. The portion of the grape affected determines the sub-variety. Naturally it can be categorized: [4]

a) Centrilobular (proximal acinar) is the most common type and is often associated with smoking. It can also be seen in pneumoconiosis of coal workers.
b) Panacinar is often seen in the absence of a single alpha antitrypsin.
c) Paraseptal (distal acinar) can occur alone or in combination with the previous two. When it occurs alone, the most common association is autoimmune pneumothorax in a young adult.

After prolonged exposure to toxic fumes, inflammatory cells such as macrophages, neutrophils and T lymphocytes are recruited and play a key role in developing emphysema. First, macrophages that release neutrophil chemotactic elements such as leukotriene B4 and interleukin-8 are active. Once neutrophils are detected, as well as macrophages release more proteinase and lead to secretion of mucus hyper. [5]



Elastin is an important part of the outer cell matrix needed to maintain the integrity of the lung parenchyma and narrow airways. Elastase or anti-elastase imbalances increase the tendency to lung damage, leading to an increase in airway capacity. Cathepsins and neutrophil-induced proteases (i.e. elastase and proteinase) act against elastin and eliminate tissues connected to the lung parenchyma. Cytotoxic T cells secrete TNF-α and perforins deplete epithelial cells in the alveolar wall. Cigarette smoking not only causes the release of mucus hyper and the release of neutrophil proteolytic enzymes, but also inhibits antiproteolytic enzymes and alveolar macrophages. Genetic polymorphisms play a role in the production of insufficient antiproteases in smokers. All of this contributes to the development of emphysema. The lung parenchyma produces alpha one antitrypsin (AAT) which prevents trypsinization and neutrophil elastase in the lungs. AAT deficiency can cause panacinar emphysema.

III. HISTORY

Most patients present with mild to moderate symptoms of cough and cough with or without sputum production. As the disease process progresses, shortness of breath and coughing become worse. Initially, there is a distinct dyspnea with significant physical activity, especially the function of the

arm at shoulder level or higher by advancing dyspnea with simple daily activities and even rest. Some patients may experience shortness of breath due to airway obstruction. [6]

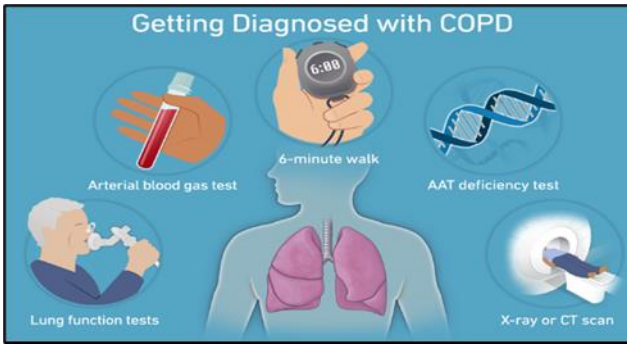
As COPD progresses, patients may lose vital weight due to systemic inflammation and increased energy exposure to respiratory function.

In addition, there are occasional recurrences of air obstruction. COPD flare-ups can indicate increased breathing, increased coughing, and increased coughing often caused by an infection or environmental factor.

Strong strokes may be common at the onset of the disease. It can be from prolonged breathing or shortness of breath during forced breathing to additional MRI showing increased elevation as airway obstruction increases. Distant breathing sounds, whirlwinds, pulmonary edema, and / or distant heart sounds are heard in auscultation.

TEST

Emphysema is a diagnosis of disease. As a result, standard laboratory and radiographic studies were not shown. [8]



Pulmonary function tests (PFTs), especially spirometry, are the cornerstone of the diagnostic room. Post bronchodilator tests can be performed on those with unusual values. COPD is reversed slightly or irreversibly with a bronchodilator with a post-bronchodilator FEV1 / FVC less than 0.07, which is diagnostic.

