International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

Clinical Review on Diagnosis and Treatment of Chronic Obstructive Pulmonary Disease.

M.Narmatha*, B.Naveena, A. Melwin raj, P. Pratheesh Xavier, prof. P. Thirupathi kumarasen,
Arulmigu kalasalingam college of pharmacy, krishnankoil.

Submitted: 10-10-2022 Accepted: 21-10-2022

ABSTRACT

This paper is about the diagnosis and treatment of chronic obstruction pulmonary disease. COPD is a curable disease which is characterized by progressive airflow obstruction and inflammation in the airway of the lungs. Patients with mild COPD have mild to moderate hypoxemia without hypercapnia. The diagnosis of severe AAT deficiency is confirmed when the serum level falls below the protective threshold value of 11 mmol/L.A frontal and lateral chest radiographs of patients with emphysema reveal signs of hyperinflation, including flattening of the diaphragm, increased retrosternal air space, and a long, narrow heart shadow. Genetic and environmental variables can have an impact on the complex, multidimensional disease known as COPD. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) recommendations, bronchodilators should be the cornerstone of COPD treatment and can be taken orally or by inhalation. In general, antimuscarinic bronchodilators are thought to be more efficient for COPD than for asthma.Beclomethasonedipropionate, budesonide, flunisolide, fluticasone propionate, and triamcinoloneacetonide are some of the currently accessible inhaled glucocorticoids.

KEYWORDS: Arterial blood gas, spirometry, Pulmonary rehabilitation.

INTRODUCTION

The American Thoracic Society describes chronic obstructive pulmonary disease (COPD) as a averting and curable disease which is characterized by progressive airflow obstruction and inflammation in the airway of the lungs caused by significant exposure to noxious particles or gases such as cigarette smoking [1]. The chronic exacerbation of COPD is characterized by dyspnea, increased cough and sputum production. The exacerbation is caused by recurrent bacterial or viral infection. These COPD exacerbation decline the quality of life of patient [2]. The diagnosis of COPD is done through a detailed medical history by estimating the exposure to risk factors , family history, co-morbid conditions, smoking cessation. The aim of Global Initiative for chronic obstruction Lung Disease (GOLD) has four constituent for clinical management of COPD includes assessing and monitoring the diseased condition, reducing risk factor managing the COPD and management of exacerbations [3]. In acute exacerbation, COPD patient may become transiently severe hypoxemic such as Spo2 ≤ 88%or Pao2 ≤ 55 mmHg and the condition is treated with "short term oxygen therapy" [4].

COPD comprises of chronic bronchitis and emphysema. ATS defined chronic bronchitis as the presence of cough and sputum for atleast 3 months in each 2 successive years. Chronic bronchitis associated with high risk of mortality in COPD patients. The risk factors also includes limitation in physical activity, progression of diseased condition, deterioration in respiratory and general health status [5]. Emphysema is defined as a condition distinguished by uncommon, permanent elongation of airspace distal to the terminal bronchioles of the lungs associated with the destructive changes of the alveolar walls. In different stages of COPD patient, there is a complete narrowing of terminal bronchioles with minimal damage to distal parenchyma [6]. COPD is one of the major leading causes of morbidity and mortality in worldwide. In 2017, approximately 3.2 million death occurs related to COPD globally. It is the third leading causes of death worldwide. Patient with co-morbid conditions such as diabetes, anemia and heart disease has a impairment in health related quality of life [7].

DIAGNOSIS

ARTERIAL BLOOD GAS ANALYSIS

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

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Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

of disease exacerbation. Patients with mild COPD have mild to moderate hypoxemia without hypercapnia. As the disease progresses, hypoxemia worsens and hypercapnia may develop, with the latter commonly being observed as the FEV1 falls below 1 L/s or 30% of the predicted value. Lung mechanics and gas exchange worsen during acute exacerbations. Serum Chemistries Patients with COPD tend to retain sodium. In addition, serum potassium should be monitored carefully, because diuretics, beta-adrenergic agonists, and theophylline act to lower potassium levels. Beta-adrenergic agonists also increase renal excretion of serum calcium and magnesium, which may be important in the presence of hypokalaemia. Chronic respiratory acidosis leads to compensatory metabolic alkalosis. In the absence of blood gas measurements, bicarbonate levels are useful for following disease progression .An arterial blood-gas analysis is obligatory only in severe cases of COPD or in patients with polycythemia or corpulmonale. It is also recommended for patients whose oxygen saturation level in pulse oximetry is below 92% [8].

