EDITORIAL

EXACERBATION OF COPD

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Exacerbation of COPD can be defined from patient prospective as changes in Symptoms beyond usual day to day variability. This may include new or increased sputum purulence or volume with increased breathlessness. Unexplained fever sore throat, nasal discharge, increased cough or with recent exposure to noxious agents may accompany or cause COPD exacerbation.

Bacterial, viral, atypical infections as Mycoplasma pneumonia, Chlamydia pneumonia have been implicated. Bacterial load should rise above 106 colony forming units per unit of airway secretions to be of significance. Air pollution (Sulfur dioxide, nitrogen dioxide, Ozone, particulate matter, current smoking) and co-morbid diseases will influence the course of exacerbation. Sputum purulence will determine antibiotic prescription. Many exacerbations are preceded by viral infections and the respiratory syncitial virus is the most relevant; secondary bacterial super infection may usually occur. The presence of neutrophils in sputum suggests bacterial infection; if eosinophils are present the presence of asthmatic component should be suspected and anti – asthma therapy should be considered in the treatment.

Exacerbations of COPD cause deterioration in pulmonary functions, mainly decreased inspiratory capacity, hyperinflation, decrease in arterial oxygen saturation, and increase in carbon dioxide arterial tension; arterial PH may not show acidosis as a result of raised bicarbonates from renal compensation despite the rising carbon dioxide. Pulmonary arterial pressure may rise and precipitate right heart failure. Owing to decrease venous return as a result of dynamic hyperinflation impaired left ventricular filling occurs in severe exacerbations.

There is shortening of diaphragm and respiratory muscles during exacerbations leading to muscle dysfunction and fatigue. With dyspnoea there is increased respiratory and heart rate, central cyanosis and signs of hypercapnia such as confusion, tremors and warm peripheries, increased work of breathing by accessory muscles of respiration, breath sounds are reduced , wheezing may not be heard because of hyperinflation.

The manifestations of impending respiratory failure include increased respiratory rate > 30 per

minute, increased heart rate > 120 beats per minute, decreased level of consciousness and inability to complete a sentence while talking. Impaired consciousness or confusion should be considered a dangerous sign of impending respiratory failure.

Pneumothorax, pneumonia, pulmonary embolism, pulmonary eodema and heart failure should be excluded in all patients. Measurement of pulmonary functions (FEV1, PEFR) have not been validated in the management of exacerbations in COPD.

It is more valuable to measure arterial blood gases and arterial PH to identify patients in need of controlled oxygen therapy or non invasive ventilation.

Inhaled short acting bronchodilators are the corner stone for treatment. Short acting bronchodilators are better administered via a nebulizer by giving high doses; if not available MDI up to 3 times per hour is used.

Long acting anticholinergic if given by inhalation once daily improves airflow limitation, reduces air trapping, increases activity and reduces number of exacerbations.

Prednisone 30-40 mgm daily, should be started early and for 1-2 weeks especially in all hospitalized patients. It is advisable to give antibiotics especially in patients having green sputum (purulent) or with a positive sputum culture. The need for antibiotics is reduced by 50% based on observing sputum colour alone even without waiting sputum cultures.

Controlled oxygen therapy is essential (24% O2) given by venturi mask, if this is unavailable the use of nasal prong [1L / min = 24% and 2L / min = 28%]is an alternative to prevent rise in Paco2 and improve arterial O2 tension and maintain O2 saturation between 85-92%

If a good response is not achieved, pulmonary embolism, heart failure or pneumonia should be looked for. Theophylline at subtherapeutic level has an anti inflammatory action. Its routine use in nonacidotic exacerbation is doubtful as it has a narrow therapeutic index and if given in high dose it's side effects may be more than its bronchodilator action; In stable COPD there is a trend to accept it in low dosage. If the patient is not improving and increasing fatigue occurs with rising CO2 tension and confusion and low arterial PH < 7.35 despite maximum care with other therapies mentioned, non invasive ventilation should be considered; if there is no improvement in the initial 4-6 hours invasive ventilation should be considered in intensive respiratory care unit.

Weaning of patients from ventilators can be difficult; however non invasive ventilation may be of help.

Mucolytics by decreasing mucus viscosity and plugging of airways are beneficial but enough data are lacking to support its use during exacerbation. Chest physiotherapy is more effective in stable COPD. Fluid and electrolyte balance should be followed and prophylaxis against venous thrombosis should be given unless contraindicated. After hospital discharge, O2 home therapy might be needed and rehabilitation with physiotherapy is recommended.

REFERENCES

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