The GOLD rating based on the size of the air flow is as follows:

- Gentle with FEV1 greater than or equal to 80% of the forecast
- Average FEV1 less than 80% predicted
- Most with FEV1 less than 50% prediction
- Too much with the expected FEV1 below 30%

Lung volume measurements indicating air intake into emphysema indicate an increase in residual volume and total lung capacity. The distribution capacity of carbon monoxide decreases due to bone marrow destruction of the alveolus-capillary membrane.

Chest X-ray is only useful in diagnosing emphysema, but it is usually the first step in COPD, which is suspected to have other causes. Damage to the alveoli and airway obstruction causes hyperinflation of the lungs by softening of the lungs, and the heart appears longer and more congested. [9]

Arterial blood gases are rarely needed for mild to moderate COPD. It is done when oxygen saturation falls below 92% or there is a severe blockage of air into hypercapnia.

IV. TYPES OF EMPHYSEMA

1. CENTRIACINARY (CENTRILOBULAR) EMPHYSEMA

Centriacinate or centrilobular emphysema is one of the most common types. It is characterized by the initial involvement of the respiratory bronchioles, which is the central or proximal part of the acinus. This is the type of emphysema that usually coexists with chronic bronchitis and occurs predominantly in smokers and coal miners' pneumoconiosis.

2. PANACINAR (PANLOBULAR) EMPHYSEMA

Pan-lobular emphysema is the other common type. In this type, all portions of the grape are affected but not the entire lung. Panacin emphysema is most frequently associated with α1-antitrypsin deficiency in middle-aged smokers and is the one that produces the most characteristic anatomical changes of the lung in emphysema.

3. PARASEPTAL EMPHYSEMA (DISTAL ACINAR)

The type of emphysema involves the distal part of the acinus while the proximal part is normal. Paraseptal or distal acinar emphysema is located along the pleura and along the perilobular septa. The involvement is visible adjacent to the areas of fibrosis and atelectasis and affects the upper part of the lungs more severely than the lower part. This form of emphysema is rarely associated with COPD, but it is the common cause of spontaneous pneumothorax in young adults. Importantly, the subpleural portion of the lung shows air-filled cysts, 0.5–2 cm in diameter.

4. IRREGULAR EMPHYSEMA (PARACICATRICIALE)

This is the most common form of emphysema, seen around scars from any cause. The affectation is irregular both in the affected grape portion and in the lung as a whole. During life, irregular emphysema is often asymptomatic and can only be an accidental autopsy finding.

5. MIXED EMPHYSEMA (UNCLASSIFIED)

Very often, the same lung can show more than one type of emphysema. It is usually due to a more serious condition that results in the loss of a clear distinction between one type of emphysema and another. Thus,

the lungs of an elderly smoker at autopsy may show continuation of centriazine emphysema in the upper lobes, panacinare in the lower lobes, and paraseptal emphysema in the subpleural region.

RISK FACTOR

Here, the dietary autosomal recessive alpha disk is an antitrypsin deficiency, it can present with bibasilar emphysema pan caviar early in life. [10]

Clinical features / symptoms

Pink weight puffers (oxygenated and tachyphonic), right heart failure and hypercapneic respiratory failure, x-ray of a small heart with hyperinflated lungs.

Indeed, the pathogenic factor can only be detected by ice and histological examination of the entire lung phase.

Age between 40 and 60 years (tobacco related)

Exposure to toxic fumes: inhaling chemical dust or granules, cotton, etc.

PROBLEMS

Patients with emphysema often have a variety of problems, some of which are life-threatening. The following are some of the most common side effects of emphysema. [11]

Shortness of breath or failure
Pneumonia
Pneumothorax
Chronic atelectasis
Cor pulmonary
Internal emphysema
Common respiratory infections
Respiratory acidosis, hypoxia, and coma

PREVENTION

Quitting smoking
Pneumococcus and Fluenza vaccines

DIFFERENT TESTS

The disease produces unexplained symptoms, so it has a wide range of diseases. These include: [12]