ALPHA1-ANTITRYPSIN

Measure alpha1-antitrypsin (AAT) in all patients younger than 40 years, in those with a family history of emphysema at an early age, or patients with emphysematous changes with no smoking history. The diagnosis of severe AAT deficiency is confirmed when the serum level falls below the protective threshold value of 11 mmol/L (ie, in the range of 3-7 mmol/L).

SPUTUM EVALUATION

In persons with stable chronic bronchitis, the sputum is mucoid and macrophages are the predominant cells. With an exacerbation, sputum becomes purulent because of the presence of neutrophils. Although the quality of sputum can vary between patients in chronic stable disease, an increase in the quantity of sputum production is often a sign of an acute exacerbation. Amixture of organisms often is visible with Gram stain. The pathogens cultured most frequently during exacerbations are Streptococcus pneumoniae and Haemophilusinfluenzae. Moraxellacatarrhalis is also a common organism, and Pseudomonas aeruginosa can be seen in patients with severe obstruction.

B-TYPE NATRIURETIC PEPTIDE

Human B-type natriuretic peptide (BNP) binds to particulate guanylatecyclase receptors of vascular smooth muscle and endothelial cells. Binding to the receptors causes an increase in cyclic guanosinemonophosphate (GMP), which serves as a secondary messenger to dilate veins and arteries. BNP is secreted by the ventricles of the heart when there is increased stretch of the myocytes (i.e., in CHF).By measuring BNP, it was thought that the ability to differentiate between CHF and COPD exacerbations would become much easier. However, clinical observation and research have shown that in cases of mild CHF exacerbation, differentiation between CHF and COPD is still not straightforward. A mild elevation of BNP must be taken in context with the overall clinical picture.

CHEST RADIOGRAPHY

A frontal and lateral chest radiographs of patients with emphysema reveal signs of hyperinflation, including flattening of the diaphragm, increased retrosternal air space, and a long, narrow heart shadow. Rapidly tapering vascular shadows accompanied by hyperlucency of the lungs are other signs of emphysema. Chronic bronchitis is associated with increased bronchovascular markings and cardiomegaly .With complicating pulmonary hypertension, the hilar vascular shadows are prominent, with possible right ventricular enlargement and opacity in the lower retrosternal air space.

COMPUTED TOMOGRAPHY

High-resolution CT (HRCT) scanning is more sensitive than standard chest radiography and is highly specific for diagnosing emphysema (outlined bullae are not always visible on a radiograph).HRCT scanning may provide an adjunct means of diagnosing various forms of COPD (ie, lower lobe disease may suggest AAT deficiency) and may help the clinician to determine whether surgical intervention would benefit the patient.

TWO DIMENSIONAL ECHOCARDIOGRAPHY

Many patients with long-standing COPD develop secondary pulmonary hypertension from chronic hypoxemia and vascular remodeling. This may result in eventual right-sided heart failure (corpulmonale). However, even with severe COPD, the degree of pulmonary hypertension is usually only mild to moderate. Findings of severe pulmonary hypertension on echocardiogram or cardiac catheterization warrant further workup. Two-dimensional echocardiography may be helpful as a screening tool to estimate pulmonary arterial

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

systolic pressure and right ventricular systolic function, although formal cardiac catheterization is necessary to accurately confirm the diagnosis.

PULMONARY FUNCTION TEST

Pulmonary function tests are essential for the diagnosis and assessment of the severity of disease, and they are helpful in following its progress. FEV1 is a reproducible test and is the most commonly used index of airflow obstruction. In addition to the spirometry findings that define the disease, lung volume measurements often show an increase in total lung capacity, functional residual capacity, and residual volume. The vital capacity often decreases. Dynamic hyperinflation during exercise is now thought be a greater contributor to the sensation of dyspnoea than airflow obstruction alone (as measured by FEV1).As many as 30% of patients have an increase in FEV1 of 15% or more after inhalation of a bronchodilator. However, the absence of bronchodilator response does not justify withholding therapy. spirometry is accepted as the diagnostic test to assess airflow obstruction and classify severity of disease, based on specific cut points for FER (FEV1/FVC <0.7 after bronchodilator) and FEV1 (mild ≥80% predicted, moderate 50-80%, severe 3049% predicted, very severe <30% predicted). FEV1 normally decreases with age, and the rate of fall is an important spirometric indicator of disease progression in COPD. In healthy non-smoking adults the decrease is about 30 mL/year with an upper limit of about 50 mL/year [9].

