ANALYSIS OF FIFTY CASES OF CHRONIC OBSTRUCTIVE LUNG DISEASE WITH CLINICAL PARAMETERS, ECG AND ECHO

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

for

M.D. Degree in General Medicine

March 2008

DEPARTMENT OF GENERAL MEDICINE
COIMBATORE MEDICAL COLLEGE & HOSPITAL
COIMBATORE

CERTIFICATE

This is to certify that the Dissertation entitled **" Analysis of fifty cases of Chronic obstructive lung disease with clinical parameters , ECG, and ECHO"** herewith submitted by **Dr. L .Saravanan** , post graduate in General Medicine, oimbatore medical college to the Tamilnadu Dr . M. G.R University is a record of a bonafide research work carried out by him under my guidance nd supervision from Jan 2006 to Jan 2007.

Professor Dr .Ramasamy M.D **Professor.Dr .Umakanthan M.D**

Prof and unit chief Head of Department of Medicine

DECLARATION

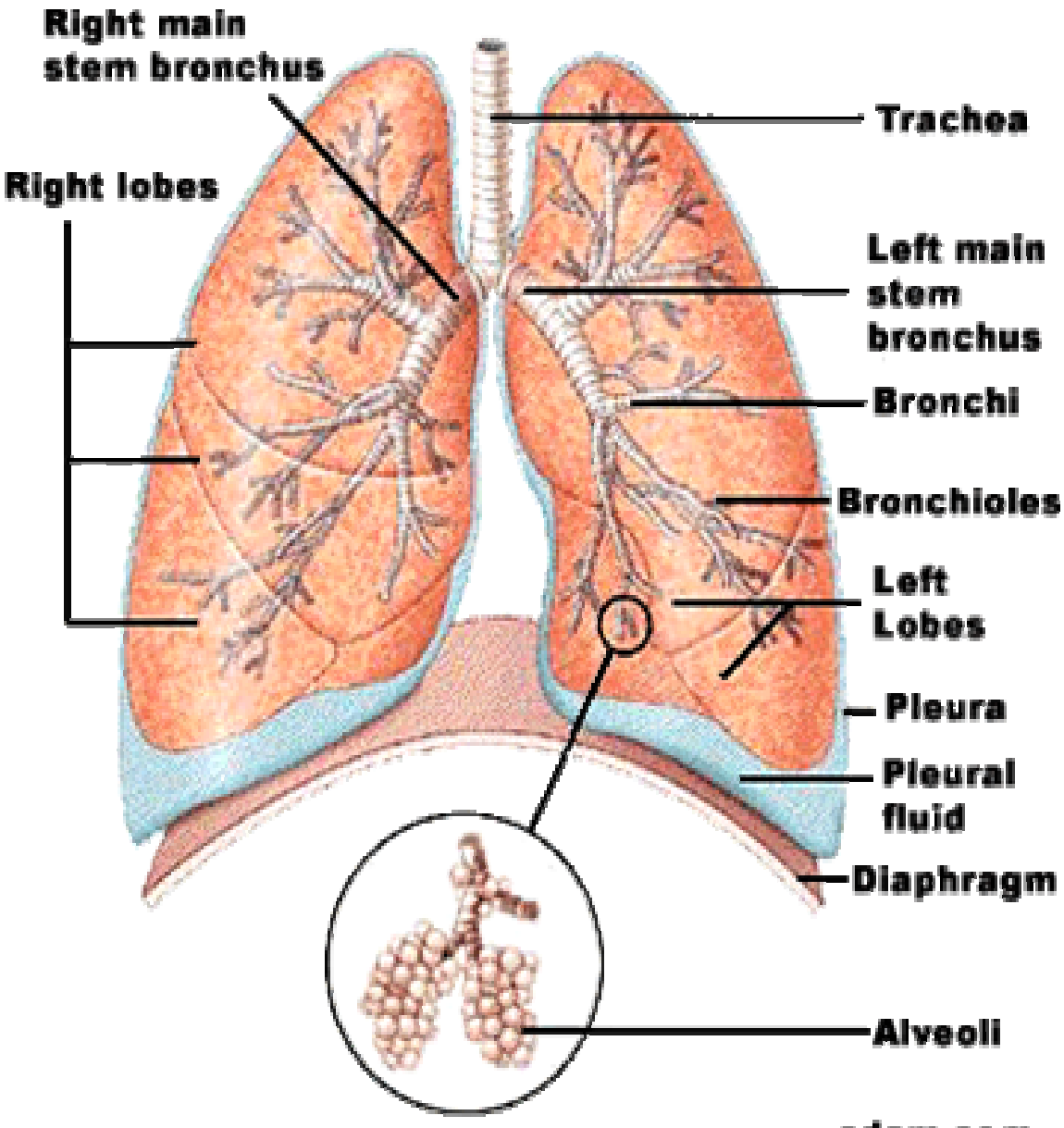
I solemnly declare that the Dissertation titled **" Analysis Of fifty cases Of Chronic obstructive lung disease with clinical parameters , ECG, and ECHO" ,** was done by me at Coimbatore Medical College & Hospital during the period from Jan 2006 to Jun 2007 under the guidance and supervision of Prof .Dr .k.Umakanthan and Prof . Dr. Ramasamy.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical university towards the partial fulfillment of the Requirement for the award of M.D. Degree[Branch 1] in General Medicine.

Place : Coimbatore **Dr.L. Saravanan**

Date :

LUNGS


Right main stem bronchus
Right lobes
Trachea
Left main stem bronchus
Bronchi
Bronchioles
Left Lobes
Pleura
Pleural fluid
Diaphragm
Alveoli
adam.com

ACKNOWLEGEMENT

I wish to express my sincere thanks to our respected dean Dr.HEMALATHA for having allowed me to conduct his study

I owe a great debt of gratitude to our HOD Dr.UMAKANTHAN M.D for his excellent guidance.

I wish to express my gratitude to my unit chief Dr.RAMASAMY M.D without whose advice this work would not have been possible

My sincere thanks to Dr. CHITRAKUMAR thoracic medicine chief for his guidance

MY sincere thanks to the cardiologist Dr. BALASUBRAMANIUM for his guidance

Last but not the least I express my gratitude to professors and asst professors DR . S. USHA. M.D, Dr. RAVIKUMAR M .D and DR .MANSUR M.D for their help

CONTENTS

INTRODUCTION

DEFINITION

The American Thoracic Society defines COPD as a disorder characterized by abnormal tests of expiratory flow [on a structural or functional basis] tha do not change markedly over several months of observation. The diagnosis usually refers to the presence of chronic obstructive bronchitis with varying degrees of emphysema and bronchospasm[asthmatic bronchospasm] .

The American Thoracic Society , defines chronic obstructive pulmonary Disease [COPD] as a disease state characterized by the presence of airwayObstruction due to chronic bronchitis or emphysema ; the airflow obstruction is

Generally progressive ,may be accompanied by airway hypersensitivity and
may be partially reversible . Diagnostic of this non specific entity are chronic productive cough, breathlessness on exertion, physiological evidence of airflow

Limitation[reduced FEV1] ,and poor reversibility[e.g , response to bronchodilator]

EMPHYSEMA

This entity is defined in anatomic terms -- abnormal permanentEnlargement of the gas –exchanging units of the lungs[acini] in associationThe predominant physiologic consequence of these anatomic abnormalitiess,a decrease in the elastic recoil of the lungs – which inturn causesOutward displacement of the chest wall and flattening of the diaphragm. Hyperinflation of the lungs , and increased resistance to airflow .which inurn Increases the work ok breathing although imbalances in ventilation Perfusion are seldom marked in as those in chronic bronchitis, derangement sufficient to cause arterial hypoxemia are common .