Chronic obstructive pulmonary disease
Chronic bronchitis with normal spirometry
Cystic fibrosis
Bronchopulmonary mycosis
Medium ventilation
Bronchiectasis
Heart failure
Tuberculosis
Preventative bronchiolitis
Problems
High blood pressure in the lungs
Cor pulmonary
Chronic respiratory failure
Automatic pneumothorax

TREATMENT

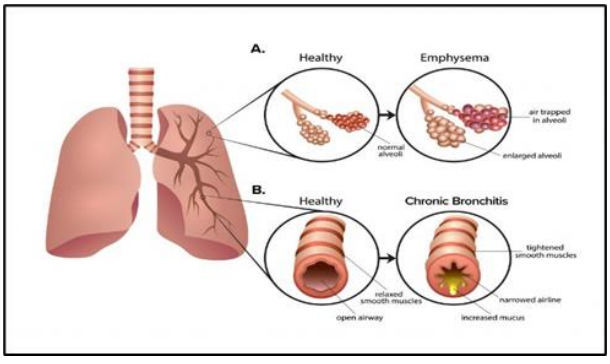
No specific treatment is known to change the disease process. However, reversing harmful substances and treating symptoms have been shown to be effective in reducing disease progression and increasing the quality of life. Depending on the symptoms and the number of increases, we can classify the disease into 4 stages of COPD GOLD and change the treatment accordingly. [16]

MEDICAL TREATMENT

Medical treatment involves the use of a bronchodilator alone or in combination with anti-inflammatory drugs (corticosteroids and four phosphodiesterase inhibitors).

BRONCHODILATOR

The main mechanisms of action can be divided into two categories: beta2 agonists and anticholinergic drugs. They are first-line drugs for COPD and are controlled by inhalation. They are known for enhancing FEV1 by altering the smooth muscle tone of the air and thus improving exercise tolerance. Bronchodilators are usually given regularly to prevent and reduce symptoms, exacerbations, and hospitalizations.



Beta-acting agonists (SABA) and active muscarinic antagonists (SAMA) were generally prescribed as a

treatment for moderate dyspnea. Long-acting beta2 agonists (LABA) and long-acting muscarinic antagonists (LAMA) are mainly used for increased or recurrent dyspnea. If symptoms persist while taking a bronchodilator, another bronchodilator should be added. [17]

Beta2 agonists cause smooth muscle relaxation in the airways. SABA, like albuterol, can be used with or without anticholinergics. SABA is central to the development of COPD. LABA includes formoterol, salmeterol, indacaterol, olodaterol, vilanterol, among others. Side effects are arrhythmias, tremors, and hypokalemia. Caution should be exercised in heart failure, as tachycardia can reduce heart failure. Anticholinergics inhibit acetylcholine induced by bronchoconstriction. SAMA includes pratropium and oxitropium. LAMA as a tiotropium can be administered once a day.

Oral phosphodiesterase-4 inhibitors such as Roflumilast work by reducing inflammation and can be added to a large airway block without improving in previous medications. Triple inhalation therapy (LABA + LAMA + ICS) has recently been approved by the FDA and taken once a day. Intravenous treatment aloha1 antitrypsin supplementation for patients with AATAD. High costs and lack of availability are the main limitation of this treatment.

SUPPORTIVE TREATMENT

Supportive care includes oxygen therapy and ventilator support, lung rehabilitation, and comfort care. [18]

Normal extra oxygen does not improve the quality of life or clinical outcomes in stable patients. Long-term continuous management is recommended, ie more than 15 hours of extra oxygen in COPD patients with PaO2 levels below 55 mmHg (or oxygen supplementation below 88%) or PaO2 below 59 mm Hg in the case of -cor pulmonary. Oxygen therapy has been shown to increase survival in these hypoxemic patients at rest. For those who have given up exercise, internal oxygen will help. The goal is to keep oxygen levels above 90%.

The main cause of hypoxemia in COPD is due to poor ventilation-perfusion (V / Q mismatch), especially in low V / Q areas. Hypoxic vasoconstriction of the pulmonary arteries aims to improve the efficiency of gas exchange. The extra oxygen can effectively reach the alveoli in the lungs, preventing vasoconstriction and thus increase absorption and improve gas exchange, leading to the development of hypoxemia.