MANAGEMENT

Challenges in management of COPD

Genetic and environmental variables can have an impact on the complex, multidimensional disease known as COPD. Due to the variety of COPD, there is growing interest in categorising patients into several phenotypes based on their clinical traits. To manage the disease, healthcare practitioners must collaborate with their patients to identify the ideal mix of pharmacological and non-pharmacological therapy approaches [10].

Abnormal lung function growth trajectories may develop starting at infancy as a result of environmental and genetic causes. Recent research has linked these trajectories to clinical consequences, including the likelihood of developing respiratory diseases. There are certain people who never attain their peak lung function but instead endure "normal" rates of deterioration, according to studies. Others may have the quickest deterioration in lung function far sooner than initially anticipated [11].

Treatment Goals

The majority of nations considering the important therapeutic objectives are

- Identified delaying disease progression
- Lowering mortality
- Minimising future risk for exacerbations
- Lessening the severity of existing symptoms .

The treatment objectives listed in the recommendations from the Czech Republic, Finland, France, Poland, Portugal, and Sweden included easing symptoms, stopping the disease's natural progression, enhancing physical activity, enhancing quality of life, avoiding complications and negative outcomes, and lengthening life expectancy.

Improvements in symptoms, exercise tolerance, quality of life, and a decrease in exacerbation frequency were noted in the German and Spanish studies.

The Russian recommendations contained two sets of long-term objectives: limiting disease progression and exacerbations; and lowering mortality. The short-term objectives were symptom alleviation, improvement of exercise tolerance, and quality of life [12].

NON PHARMACOLOGICAL TREATMENT

Smoking cessation

When tobacco is burned insufficiently, a heterogeneous aerosol made up of more than 4000 different chemicals is emitted as cigarette smoke. It results in a complicated alteration of the cellular and humoral immune response, which in turn promotes respiratory and systemic infections and weakens the immune system's ability to manage mutations [15]. It is well established that smoking poses the greatest risk for developing COPD in vulnerable hosts. According to the Lung Health Study, those with mild to moderate COPD who gave up smoking had an improvement in their FEV1 in the year after their quit, and their future rate of FEV1 decrease was half that of people who kept smoking [13]. Reduced cigarette use or intermittent stopping did not result in the same rate of fall in FEV1 as complete and sustained cessation, unless the percent reduction was extremely significant (>85%). Complete cessation and abstinence have both showed a lower rate of decline in FEV1 [14].

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

Pulmonary rehabilitation

Pulmonary rehabilitation in the treatment of patients with chronic respiratory diseases has expanded significantly, and it is now well established that this all-encompassing intervention reduces dyspnea, enhances exercise capacity, and enhances health-related quality of life (HRQL)[17]. Reducing symptoms, enhancing activity and everyday function, and restoring the maximum level of independent function are the objectives of pulmonary rehabilitation programmes for patients with lung diseases. Physiological and psychological outcome measurements can be used to evaluate the success of reaching these aims [16]. Secondary impairments, such as peripheral muscular, cardiac, nutritional, and psychological dysfunction, as well as inadequate self-management measures, cause significant morbidity in COPD patient. Patient education entails more than just presenting didactic knowledge. To encourage self-management abilities and self-efficacy, it combines education, counselling, and behaviour modification strategies. The entire treatment plan should include end-of-life decision-making as part of the patient education process. The best method for enhancing muscular function in COPD and other chronic respiratory disorders is exercise training, which is widely considered as the cornerstone of pulmonary rehabilitation . There hasn't been much research done on the ideal amount of time for pulmonary rehabilitation exercise training. Four weeks of outpatient exercise training with two or three weekly sessions yielded less effect than seven weeks of the same training . Additionally, it has been shown that thorough pulmonary rehabilitation over 20 sessions significantly improves many outcomes compared to just 10 sessions. Furthermore proven successful are short-term, intense programmes (20 sessions compressed into 3–4 weeks). Most experts agree that lengthier programmes produce greater, more persistent training results. In order to get the most physiologic benefits, patients should exercise at least three times per week and under frequent supervision. Due to the limitations of the programme, twice-weekly supervised fitness instruction plus one or more unsupervised sessions at home may be an option, however it is unknown if this will be as successful [17].