CHRONIC OBSTRUCTIVE

Lung disease patients are divided into chronic bronchitisEmphysematous patients . Typically they have reduced FEV1And reversibility less than 15 percent while bronchial asthma patient have more 15 percent reversibility .Then analysis of symptoms and signs ofrpulmonale ,pulmonary hypertension was undertaken

Then ECG , And ECHO CARDIOGRAM , X RAY CHEST PA view, lateral view were taken . And the ECG is looked for any evidence of pulmonary hypertension , right axis deviation , p wave morphology, RBBB, P wave amplitude in 2, 3, avF . nd the echocardiogram was taken to look for any evidence of pulmonary hypertension ,and tricuspid regurgitation. X-RAY chest PA view , and lateral were views taken to look for the presence Right heart hypertrophy , or small .pendulous heart of Emphysema

LUNG FUNCTION

GOLD has classified COPD as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases" 18. This definition has also been adopted in the new ATS/ERS guidelines, with the important observation that COPD is both preventable and treatable 4. Impaired lung function is of central importance in the diagnosis of COPD. Airflow limitation is the slowing of expiratory airflow as measured by spirometry, with a persistently low forced expiratory volume in one second (FEV1) and a low FEV1/forced vital capacity (FVC) ratio despite treatment. The current GOLD and ATS/ERS definition for airflow limitation is an FEV1/FVC ratio of <70% 4, 18. Although this "fixed" ratio is easy to remember and simple, there is some concern that it may underestimate COPD in younger populations, overestimate it in older ones, and misclassify other patients 19, 20. Declining lung function over time is an important component in understanding the natural history of COPD. The concept that different populations (i.e. susceptible smokers, nonsusceptible smokers, nonsmokers) have different trends in their lung function decline was developed by Peto et al. 21 and expanded by Burrows et al. 22. Interventions, such as smoking cessation, have been shown to alter this trend in populations, although individual patients may have a great deal of variability in their lung function decline over time 23.

EPIDEMIOLOGY

Prevalent COPD in the WORLD is influenced predominantly by smoking and age. Figure 1 shows age-specific GOLD categories of COPD stratified by smoking status, and shows that a high proportion of people aged 65 yrs have evidence of COPD. Impaired lung function is an excellent predictor of both morbidity and mortality, including the development of lung cancer 25, functional impairments 26, elevated C-reactive protein levels 27, osteoporosis 28 and death 29. However, people with similar levels of impaired lung function may have markedly different outcomes. In an analysis of participants in the first National Health and Nutrition Examination Survey (NHANES) and follow-up, current and former smokers with GOLD stage 3 or 4 COPD had a significantly increased mortality risk (compared with participants with no lung disease), whereas never-smokers with GOLD stage 3 or 4 COPD did not have an increased mortality risk 29. The NHANES findings regarding lack of increase in mortality in nonsmokers with more severe disease has not been replicated in other studies. Celli et al. 30 demonstrated that an index that includes body mass, dyspnoea and the 6-min walk, in addition to lung function, is much better at predicting mortality than lung function alone.

AIM OF STUDY

The study selects patients with cough , dyspnoea ,wheezOn the basis of pulmonary function test and reversibility and the presenting signs and symptoms are analysed with ECG ,ECHO TO FIND

1. **To know the cardiovascular complications of COPD like pulmonary hypertension**
2. **ECG changes of COPD , 2 ,3 , AVF P WAVE amplitude Morphology , RBBB, RVH , OR PHT**
3. **Concurrent coronary artery disease patients have increased incidence of right heart failure**
4. **to confirm with echocardiogram the presence of pulmonary hypertension and tricuspid regurgitation and right side failure and analyse the incidence of right heart failure and pulmonary hypertension**

HISTORICAL OVERVIEW

Laennec divided bronchitis into two humid [copious expectorationAnd dry[scarcely any expectorationRecognition of chronic bronchitis as a potentially grave illness rather thanAs a trivial but nondisabling disease had to await the LONDON FOG 1952This fog brought about by bad weather and air pollutants carried with it a surge in morbidity and mortality .Epidemiologists in the united states ,netherlands , elsewhere began to take serious stock of the prevalence of chronic bronchitis and emphysema and to wonder why the diagnosis of emphysema rarely appeared on the death certificates in Great Britain andwhy the diagnosis of chronic bronchitis never appeared inthe United states

REVIEW OF LITERATURE

DEFINITIONS

Emphysema

This entity is defined in anatomic terms -- abnormal permanent Enlargement of the gas –exchanging units of the lungs[acini] in association With estruction of he alveolar walls and without obvious fibrosis The predominant physiologic consequence of these anatomic abnormalities. Is a decrease in the elastic recoil of the lungs – which in turn causes Outward displacement of the chest wall and flattening of the diaphragm Hyperinflation of the lungs , and increased resistance to airflow .which inurn Increases the work ok breathing . although imbalances in ventilation Perfusion are seldom marked in as those in chronic bronchitis ,derangement sufficient to cause arterial hypoxemia are common .

CHRONIC BRONCHITIS

As has been pointed out chronic bronchitis is not a single clinicalEntity , and it is the airflow limitation that it produces that leads toMorbidiy and mortaliy . this limitation has been identified from a Decrease in FEV1

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The American Thoracic Society , defines chronic obstructive pulmonaryDisease [COPD] as a disease state characterized by the presence of airwayObstruction due to chronic bronchitis or emphysema ; the airflow obstruction is Generally progressive ,may be accompanied by airway hypersensitivity and may be partially reversible . Diagnostic of this non specific entity are chronic productive cough, breathlessness on exertion, physiological evidence of airflow Limitation[reduced FEV1] ,and poor reversibility[e.g , response to bronchodilator

Asthma

As a rule clinicians believe that they can identify most patients with atopic or extrinsic asthma that begins in childhood and thosewith intrinsic asthma of middle age .However ,uncertaintycreeps in when wheezing accompanies limitation to airflow insome patients with an acute exacerbation of bronchitis in thecourse of COPD .The key elements in the differential diagnosisare the reversibility in asthma to the obstruction to airflow [either by treatment or spontaneously] and hyper responsivenessof the airways .The diverse cause of asthma are reflected in avariety of qualifiers , such as atopic occupational,cardiac, and cold induced. It is the hyper responsiveness Of asthma that is responsible for the widespread but reversible , narrowing of airways In asthma , the larger bronchi are predominantly affected, goblet cellsUndergo hyperplasia, bronchial glands hypertrophy, thebasement membrane thickens , airway smooth muscle hypertrophies, and the luminal walls are invaded by eosinophils .f and of shed necrotic bronchiolar epithelium. The inflammatory changes coupled with submucosal edema , contribute to the airway obstruction.

Bronchiectasis

Localizedbronchiectasis is ofen present in patients COPD . As a rule , however bronchiectasis is characerised by Dilatation of airways rather than by narrowing.it does not contribute to obstruction of air ways in cysic fibrosisbronchiecatsis is a common finding .proximal airwaysdilated distal airways are obstructed by mucoprulentexudates and narrowed by obliterated airways.