Rehabilitation of the lungs in patients with severe symptoms and further increase reduces dyspnoea and hospitalization and is recommended for stages of GOLD B, C and D. [19]

NOVEL TREATMENT

Provision of reduced lung volume reduces inflation and improves availability and expansion

Lung transplantation where FEV1 and DLCO are less than 20%.

Additional interventions

Identification and reduction of exposure to risks. Tips to quit smoking, as it is the most important intervention that slows the progression of the disease. Reduce open fire exposure and promote better ventilation

Daily oral opioids for severe symptoms of COPD are contraindicated in treatment. Nutritional supplementation in malnourished COPD patients

Pneumococcal 23 vaccine is effective every 5 years for COPD patients over the age of 65 or for another cardiopulmonary disease and flu vaccine for all COPD patients annually

Learning levels can be reduced with advice on the proper use of metered dose inhalers (MDIs). 5. Exercise for all COPD patients

MEDICATION

It cannot be completely cured but a special aid club is provided with medication. [21]

Antibiotic treatment for bronchitis pneumonia such as bacterial infections

Bronchodilators to reduce respiratory implants are a last resort

LIFE STYLE

Exercise or yoga should be done to increase lung strength. It is definitely helpful to avoid any respiratory problems.

Quit smoking that will keep you away from all diseases like emphysema that you can control by joining a smoking time program.

Avoid contact with toxic gases, dirt, keep the home cool.

Avoid cold air or fluids that can cause bronchial disorders and difficulty breathing.

CONCLUSION

Here we can conclude that if you want to be safe from emphysema, you need to do daily exercises and avoid bad habits such as smoking and chewing tobacco.

If you become a patient with emphysema, you need to check your oxygen level regularly. You should avoid cold air. Steroids can be a better source of treatment for this condition.

Proper nutrition and staying healthy are important not only in reducing symptoms but also in maintaining good health. Lung rehabilitation programs provide supervised exercise and education for people with respiratory problems and should be part of a comprehensive treatment plan for anyone with COPD. Community support groups can provide education and opportunities for COPD patients and their carers to share their knowledge with other people and families with COPD.

All safety precautions and controls should be performed regularly, following the doctor's advice.

REFERENCES

[1] Rustagi N, Singh S, Dutt N, Kuwal A, Chaudhry K, Shekhar S, KirubakaranR. Efficacy and Safety of Stent, Valves, Vapour ablation, Coils and SealantTherapies in Advanced Emphysema: A Meta-Analysis. Turk Thorac J. 01; 20 (1):43-60, Jan 2019.

[2] Dunlap DG, Semaan R, Riley CM, Sciurba FC. Bronchoscopic deviceintervention in chronic obstructive pulmonary disease. Curr Opin PulmMed.25 (2):201-210; March 2019.

[3] Fernandez-Bussy S, Labarca G, Herth FJF. Bronchoscopic Lung VolumeReduction in Patients with Severe Emphysema. Semin Respir Crit CareMed;39(6):685-692; Dec 2018.

[4] Thomson NC. Challenges in the management of asthma associated withsmoking-induced airway diseases. Expert Opin Pharmacother; 19(14):1565-1579 Oct 2018.

[5] Zhang H, Dong L, Kang YK, Lu Y, Wei HH, Huang J, Wang X, Huang K.Epidemiology of chronic airway disease: results from a cross-sectionalsurvey in Beijing, China. J Thorac Dis; 10(11):6168-6175; Nov 2018.

[6] Harrison R, Knowles S, Doherty C. Surgical Emphysema in a PediatricTertiary Referral Center. Pediatr Emerg Care; 36(1):e21-e24 Jan 2020.