Immunization

Vaccines are useful preventative interventions in people with respiratory disorders, particularly those with COPD. Anti-pneumococcal and anti-influenza vaccinations are typically advised for patients with this condition. Various vaccinations in COPD patients in the recent introduction of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) vaccine has also been effective.

There are two different forms of pneumococcal vaccines: polysaccharide and conjugate. Because the immune response from the 13-valent conjugate vaccination depends on both B lymphocytes and T lymphocytes, it creates an immunological memory and has a longer-lasting impact. In this approach, pneumococcal conjugate immunisation for COPD patients is advised by both national and international recommendations.

The influenza vaccine is an attenuated viral vaccine, not like the pneumococcal vaccination. Furthermore, no rise in Exacerbations of COPD was seen within 14 days of dosing in a systematic evaluation of the literature. The influenza vaccine was demonstrated to minimise exacerbations in COPD patients in another meta-analysis of randomised clinical trials, despite a modest rise in local adverse effects. The GOLD and recommendations advise people with COPD to receive an annual influenza vaccination, similar to how the pneumococcal vaccine is administered [18].

According to the Centers for Disease Control and Prevention (CDC), people with COPD should have the indication of pertussis and herpes zoster vaccinations. Since it is a part of the childhood vaccination schedule, the pertussis vaccine, which is incorporated into the combination diphtheria, tetanus, and pertussis vaccine, is often given to children. Since the majority of COPD patients were immunised as children, it is typically not required to mention this vaccination when a patient is newly diagnosed with COPD. However, it is vital to consider this vaccine when a patient has never been immunised before [19].

Non invasive ventilation

In COPD patients who have acute respiratory failure, non-invasive positive pressure ventilation (NIPPV) has been shown to reduce morbidity, increase survival, and lessen the need for mechanical ventilation. Its effectiveness in treating patients with moderate to severe respiratory acidosis [20]. Ventilator-associated pneumonia (VAP) and other nosocomial infections are some of the side effects of invasive ventilation that are connected to the intubation or tracheotomy surgery. Most difficulties associated with invasive ventilation can be avoided with non-invasive mechanical ventilation (NIV). NIV failure is more

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

common in the first few hours of ventilation and has been linked to the presence of co-morbidities, severe acidosis, high severity scores, severe impairment of consciousness, and a lack of improvement in arterial blood gases during the first one to two hours of ventilation . However, despite a brief initial improvement, COPD patients with severe ARF treated with NIV, especially those with more severe functional impairment during the stable state, may experience a late deterioration (after > 48hrs), frequently necessitating endotracheal intubation [21].

Long term oxygen therapy

According to the British Thoracic Society (BTS), patients with a PaO2≤7.3kPa or a PaO2 between 7.3kPa and 8kPa with symptoms of peripheral edoema, polycythemia (hematocrit>55%), or pulmonary hypertension should get long-term oxygen treatment. Long-term oxygen therapy is one of the few treatments that improve life expectancy in this group. The most dangerous result of hypoventilation, especially when sleeping, is hypoxaemia, hence treating any illness linked to respiratory insufficiency during sleeping with the proper oxygen treatment is crucial. In patients with COPD, care must be taken to ensure that hypercapnia does not aggravate the treatment of hypoxaemia since respiratory drive in such patients may be somewhat dependent on the stimulating impact of hypoxaemia [22].

Extracorporeal membrane oxygenation

In order to alleviate the demand for mechanical breathing and the concomitant morbidity associated with intubation, ECMO in the form of an extracorporeal CO2 remover (ECCO2R) is a novel treatment modality being employed to treat COPD exacerbations [20]. In patients with severe COPD exacerbations and threatening respiratory failure, extracorporeal CO2 removal has been used successfully as an addition to, or in place of, mechanical ventilation. An ECCO2R device that operates at low blood flow rates through a smaller, perhaps safer cannula may be able to alleviate the hypercapnic respiratory failure found in acute exacerbations of COPD and prevent the requirement for invasive mechanical ventilation [23].

Lung volume reduction surgery (LVRS) and endobronchial procedures

The National Emphysema Treatment Experiment (NETT) trial found that LVRS improved medical therapy. Lower BORG dyspnea scores and better maximum breathing rate and tidal volume are all benefits of LVRS. Surgery may associated with a substantial risk of morbidity, especially for patients with severe COPD who also have other co-morbidities. Therefore, innovative, less invasive, non-surgical techniques have been developed to perhaps aid individuals who would not be candidates for LVRS. This non-surgical treatments is called as Bronchoscopic lung volume reduction. Patients with heterogeneous upper zone emphysema and decreased exercise tolerance can benefit from LVRS. The most common bronchoscopic lung volume reduction devices include: endobronchial valves, foam sealant, metallic coils, airway bypass stents and vapor thermal ablation [20].