Bronchilolitis obliterans (and BOOP)

The lesions affected the terminal branches of Branchial tree where bronchi became devoid of their cartilage in their wall caused by plugging of the terminal bronchioles ,fromwithin by organizing exudates , by constriction of bronchioles dueto inflammation and fibrosis, broncho alveolar process .causes are connective tissue disorders , toxic inhalants ,and cigarette smokingimmulogic disorders ,infections in immunocompromised individualsafter allogenic bone transplantation as graft versus host reaction cytomegalo virus , adeno virus infection ,the hall mark is presence of ranulation tissue in he airways

General appearance	Mesomorphic overweight dusky Suffused conjuctiva warm extremities	Thin ,emaciated ,pursed lip breathing,anxious,prominent Use of accessory muscles ,normal or cool extremities
Age		
Onset	40--55	50---75
	cough	dyspnea
Cyanosis		
	marked	slight o none
Cough		
	more evident than dyspnea	less evident than dyspnea
Sputum		
	copious	scanty
Uri		
	common	occasional
Breath sounds		
	moderately diminished	markedly diminished
Corpulmonale		
	common	terminally
Radiograph		
	normal diaphragm cardiomegaly ,lungs normal with increased bronchovascular markings	low ,flat diaphragm ,small pendulous heart,areas of increased radiolucency
course		
	ambulatory but constantly on verge of right sided heart failure and coma	incapacitating breathlessness punctuated by life threatening bouts of upper respiratory infections,prolonged course culminating in right sided failure and coma

FEV1	REDUCED	REDUCED
FRC	Mildly increased	Markedly increased
TLC	Normal or slightly increased	Considerably increased
RV	Normal or slightly increased	Considerably increased
LUNG COMPLIANCE	Normal or low	Normal or low
Recoil pressure	Normal or high	Low
MVV	Moderately decreased	Markedly decreased
Airway resistance	Increased	Normal or slightly increased
Dco2	Normal or low	Low
Arterial po2	Marked reduction	Slight to moderate
Arterial hypercapnia	Chronic	Only during acute respiratory infection
Hematocrit	High --70	Normal 55
Pulmonary artery pressure	Increased	Normal or slightly increased

Proposed risk factors for COPD

Risk factor	*comment*
Increasing age	ventilatory impairment primarily reflects cumulative life time smoking
gender	males more risk
smoking	relation to cigarettes smoking per day cumulative pack years
environmental	more in urban
occupation	more in coal ,gold miners , farmers, cement cotton workers
socioeconomic status	common in low socio
diet	high fish may reduce risk
genetic	alpha 1 anti trypsin deficiency
birthweight	low birth weight lowFEV1 AND high COPD later in life
recurrent broncho pulmonary infections	short term decline in lung function
Allergy and airway hyper responsiveness	increased eosinophils subgroup of smokers

Proteinase inhibitors present in the Lung Parenchyma

Proteinase inbitor	Cell of origin	proteinase inhibited
Alpha 1 AT	HEPATOCYTE	Serine proteinase
SLPI	Type 2 pneumocytes	Serine proteinase
Elafin	Large airway epithelial cells	Serine proteinase
Aipha2 macroglobulin	Hepatocytes,lung fibroblasts	Serine proteinase
TIMP-1	Macrophages, lung resident cells	MMPS
Cystatin C	Bronchial epithelial cells	cysteine proteinase

INDICATIONS FOR HOSPITALISATION OF COPD PATIENTSAcute exacerbation [increased dyspnea or sputum production]inadequate response to op management

1. Inability to ambulate
2. Inability to eat or sleep de to dyspnea
3. Inadequate home care resources
4. Serious co morbid conditions
5. Prolonged progressive symptoms
6. Altered mentation
7. Worsening hypoxemia
8. Worsening hypercapnia
9. New or worsening cor plmonale
10. Surgical or diagnostic procedure

Algorithm for the management of COPD

Ipratropim [via MDI with spacer
3, 6 pffs ,4 times a day

Suboptimal
response

Add b2 agonist 2-6 puffs every 3-6
hours as needed

Suboptimal
response

Add long acting theophyline
200 – 400 mg at bed time serum
level 8—12 micro gram/ml

Continued
symptoms on
maximal
bronchodilators

Trial of prednisone

40 mg /day for 14 days

<15
improvement
inFEV1discontue
prednisone

>15
improvement
inFEV1 low dose
prednisone

BETA ADRENERGICAGONISTS

Beta adrenergic agonists have been the mainstay of treatment of obstructive lung disease. These agents increase cyclic adenosine monophosphate formation which alters intracellular calcium homeostasisand modifies vascular tone .The drugs are best taken on as needed basis, hence they may be used as primary therapy ,in patients who have dyspnea intermittently. They may be added to the regular doses of ipratropium when patients have daily symptoms .Excessive use of beta agonist may result in tachyphylaxis via a down regulation of receptors. The side effects of beta agonists are tachycardia ,dysrhythmias,exacerbation of myocardial ischemia, and hypo or hypotension. Nervous system side effects include tremor ,agitation, and insomnia. Fortunately tolerance develops to most of the side effects. Hypokalemia has been noted in patients receiving large dose sisoproterenol, isoetharine ,meaproterenol, terbutaline,albuterol,bitolterol pirbuteral ,salmeterol are some of the beta adrenergic agonists

CORTICOSTEROIDS

Based on the common pathologic finding of airway inflammation in COPD , use of corticosteroids in preventing decline in lung function has been investigated. In one retrospective study , daily oral doses of 10 mg of prednisone produced a reduction in the rate of decline ofFEV1 over 14 – 20 years in patients with COPD . More recently ,results. From a ongoing study indicate addition of inhalational steroid to a bronchodilator regimen may delay the expected delay in FEV1 in patients with COPD .In this study which includes patients with reversible airway obstruction , long term inhaled corticosteroids appear to be most effective in subjects with the greatest variability of air flow. Furthermore , although the decline in FEV1 continued over time , airflow started from a new higher level hence the anti inflammatory effects of corticosteroids mayaffect the long term decline in FEV, in COPD.

Anti-cholinergic Agents

Acetylcholine is a chemical released by nerves that attaches to receptors on the muscles surrounding the airway causing the muscles to contract and the airways to narrow. Anti-cholinergic drugs such as ipratropium bromide (Atrovent) dilate airways by blocking the receptors for acetylcholine on the muscles of the airways and preventing them from narrowing. Ipratropium bromide (Atrovent) usually is administered via a MDI. In patients with COPD, ipratropium has been shown to alleviate dyspnea, improve exercise tolerance and improve FEV1. Ipratropium has a slower onset of action but longer duration of action than the shorter-acting beta-2 agonists. Ipratropium usually is well tolerated with minimal side effects even when used in higher doses. Tiotropium (SPIRIVA) is a long acting and more powerful version of Ipratropium and has been shown to be more effective. In comparing ipratropium with beta-2 agonists in the treatment of patients with COPD, studies suggest that ipratropium may be more effective in dilating airways and improving symptoms with fewer side effects. Ipratropium is especially suitable for use by elderly patients who may have difficulty with fast heart rate and tremor from the beta-2 agonists. In patients who respond poorly to either beta-2 agonists or ipratropium alone, a combination of the two drugs sometimes results in a better response than to either drug alone without additional side effects.