[7] Bhatt SP, Bhakta NR, Wilson CG, Cooper CB, Barjaktarevic I, Bodduluri S,Kim YI, Eberlein M, Woodruff PG, Sciurba FC, Castaldi PJ, Han MK,Dransfield MT, Nakhmani A. New Spirometry Indices for Detecting MildAirflow Obstruction. Sci Rep; 8(1):17484 Nov 2018.

[8] Storre JH, Callegari J, Magnet FS, Schwarz SB, Duiverman ML, WijkstraPJ, Windisch W. Home noninvasive ventilatory support for patients with chronic obstructive pulmonary disease: patient selection and perspectives.Int J Chron Obstruct Pulmon Dis;13:753-760; 2018.

[9] Wood-Baker RR, Gibson PG, Hannay M, Walters EH, Walters JA. Systemiccorticosteroids for acute exacerbations of chronic obstructive pulmonarydisease. Cochrane Database Syst Rev; 25 ;(1):CD001288; Jan 2005.

[10] Takei N, Suzuki M, Makita H, Konno S, Shimizu K, Kimura H, Kimura H,Nishimura M. Serum Alpha-1 Antitrypsin Levels and the Clinical Course ofChronic Obstructive Pulmonary Disease. Int J Chron Obstruct Pulmon Dis; 14:2885-2893; 2019.

[11] Hough A. Physiotherapy in respiratory and cardiac care. Hampshire: Cengage Learning EMEA, 2014.

[12] Singh D, Agusti A, Anzueto A, Barnes PJ, Bourbeau J, Celli BR, Criner GJ,Frith P, Halpin DMG, Han M, López Varela MV, Martinez F, Montes de OcaM, Papi A, Pavord ID, Roche N, Sin DD, Stockley R, Vestbo J, Wedzicha JA,Vogelmeier C. Global Strategy for the

Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease: the GOLD sciencecommittee report 2019. Eur Respir J.; 53(5); May 2019.

[13] Visca D, Aiello M, Chetta A. Cardiovascular function in pulmonary emphysema. BioMed Research International 2013.

[14] Hochhegger B, Dixon S, Screaton N, Cardinal V, Marchiori S, Binukrishnan S, Holemans J, Gosney J, McCann C, Emphysema and smoking related lung diseases. The British Institute of Radiology; 20 (4); 2014.

[15] Klein JS, Gamsu G, Webb WR, Golden JA, Muller NL. High-resolution CT diagnosis of emphysema in symptomatic patients with normal chest radiographs and isolated low diffusing capacity. Radiology; 182: 817-821; 1992.

[16] Naunheim KS, Wood DE, Mohsenifar Z, Sternberg AL, Criner GJ, DeCamp MM, Deschamps CC, Martinez FJ, Sciurba FC, Tonascia J,Fishman AP. Long-term follow-up of patients receiving lung-volume-reduction surgery versus medical therapy for severe emphysema by the national emphysema treatment trial research group. The Annals of Thoracic Surgery; 82 (2): 421-443; 2006.

[17] Campo MI, Pascau J, José Estépar RS. EMPHYSEMA QUANTIFICATIONON SIMULATED X-RAYS THROUGH DEEP LEARNING TECHNIQUES.Proc IEEE Int Symp Biomed Imaging; 2018:273-276; Apr 2018.

[18] Press VG, Cifu AS, White SR. Screening for Chronic Obstructive Pulmonary Disease. JAMA; 07; 318(17):1702-1703; Nov 2017.

[19] Sanders C, Nath PH, Bailey WC. Detection of Emphysema with computed tomography correlation with pulmonary function tests and chest radiography. Investigative Radiology; 23: 262-266; 1988.

[20] Haruna A, Muro S, Nakano Y, Ohara T, Hoshino Y, Ogawa E, Hirai T, NiimiA, Nishimura K, Chin K, Mishima M. CT scan findings of emphysemapredict mortality in COPD. Chest; 138(3):635-40; Sep 2010.

[21] Voelkel NF, Gomez-Arroyo J, Mizuno S. COPD/emphysema: The vascular story. Pulmonary Circulation; 1(3): 320–326; 2011.