Lung transplantation

Since the first lung transplant performed by Dr. James Hardy at the University of Mississippi in 1963, lung transplantation has been a curative option for COPD. The International Society for Heart and Lung Transplantation states that patients with COPD must fulfil disease-specific criteria for referral, which includes having a FEV1 below 20% and utilising all available pharmacological and non-pharmacological treatments, such as bronchodilators, home oxygen, smoking cessation, and pulmonary rehabilitation. They should also have a BODE index greater than 7, if they exhibit corpulmonale symptoms or hypoxemic and hypercapnic respiratory failure [24] . The patient who is contraindicated to lung transplantation includes:

Absolute contraindication

- Apart from cutaneoussquamous and basal cell cancers, malignancy in the previous two years
- significant malfunction of another major organ system that is incurable
- Chronic extrapulmonary infections that are incurable, such as HIV, HBV, and HCV in active form
- An incurable mental illness or psychological disease characterised by a lack of compliance with or cooperation with medical treatment
- Active or recent substance abuse during the past six months.

Relative contraindication

- Older than 65 years of age
- Clinical state is unstable (e.g., shock, mechanical ventilation or ECMO)
- The BMI threshold for severe obesity is 30 kg/m2.
- Mechanical ventilation that is ongoing [20].

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

PHARMACOLOGICAL TREATMENT

BRONCHODILATORS

Beta 2 agonist

According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) recommendations, bronchodilators should be the cornerstone of COPD treatment and can be taken orally or by inhalation. Additionally, compared to inhaled short-acting bronchodilators, inhaled long-acting bronchodilators are more efficient and convenient [26]. Long-acting inhaled β2-adrenergic agonists such as salmeterol and formoterol twice daily have a prolonged bronchodilatory effect for approximately 12 hours. Ultra long acting β2-adrenergic agonists such as indacaterol and olodaterol once-daily medications with a continuous bronchodilator action for 24 hours[25]. Short acting β2-adrenergic agonists such as salbutamol and albuterol. The first-line therapy for acute asthma symptoms and exacerbations is SABAs. They are frequently used with long-acting muscarinic agonists, inhaled corticosteroids, or LABAs in the treatment of COPD. The major routes of administration for beta-2 agonists include metered dosed-inhalers, nebulizers, dry powder inhalers, orally, subcutaneously, or intravenously. The preferred route of administration for beta-2 agonists in the treatment of asthma and COPD is through inhalation. The adverse effect of beta 2 agonist includes cardiovascular, metabolic, or musculoskeletal systems. The risk of cardiac toxicity, including arrhythmias, cardiomyopathy, and ischemia, has been associated more strongly with beta-2 agonists of an older generation . Hypoxemia and hypercapnia have also been identified in several investigations as aggravating variables to the cardiotoxic effects of beta-2 agonists [27].

Antimuscarnic drugs

In general, antimuscarinic bronchodilators are thought to be more efficient for COPD than for asthma. Ipratropium aerosol inhalation reaches its peak impact 30–60 minutes after delivery; the medicine must be administered three–four times daily due to its shorter duration of action. A novel long-acting antimuscarnic medication called tiotropium bromide, which only has to be taken once daily, has better efficacy than existing bronchodilators. When used in conjunction with pulmonary rehabilitation, tiotropium raises exercise tolerance in COPD patients while they are asleep. Although tiotropium bromide is recommended for the maintenance treatment of COPD, acute bronchospasms cannot be effectively treated with it. Umeclidinium bromide and glycopyrronium bromide are the new LAMA developed for the treatment of COPD. Antimuscarinic bronchodilators should be administered with caution in glaucoma, benign prostatic hypertrophy and urinary obstruction [28].