METHYLXANTHINES

Theophylline (Theo-Dur, Theolair, Slo-Bid, Uniphyl, Theo-24) and aminophylline are examples of methylxanthines. Methylxanthines are administered orally or intravenously. Long acting theophylline preparations can be given orally once or twice a day. Theophylline, like a beta agonist, relaxes the muscles surrounding the airways but also prevents mast cells around the airways from releasing bronchoconstricting chemicals such as histamine. Theophylline also can act as a mild diuretic and increase urination. Theophylline also may increase the force of contraction of the heart and lower pressure in the pulmonary arteries. Thus, theophylline can help patients with COPD who have heart failure and pulmonary hypertension. Patients who have difficulty using inhaled bronchodilators but no difficulty taking oral medications find theophylline particularly useful. The disadvantage of methylxanthines is their side effects. Dosage and blood levels of theophylline or aminophylline have to be closely monitored. Excessively high levels in the blood can lead to nausea, vomiting, heart rhythm problems, and even seizures. In patients with heart failure or cirrhosis, dosages of methylxanthines are lowered to avoid high blood levels. Interactions with other medications, such as cimetidine (Tagamet), calcium channel blockers (Procardia), quinolones (Cipro), and allopurinol (Zyloprim) also can alter blood levels of methylxanthines. Interactions Aminoglutethimide, barbiturates, carbamazepine, ketoconazole, loop diuretics, charcoal, hydantoins, phenobarbital, phenytoin, rifampin, isoniazid, and sympathomimetics may decrease effects; effects may increase with allopurinol, beta-blockers, ciprofloxacin,corticosteroids, disulfiram, quinolones, thyroid hormones, ephedrine, carbamazepine, cimetidine, erythromycin, macrolides, propranolol, and interferon

Future Directions in COPD

As opposed to bronchial asthma, which has been well researched in the last 20 years, OPD has not been fully investigated. There is significant evidence that COPD is an inflammatory process just as is bronchial asthma, however, it seems that there are different patterns of lung inflammation in these patients. The mechanisms of baseline inflammation in COPD and inflammation during exacerbation of the disease need to be investigated and better understood. There is minimal or no information on the molecular mechanisms of inflammation in stable COPD patients. This latter issue becomes important particularly in the area of treatments. Currently, there are numerous clinical trials looking to intervene at the various inflammatory pathways. A newer class of medications that work to reduce this inflammation is being developed.They are referred to as PDE4 inhibitors. Drugs under investigation include rolipram,piclamilast, cilomilast, and Roflumilast. These inhibitors reduce the number and the activity of the different types of inflammatory cells and inflammatory substances seen in COPD

FURTHER INPATIENT CARE:

In cases in extremis, CPAP or BiPAP may be attempted prior to intubation. This can be started in the ED and continued for several hours in the hospital. Usual recommended settings are an inspiratory positive airway pressure (IPAP) of 10 cm H2O and an expiratory positive airway pressure (EPAP) of 2 cm H2O, with further adjustments based on the individual. This is contingent on the patient's ability to withstand the mask. This treatment is not a substitute for intubation; rather, it is a means of trying to avoid intubation. Heliox is an additional strategy that can be attempted prior to intubation. Whether Heliox or CPAP is used will depend on the individual patient and local hospital availability. Again, like several other therapies mentioned in this chapter, study results both for and against Heliox have been published. The current summation of that literature indicates that Heliox actually may decrease the work of breathing while the patient is breathing the mixture, but its effects are not long lasting once it is removed. The proper mixture of the gases and the ability to deliver enough oxygen to the patient also are issues. Inhaled nitric oxide has been suggested, but at this point does not seem to have a role in acute treatment. Lung volume reduction surgery has also been touted as effective, but most recent studies demonstrate varying levels of success.

FURTHER OUTPATIENT CARE:

Disposition from the ED depends on the clinical picture for each patient more than any single laboratory value or test. In general, the longer the exacerbation, the more airway edema and debris are present, making resolution in the ED increasingly more difficult. Patients who state that they "feel back to normal" and have no overt reason for admission can reasonably be discharged home with follow-up arrangements. The corollary to this is that patients who state they "do not feel comfortable," regardless of the numbers, are the best predictors of outcome and probably should be admitted. Data on risk factors for relapse and need for admission are limited at present. For patients who are sent home, nearly all should receive a short steroid burst and an increase in the frequency of inhaler therapy. Close follow-up should be arranged with the patient's regular care provider. Other therapies should be considered on a case-by-case basis. Patients with severe or unstable disease should be seen monthly. When their condition is stable, patients may be seen biannually. Check theophyl line level with each dose adjustment, then every 6-12 months. For patients on home oxygen, check ABGs yearly or with any change in condition. Monitor oxygen saturation more frequently than ABGs.

COMPLICATIONS:

Some complications that must be anticipated in COPD treatment include the following: Incidence of pneumothorax due to bleb formation is relatively high; consider pneumothorax in all patients with COPD who have increased shortness of breath. In patients who require long-term steroid use, the possibility of adrenal crisis is very real; at a minimum, patients with steroid-dependent COPD should receive stress dosing in the event of an exacerbation or any other stressor.

- ✓ Infection (common)
- ✓ Cor pulmonale
- ✓ Secondary polycythemia
- ✓ Bullous lung disease
- ✓ Acute or chronic respiratory failure
- ✓ Pulmonary hypertension
- ✓ Malnutrition

Prognosis:

Patient's age and postbronchodilator FEV1 are the most important predictors of prognosis. Young age and FEV1 greater than 50% of predicted are associated with a good prognosis. Older patients and those with more severe lung disease do worse.Supplemental oxygen (when indicated) has been shown to increase survival rates. Smoking cessation improves prognosis. Cor pulmonale, hypercapnia, tachycardia, and malnutrition indicate a poor prognosis

	Hospital Admission	inpatient bed days	general practice
Chronic branchiis	100	1500	4400
Emphysema	240	3300	2700
Asthma	410	1800	11900
Total	750	6600	19000

Lab Studies:

Arterial blood gas

Arterial blood gas (ABG) analysis provides the best clues as to acuteness and severity.

In general, renal compensation occurs even in chronic CO_2 retainers (ie, bronchitics); thus, pH usually is near normal.

Generally, consider any pH below 7.3 a sign of acute respiratory compromise.

Serum chemistry

These patients tend to retain sodium.

Diuretics, beta-adrenergic agonists, and theophylline act to lower potassium levels; thus, serum potassium should be monitored carefully.

Beta-adrenergic agonists also increase renal excretion of serum calcium and magnesium, which may be important in the presence of hypokalemia.

CBC - Polycythemia

Imaging Studies:

Chest x-ray

Chronic bronchitis is associated with increased bronchovascular markings and cardiomegaly.

Emphysema is associated with a small heart, hyperinflation, flat hemidiaphragms, and possible bullous changes.

Other Tests:

Pulse oximetry

Pulse oximetry does not offer as much information as ABG.

When combined with clinical observation, this test can be a powerful tool for instant

feedback on the patient's status.

Electrocardiogram

The presence of underlying cardiac disease is highly likely.

Establish that hypoxia is not resulting in ischemia.

Establish that the underlying cause of respiratory difficulty is not cardiac in nature.

Pulmonary function tests

Decreased forced expiratory volume in 1 second (FEV1) with concomitant reduction in FEV1/forced vital capacity (FVC) ratio

Poor/absent reversibility with bronchodilators

FVC normal or reduced

Normal or increased total lung capacity (TLC)

Increased residual volume (RV)

Normal or reduced diffusing capacity

Pulmonary hypertension , cardiac function and fluid balance

Pulmonary hypertension develops late in he course of COPD with the development of hypoxemia [pa o2 < 8 kpa ,60 mm Hg] and usually hypercapnia.

It is he most common cardiovascular complication of COPD, and is Associated with the development of right ventricular failure

Pulmonary circulation

Changes occurs characteristically in he peripheral arteries in the pulmonary circulation of lungs.Among the earliest changes in the vasculature as the airflow obstruction worsens is he thickening of the intima of the small pulmonary arteries.Medial hypertrophy then develops in he muscular arteries. Peripheral airway inflammation is associated with Pulmonary arterial thrombosis.

There is increasing evidence to suggest that endothelial dysfunction
Is an underlying factor.

Thus the putative role of nitric oxide in preventing an excessive rise in pulmonary vascular tone as a result of hypoxemia may be lost.

Factors contributing to the development of

pulmonary hypertension in COPD

Destruction of the pulmonary vascular bed

Abnormal blood gas tensions

Abnormal pulmonary mechanics

Increased cardiac output

Blood volume changes

Increased blood velocity

Endothelial abnormalities

Haemo dynamics and blood gases in74 patients with COPD

	COPD		NORMAL	
	Mean	**Range**	**Mean**	**Range**
Pao2[mmHg]	43	23—67	91	75—105
Paco2	51	33---68	38	32---- 43
Cardiac output	3.8	2.3—5.8	3.6	2.6----4.5
Right arterial pressure	3	0--- 21	5	2----- 9
Mean pulmonary arterial pressure	35	15---78	13	8----20
Pulmonary vascular Resistance[dyens/s/cm2]	660	231—1377	58	40-----200
Right ventricular stroke work Index	16	5---29	6	3------18

Clinical assessment of pulmonary haemodynamics

The presence of pulmonary hypertension produces loud p2,and a systolic parasternal heave indicates right ventricular hypertrophy

However these clinical signs are often difficult to detect in patients with COPD because of over inflation.

Extra heart sounds like tricuspid regurgitation murmurs best heard on

Inspiration may be obscured by over inflation.

The jugular venous pulse is often difficult to detect because of large swings in Intra thoracic pressure Peripheral edema is present in PHT of COPD

The presence of pulmonary hypertension can be assumed on a plain chest Radiography if the right main pulmonary artery has a width of more than 16 mm

Right ventricular hypertrophy is detected in lateral view by encroachment of retrosternal space

ECG CRITERIA right ventricular hypertrophy is detected by r wave height
More than 5mm or r/s >50%

Detectable tricuspid regurgitation by echo Doppler is probably the best Technique to measure pulmonary arterial pressure non-invasively.

Materials and Methods

Selection criteria*

Step 1 The patients with cough ,sputum production dyspnea [wheeze] are chosen and sputum AFB negative confirmed.

Step 2

Pulmonary function test was done to pick up patients With reduced FEV< 80 % were chosen

Step 3

They are nebulised with salbutamol bronchodilator And PFT was done to select patients with < 15% reversibility

Step 4

They were clinically examined for over inflation,ascites, edema, parasternal heave, JVP, loud P2 , tricuspid murmur

Step 5

X ray chest PA view , lateral view taken to rule out the right pulmonary artery dilatation > 16mm [characteristic of pulmonary arterial hypertension] lateral view to rule out retrosternal space obliteration[RVH]

Step 6

ECG was taken in patients to look for p wave Morphology , amplitude,2,+3 +,avF P amplitude > 9mm,p axis, R wave > 5mm , r/s>50% ,RBBB ,

Step 7

Echocardiogram was done for the chosen patients chosen Looked for pulmonary arterial hypertension, and tricuspid regurgitation[as it is the non invasive measure of pulmonary arterial pressure ,surrogate , for measuring PAP]

The patients were selected from the coimbatore medical College hospital .

They are interviewed for the symptoms like cough, dyspnea, wheeze.

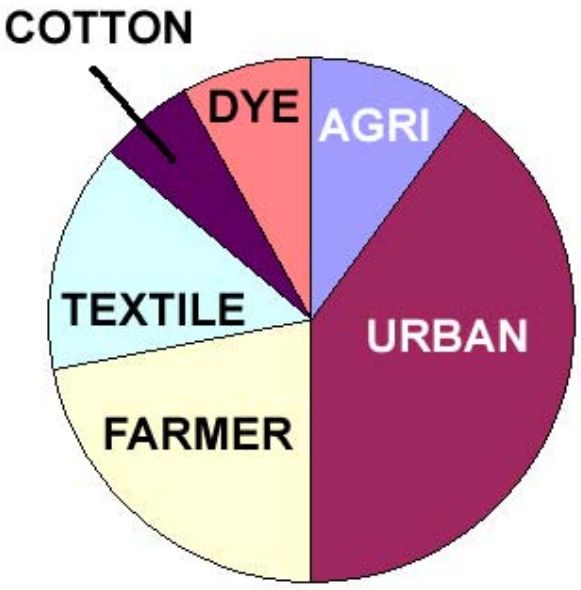
They were examined to look for signs of pulmonary hypertension, Corpulmonale.

They are subjected to pulmonary function tests to select patients with

FEV1 < 80 , and reversibility < 15

Then ECG , ECHO , taken for analysis.

Occupational Analysis of COPD



DATA ANALYSIS AND RESULTS

OCCUPATION

		percentage
Farmers[rural]	11	22%
urban people	20	40 %
Cotton workers	3	6%
Textile workers	7	14 %
Agricultural laborers[rural]	5	10 %
Workers in dye industry	4	8 %

RESULTS AND ANALYSIS COPD PATIENTS

SEX WISE ANALYSIS OF COPD PATIENTS

Sex wise analysis of patients		
		percentage
Male	27/50	54 %
Female	23/50	46%

Age wise analysis

Age group	Number of Copd	percentage
40----50	10	20 %
50----60	22	44 %
60---70	15	30 %
70—80	3	6 %

SYMPTOM ANALYSIS OF COPD PATIENTS

		Percentage
Cough	**50**	**100%**
Dyspnea	**45**	**90%**
Wheeze	**50**	**100%**

HISTORY ANALYSIS OF COPD PATIENTS

Smoker	**23/50**
More frequent	
Admissions	**35/50**

SIGN ANALYSIS OF COPD PATIENTS

Signs	num	percentage
Jugular venous pulse	10	20%
As cites	11	22%
Edema	10	20%
Parasternal heave	10	20%
Loud p2	11	22%

PULMONARY FUNCTION TEST ANALYSIS

PFT FEV1 <	80 %	
	50/50	
Reversibility	< 15 %	
	50/50	

ECG ANALYSIS OF COPD PATIENTS

ECG FINDING	NUM	percentage
P pulmonale	17/50	34%
Rt axis	29/50	58%
R > 5mm	5/50	10%
2+3+avF p wave amplitude> 9mm	9/50	18%
RBBB	2/50	4%
CAHD	9/50	18%