Theophylline

Theophylline, a methylxanthine, relaxes airway smooth muscle by primarily inhibiting phosphodiesterase (PDE) isoenzymes. The reduction of neutrophils and interleukin-8 concentration in mediated sputum from COPD patients suggests that theophylline may have anti-inflammatory effects in addition to bronchodilation at lower plasma concentrations in the respiratory tract, whereas the same effects cannot be attained after inhaling high glucocorticoid doses [29]. Due to the possibility of major toxic consequences such arrhythmias and convulsions, theophylline must be administered intravenously very gradually over a minimum of 20 minutes.Theophylline has a therapeutic dose of 10–20 g/ml. Theophylline concentrations more than 25 g/ml are linked to toxic adverse effects. When theophylline concentrations exceed 40 g/ml, there is a substantial chance of experiencing serious toxic consequences such convulsions and arrhythmias. Theophylline's primary adverse effects at therapeutic plasma concentrations are anorexia, nausea, vomiting, sleeplessness, agitation, palpitations, and hypotension [28].

Inhaled Corticosteriods

Inhaled corticosteroids have an anti-inflammatory effect in patients with COPD remains controversial.Beclomethasonedipropionate, budesonide, flunisolide, fluticasone propionate, and triamcinoloneacetonide are some of the currently accessible inhaled glucocorticoids[28]. Although they are quite efficient in treating asthma, corticosteroids may not be able to stop the inflammatory disease process in COPD. Due to the poor response of COPD patients to inhaled corticosteroids, large dosages that may cause systemic adverse effects are frequently administered to them[30]. Oropharyngealcandidiasis is a frequent adverse effect brought on by the local immunosuppressive effects of glucocorticoids. Side effects include the potential for osteoporosis even at doses as low as 500 mg/day (Undem and Lichtenstein 2001), the inhibition of endogenous cortisol synthesis by inhibiting the hypothalamic-pituitary-adrenal axis,

International Journal of Pharmaceutical Research and Applications
Volume 7, Issue 5 Sep-Oct 2022, pp: 1157-1165 www.ijprajournal.com ISSN: 2456-4494

as well as a slightly elevated risk of glaucoma, particularly when long-term treatment at high doses, and cataract formation [28].

Antibiotics

Prophylactic antibiotics and chronic antibiotic therapy had almost no influence on the incidence of exacerbations in COPD, according to several large-scale controlled trials . In the winter season, preventative antibiotics are also useless. Current available data does not support the effectiveness of prophylactic antibiotics against bacterial infections or exacerbations of COPD.

Mucolytic agents

When compared to a placebo, mucolytic medications including ambroxol, erdosteine, carbocysteine, and iodinated glycerol reduce the frequency of exacerbations in chronic bronchitis . The routine administration of mucolytic medicines in COPD is not advised.

Antioxidants

The pathogenesis of COPD has a significant function for oxidative stress. Antioxidant agent administration is thus a fascinating therapeutic approach. N-acetylcysteine, an antioxidant and mucolytic, has been shown in most clinical trials to decrease the frequency of COPD exacerbations however, a recent 3-year randomised placebo-controlled multicenter trial has revealed that N-acetylcysteine is ineffective at preventing lung function decline and exacerbations in patients with COPD controlled multicenter trial has revealed that N-acetylcysteine is ineffective at preventing lung function decline and exacerbations in patients with COPD [28].

TREATMENT FOR EXACERBATION OF COPD

COPD flare-ups damage health status and are harmful. Exacerbations occur more often in individuals with moderately severe illness (FEV1 50% expected), who have 1-2 exacerbations year. Exacerbations in people with mild COPD are characterised by worsening dyspnea, coughing, and sputum production and are frequently treated outside of the hospital. Exacerbations of severe COPD are frequently accompanied by respiratory failure, which can be deadly and necessitate hospitalisation. Bacterial and viral Infection, which is present in around 50% of patients, is one of the frequent causes of exacerbations. Exacerbations may also be caused by additional elements like pollution and temperature.

The intensity of the symptoms, the severity of the underlying COPD, and the patient's capacity to manage at home all play a role in whether an exacerbation should be treated at home or in a hospital. When two or more of the following symptoms are present, such as increased breathlessness, rising sputum volume, or increasing sputum purulence, appropriate antibiotic therapy is typically administered as well as increasing bronchodilators, if necessary, administered by nebulizer.During a COPD exacerbation, systemic glucocorticoids speed up recovery time and help individuals with moderate to severe exacerbations of the disease. Although the ideal dosage and length of therapy are unknown, giving 30 mg of prednisolone for 7–10 days seems like an acceptable compromise. The management of respiratory failure and related co-morbidities is a major emphasis of hospital care. When nurses are properly educated, non-invasive positive pressure ventilation (NIPPV), which has been found to be an effective alternative to intermittent positive pressure ventilation (IPPV) in the ICU, may be used in the general ward [31].

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