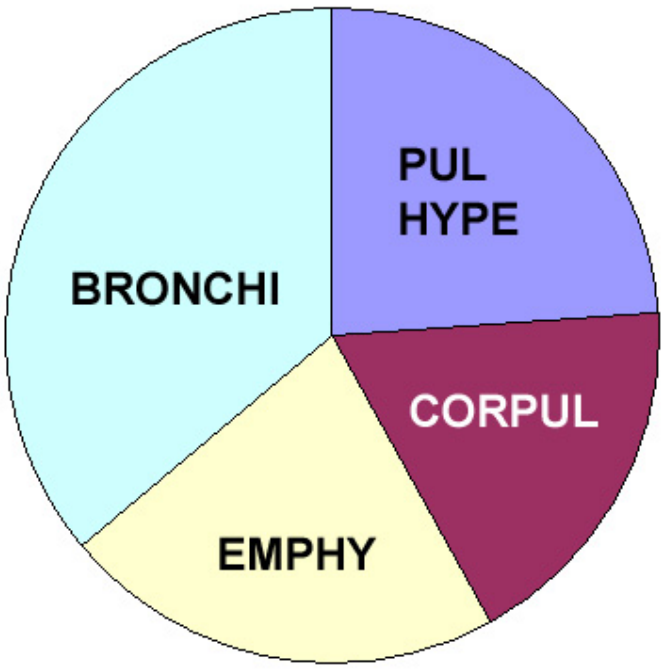
ECHO ANALYSIS

Complication		Percentage
PHT	12/5O	24%
Tricuspid Regurgitation	11/50	22%

X RAY ANALYSIS

X RAY PA VIEW	
RVH	13/50
Lateral view RVH	13/50

DISEASE WISE ANALYSIS



DISEASE ANALYSIS

Pulmonary	NO	percentage
Hypertension	12	24%
Corpulmonale	9	18%
Emphysema	11	22%
COPD Bronchitis	18	36 %

DISCUSSION

From the above data and analysis , It is clear male patients with Smoking history is more prone for COPD , in particular Emphysema. Out of the fifty patients , 27 patients were males . among them 23 Patients were smokers .out of these 23 , 13 males presented with ,cough , Dyspnea., Wheeze.

Radiologically all the 13 patients showed small , pendulous heart , with. Flattened diaphragm and increased radiolucency with widened intercostals spaces [hyperinflation]

Among the 50 , 12 were echocardiographically had pulmonary hypertension 11 had tricuspid regurgitation [which is a noninvasive measure of pulmonary arterial pressure, which we can not afford to measure via catheter.

Among the 50 patients ECG 17 patients had p pulmonale,29 Patients had right axis, deviation 5 had **r ** wave height more than 5 mm [indicating they had severe Right ventricular hypertrophy or pulmonary hypertension.

9 had 2+3+avF > 9 mm of p wave amplitude this is specifically taken Into consideration as this [2+3+avF > 9mm] is an indication for Long term oxygen therapy in copd patients as per the American Thoracic Society .

Also the ECGs were looked for any ischemic heart disease 9 Out of 50 had CAHD . as this increases the incidence of corpulmonale [smoking is an risk facor for both COPD [Emphysema, Bronchitis] and CAHD.

Industrialization and impact

Out of the fifty patients fourteen patients were working in various industries in coimbatore . seven were working in textile industry , while four were working in dye industry , three were working in cotton mills .out seven workers in textile 5 patients were smokers ,2 females are non smokers . four textile workers had bronchitis , 2 were emphysematous 1 had mild copd . generally the incidence of emphysema was more common in smokers 13 /23 in the group of 50 patients . in smokers working in textile mills it was the bronchitis that was more common. while smoking commonly produce centri acinar emphysema , in textile workers the fine fabric related chemical particles increase the incidence of bronchitis in these patients. In dye related workers ou of the four 2 were emphysematous , and 2 had mild degree of copd in the 3 cotton workers 2 had bronchitis , 1 had mild copd

Urbanization and impact

out of the fifty copd patients ,20 were from urban areas .among the twenty 9 were urban females without smoking history , but they had copd . seven had bronchitis, one had mild copd ,one had emphysema .this indicates the increased level pollution in urban areas .11 males were copd patients .all the 11 were smokers and nine out the11 were emphysematous ,2 were bronchitis patients . While just 16 out of 50 were from rural areas , the major air pollutants were released from automobiles like carbon monoxide, carbon dioxide , sulpurdioxide ,lead. So urbanization had its toll , by increasing the level of pollutants. In the air and increasing the number of copd patients even though coimbatore has green trees all around I had high level of copd patients .so we want strict pollution control acts which should be implemented strictly .

From the above data we can infer that the rapid pace of Urbanization, pollution of air , textile industry , dye industry ,cotton industry , farming activities , smoking are major triggers for copd more over the complications of copd like pulmonary hypertension corpulmonale were better found by ecg and echocardiogram. The use of ecg was in detecting the patients who need o2 Therapy [if the combined 2+3+avF P wave amplitude more than 9 mm] . the evidence of pulmonary hypertension right Ventricular hypertrophy ,detected well ,. Normally it is difficult to Estimate pulmonary arterial pressure invasively via catheter but Easy to measure it indirectly if we measure it by detecting Tricuspid regurgitation nonivasively by echocardiogram .11 patients had tricuspid regurgiation.

Cardiovascular complications

Out of the 50 COPD patients , 24 % developed pulmonary hypertension, 22 % developed tricuspid regurgitation .the chronic hypoxia of COPD is responsible for the development of pulmonary hypertension.the tricuspid regurgitation is the best possible non invasive way of measuring pulmonary artery pressure

34 % patients had p pulmonale , 18 % patients had the total p wave amplitude in 2, 3 , avF more than 9 mm , this is important because this one of the indication for life long oxygen therapy.

18% patients had concomitant CAHD ,this observation is important, becase systemic inflammation plays enhanced role in atherosclerosis, diabetes. TNF alpha is increased in COPD, patients

CONCLUSION

Pulmonary hypertension was the major cardiovascular complication of COPD , the chronic hypoxia of COPD is responsible for the development of pulmonary hypertension .24 percent of the total cases had pulmonary hypertension. The need for life long oxygen therapy was reemphasized in this study by establishing that nine patients had p wave amplitude summation in 2 , 3 , av F of more than 9 mm .

The study also established that the best non invasive way to detect raised pulmonary arterial pressure was detecting tricuspid regurgitation by echocardiogram.

Males Predominantly had Emphysema female had predominantly Chronic Bronchitis with corpulmonale

9 /50 had 2+3+avF *P* wave amplitude > 9m as this the one of the indication for **Long Term Oxygen Therapy as per The American Thoracic Society**.

Urbanization , pollution of air , textile industry , dye industry ,cotton industry , farming activities , smoking are major triggers for copd more over the complications of copd like pulmonary hypertension corpulmonale were better found by ecg and echocardiogram.

The use of ecg was in detecting the patients who need o2 Therapy [if the combined 2+3+avF P wave amplitude more than 9 mm] . the evidence of pulmonary hypertension right Ventricular hypertrophy were, detected well,. Normally it is difficult to Estimate pulmonary arterial pressure invasively via catheter but Easy to measure it indirectly if we measure it by detecting Tricuspid regurgitation nonivasively by echocardiogram. 18% patients had concomitant CAHD ,this observation is important, because systemic inflammation plays enhanced role in atherosclerosis , diabetes .[TNF alpha is increased in COPD, patients]

Finally the study by analyzing COPD patients with clinical arameters , ECG , ECHOCARDIOGRAM, concluded that pulmonary hypertension was the most common cardiovascular complication leading to cor pulmonale, and emphysema was more common in smokers , bronchitis was more common in females , the prevalence of COPD was more in urban areas due o pollution of air by automobiles. The textile based industries were mainly responsible for the increased prevalence of COPD .

BIBILIOGRAPHY

1. Fishman text book of pulmonalogy

2. Crofton text book of pulmonalogy

3. Harrisson book of internal medicine

4. American Thoracic Society** Chronic bronchitis,ashma andplmonary emphysema* Definitions and classification,Am Rev Respir Dis 85—762—768, 1962

5. American Thoracic Society * Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease 78-79--1995

6. American Thoracic Society Official statement

7. Baile

8. Bates Respiraory Function in Disease 3rd ed Philadelphi sanders

9. Eriksson 30 year perspective on alpha 1 antitrypsin deficiency

10. Thurlbeck WM * Chronic airflow obstruction*

11. Aaron SD, Vandemheen KL, Hebert P, et al: Outpatient oral prednisone after emergency treatment of chronic obstructive pulmonary disease. N Engl J Med 2003 Jun 26; 348(26): 2618-25[Medline].

12. Alter HJ, Koepsell TD, Hilty WM: Intravenous magnesium as an adjuvant in acute bronchospasm: a meta-analysis. Ann Emerg Med 2000 Sep; 36(3): 191-7[Medline].

13. Baigorri F, Joseph D, Artigas A, Blanch L: Inhaled nitric oxide does not improve cardiac or pulmonary function in patients with an exacerbation of chronic obstructive pulmonary disease. Crit Care Med 1999 Oct; 27(10): 2153-8[Medline].

14. Balter MS, La Forge J, Low DE, et al: Canadian guidelines for the management of acute exacerbations of chronic bronchitis. Can Respir J 2003 Jul-Aug; 10 Suppl B: 3B-32B[Medline]

15. Barr RG, Rowe BH, Camargo CA: Methyl-xanthines for exacerbations of chronic obstructive pulmonary disease. Cochrane Database Syst Rev 2001; CD002168[Medline]

16. Cydulka RK, Emerman CL: Effects of combined treatment with glycopyrrolate and albuterol in acute exacerbation of chronic obstructive pulmonary disease. Ann Emerg Med 1995 Apr; 25(4): 470-3[Medline]

17. Dailey RH: Chronic obstructive pulmonary disease. In: Rosen P, et al, eds. Emergency Medicine Concepts and Clinical Practice. 3rd ed. Mosby-Year Book Inc; 1992:1093-1111.

18. De Palo VA: Pulmonary disease: pneumonia, chronic obstructive pulmonary disease, asthma, and thromboembolic disease. J Am Podiatr Med Assoc 2004 Mar-Apr; 94(2): 157-67[Medline].

19. Dewan NA, Daniels A, Zieman G, Kramer T: The National Outcomes Management Project: a benchmarking collaborative. J Behav Health Serv Res 2000 Nov; 27(4): 431-6[Medline]

20. Faulkner MA, Hilleman DE: Pharmacologic treatment of chronic obstructive pulmonary disease: past, present, and future. Pharmacotherapy 2003 Oct; 23(10): 1300-15[Medline].

21. Ferguson GT, Cherniack RM: Management of chronic obstructive pulmonary disease. N Engl J Med 1993 Apr 8; 328(14): 1017-22[Medline].

22. FitzGerald JM, Shragge D, Haddon J, et al: A randomized, controlled trial of high dose, inhaled budesonide versus oral prednisone in patients discharged from the emergency department following an acute asthma exacerbation. Can Respir J 2000 Jan-Feb; 7(1): 61-7[Medline].

23. Fromm RE Jr, Varon J: Acute exacerbations of obstructive lung disease: What to do when immediate care is critical. Postgraduate Medicine 1994; 95(8): 101-6[Medline].

24. Greenfield RH: Pulmonary disease in the elderly. Lecture presented at the ACEP Scientific Assembly. 1994.

25. Hirschmann JV: Do bacteria cause exacerbations of COPD?. Chest 2000 Jul; 118(1): 193-203[Medline].

26. Hirshberg AJ, Dupper RL: Use of doxapram hydrochloride injection as an alternative to intubation to treat chronic obstructive pulmonary disease patients with hypercapnia. Ann Emerg Med 1994 Oct; 24(4): 701-3[Medline].

27. Ingram RH: Chronic bronchitis, emphysema, and airway obstruction. In: Fauci AS, et al, eds. Harrison's Principles of Internal Medicine. McGraw-Hill Companies; 1991: 1074-1082.

28. Jaber S, Fodil R, Carlucci A, et al: Noninvasive ventilation with helium-oxygen in acute exacerbations of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000 Apr; 161(4 Pt 1): 1191-200[Medline].

29. Kino RJ: The difficult COPD patient: Alternative therapy regimens. Lecture presented at the ACEP Scientific Assembly. 1996.

30. Korosec M, Novak RD, Myers E, et al: Salmeterol does not compromise the bronchodilator response to albuterol during acute episodes of asthma. Am J Med 1999 Sep; 107(3): 209-13[Medline].

31. Lieberman D, Lieberman D, Ben-Yaakov M, et al: Infectious etiologies in acute exacerbation of COPD. Diagn Microbiol Infect Dis 2001 Jul; 40(3): 95-102[Medline]

32. Maltais F, Ostinelli J, Bourbeau J, et al: Comparison of nebulized budesonide and oral prednisolone with placebo in the treatment of acute exacerbations of chronic obstructive pulmonary disease: a randomized controlled trial. Am J Respir Crit Care Med 2002 Mar 1; 165(5): 698-703[Medline].

33. Peng CC, Aspinall SL, Good CB, et al: Equal effectiveness of older traditional antibiotics and newer broad-spectrum antibiotics in treating patients with acute exacerbations of chronic bronchitis. South Med J 2003 Oct; 96(10): 986-91[Medline].

34. Pierson DJ: Indications for mechanical ventilation in adults with acute respiratory failure. Respir Care 2002 Mar; 47(3): 249-62; discussion 262-5[Medline].

35. Saint S, Bent S, Vittinghoff E, Grady D: Antibiotics in chronic obstructive pulmonary disease exacerbations. A meta-analysis. JAMA 1995 Mar 22-29; 273(12): 957-60[Medline].

36. 36 Schmidt GA, Hall JB: Acute chronic respiratory failure: Assessment and management of patients with COPD in the emergent setting. JAMA 1989; 261(23): 3444-3453[Medline].

37. Sclar DA, Legg RF, Skaer TL, et al: Ipratropium bromide in the management of chronic obstructive pulmonary disease: effect on health service expenditures. Clin Ther 1994 May-Jun; 16(3): 595-601; discussion 594[Medline].

38. Siedman JC: Chronic obstructive pulmonary disease. In: Tintinalli J, et al, eds. Emergency Medicine: A Comprehensive Study Guide. McGraw-Hill Com; 1992: 298-302.

39. Skorodin MS, Tenholder MF, Yetter B, et al: Magnesium sulfate in exacerbations of chronic obstructive pulmonary disease. Arch Intern Med 1995 Mar 13; 155(5): 496-500[Medline].

40. Skorodin MS: Pharmacotherapy for asthma and chronic obstructive pulmonary disease. Current thinking, practices, and controversies. Arch Intern Med 1993 Apr 12; 153(7): 814-28[Medline].

41. Sohy C, Pilette C: Acute exacerbation of chronic obstructive pulmonary disease and antibiotics: what studies are still needed? Euorpean Respiratory Journal 2002; 19: 966-75.

42. dejouse JC, Billings CG, et al: Effects of angiotensin converting enzyme inhibition on sodium excretion in patients with hypoxemic chronic obstructive pulmonary disease. Thorax 1994; 49(10): 995-8[Medline].

43. Varkey B: Obstructive, occupational, and environmental diseases. Curr Opin Pulm Med 2004 Mar; 10(2): 97[Medline].

44. Whittle A: COPD guidelines. Thorax. Zehner WJ, Scott JM, Iannolo PM, et al: Terbutaline vs albuterol for out-of-hospital respiratory distress: randomized, double-blind trial. Acad Emerg Med 1995 Aug; 2(8): 686-91[Medline].

45. World Health Organization. Chronic cor pulmonale: a report of the expert committee. Circulation 1963;27:594-598. Advertisement

46. MacNee W. Pathophysiology of cor pulmonale in chronic obstructive pulmonary disease: part one. Am J Respir Crit Care Med 1994;150:833-852.

47. Fishman AP. Clinical classification of pulmonary hypertension. Clin Chest Med 2001;22:385-391.

48. Tartulier M, Bourret M, Deyrieux F. Les pressions artérielles pulmonaires chez l'homme normal: effets de l'âge et de l'exercice musculaire. Bull Physiopathol Respir 1972;8:1295-1321.

49. Bishop JM. Cardiovascular complications of chronic bronchitis and emphysema. Med Clin North Am 1973;57:771-780.

50. Fishman AP. Hypoxia on the pulmonary circulation: how and where it acts. Circ Res 1976;38:221-231.

51. Segel N, Bishop JM. The circulation in patients with chronic bronchitis and emphysema at rest and during exercise, with special reference to the influence of changes in blood viscosity and blood volume on the pulmonary circulation. J Clin Invest 1966;45:1555-1568.

52. Burrows B. Arterial oxygenation and pulmonary hemodynamics in patients with chronic airways obstruction. Am Rev Respir Dis 1974; 110:64-70.

53. Burrows B, Kettel LJ. Niden AH, Rabinowitz M, Diener CF. Patterns of cardiovascular dysfunction in chronic obstructive lung disease. N Engl J Med 1972;286:912-918.

54. Weitzenblum E, Loiseau A, Hirth C, Mirhom R, Rasaholinjanahary J. Course of pulmonary hemodynamics in patients with chronic obstructive pulmonary disease. Chest 1979;75:656-662

55. Weitzenblum E, Sautegeau A, Ehrhart M, Mammosser M, Hirth C, Roegel E. Long-term course of pulmonary arterial pressure in chronic obstructive pulmonary disease. Am Rev Respir Dis 1984;130:993-998.

ABBREVIATIONS IN MASTER CHART

Paraste---------- parasternal heave

Freqadi---------- frequency of admission

Reversi---------- reversibility

P pulmonale---------p pulmonale

Rbbb----------right bundle branch block

CAHD---------- coronary artery disease

PHT----------pulmonary hypertension

TR----------tricuspid regurgitation

p----------present,

A---------- absent

R----------right axis deviation

RVH---------- right ventricular hypertrophy

Small hear---------- small heart

Ne---------- sputum AFB negative

N---------- normal axis

Ag ---------- agricultural worker

urb ---------- urban

Fa ----------farmer

Co ---------- cotton

Tex ----------Textile worker

Dye---------- dye industry worker

Oc---------- occupation

S.No	IP no	Age	Sex	cough	dyspnea	wheeze	jvp	ascites	pedal ede	parasthe	loud p2	smoker	frequadl	FEV1<80	reversi<15	p pulmona	
1	446022	56	male	p	A	P	A	A	A	A	A	P	P	P	P	A	
2	467211	42	female	P	P	P	P	P	P	P	P	A	P	P	P	P	
3	468123	57	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
4	487199	63	female	p	p	p	p	p	A	A	A	A	A	P	P	P	
5	486612	57	female	P	P	P	A	A	A	A	A	A	P	P	P	P	
6	486954	60	MALE	P	P	P	A	A	A	A	A	P	P	P	P	A	
7	495163	55	female	p	p	p	A	A	A	A	A	A	A	P	P	A	
8	494251	40	female	p	A	P	A	A	A	A	A	A	A	P	P	A	
9	498190	51	MALE	P	P	P	P	P	P	P	P	P	P	P	P	P	
10	504253	65	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
11	467239	60	female	P	P	P	A	A	A	A	A	A	P	P	P	A	
12	516345	63	male	P	P	P	A	A	A	A	A	PP	P	P	P	A	
13	485273	56	female	P	P	P	A	P	P	P	P	A	P	P	P	P	
14	494590	61	female	p	p	p	A	A	A	A	AA	A	A	P	P	A	
15	495662	71	female	P	P	P	P	P	P	P	P	P	P	P	P	P	
16	506011	63	male	P	P	P	A	A	A	A	A	A	P	P	P	A	
17	506155	52	male	p	p	P	A	A	A	A	A	P	P	P	P	A	
18	515342	61	female	P	P	P	P	P	P	P	P	A	P	P	P	P	
19	512234	65	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
20	503341	43	female	p	p	p	A	A	A	A	A	A	A	P	P	A	
21	512345	51	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
22	523445	60	male	P	P	P	P	P	P	P	P	P	P	P	P	P	
23	523612	45	female	P	P	P	A	A	A	A	A	A	A	P	P	A	
24	534323	56	female	P	P	P	A	A	A	A	A	A	P	P	P	A	
25	533223	70	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
26	543122	56	male	P	A	P	A	A	A	A	P	P	P	P	A	A	
27	542231	44	female	P	A	P	A	A	A	A	A	A	A	P	P	A	
28	523423	67	MALE	P	P	P	A	A	A	A	A	PPP	P	P	A	N	
29	543121	41	female	p	p	p	A	A	A	A	A	A	A	P	P	P	
30	554554	60	male	P	P	P	A	A	A	A	A	A	A	P	P	P	
31	556231	54	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
32	551133	46	female	P	A	P	A	A	A	A	A	A	A	P	P	A	
33	551245	57	male	P	P	P	A	A	A	A	A	PP	P	P	P	A	
34	555672	75	female	p	p	p	p	p	p	p	p	A	P	P	P	P	
35	562136	63	male	p	p	p	p	p	p	p	P	A	P	P	P	P	
36	567341	69	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
37	564672	49	female	P	P	P	A	A	A	A	A	A	A	P	P	P	
38	564533	46	male	P	P	P	A	A	A	A	A	A	A	P	P	P	
39	579447	61	male	P	P	P	A	A	A	A	A	PP	P	P	P	A	
40	576764	59	female	p	P	P	A	A	A	A	A	A	P	P	P	A	
41	576642	65	male	p	P	P	A	A	A	A	A	P	P	P	P	A	
42	587554	52	female	P	P	P	A	A	A	A	A	A	P	P	P	P	

43	586121	45	male	P	P	P	A	A	A	A	A	P	A	P	P	A	
44	596214	54	female	P	P	P	A	A	A	A	A	A	P	P	P	A	
45	595342	65	male	P	P	P	A	A	A	A	A	P	P	P	P	A	
46	602342	60	male	p	p	P	A	A	A	A	A	A	A	P	P	A	
47	607234	72	female	p	p	p	P	P	P	P	P	A	P	P	P	P	
48	607245	59	male	P	P	P	A	A	A	A	A	P	A	P	P	A	
49	607754	64	female	P	P	P	P	P	P	P	P	A	P	P	P	P	
50	608765	65	male	P	P	P	A	A	A	A	A	PP	P	P	P	A