CHRONIC OBSTRUCTIVE PULMONARY DISEASE-CURRENT STATUS AND MANAGEMENT

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There has been a lot of confusion on the terminology and definition of chronic obstructive pulmonary disease (COPD). Essentially, it is a chronic pulmonary disorder characterized by airway obstruction. It has been aptly described as chronic airway disease (COAD) by different investigators. But none has fulfilled the criteria to comprehensively describe the disease. On the other hand, a few other conditions continue to be misclassified under COPD. Bronchial asthma, for example is a distinct disorder which is occasionally included under COPD. Similarly, "senile" and "compensatory" emphysema are misnomers rather than true emphysema.

Chronic obstructive pulmonary disease (COPD) is a term generally restricted to the two diseases, chronic obstructive bronchitis and/or emphysema, even though the chronic obstructive diseases of the lung include bronchiolitis and bronchial asthma [1]. Both chronic bronchitis and emphysema often coexist. There is so much overlap of clinical, and physiological characteristics between the two disorders that the term COPD has persisted in the text books of medicine. Chronic bronchitis is the "hypersecretory" disorder, characterized by chronic cough and spit. Emphysema on the other hand, is defined by an increase in the size of air spaces distal to the terminal bronchioles with destruction of their walls [2].

Airways obstruction, which may develop in the later stages of chronic bronchitis is attributed to the lumen encroachment of the airways due to mucous-gland hypertrophy, wall-thickening and mucosal oedema.

It has been debated if chronic bronchitis may lead to respiratory failure. It is the chronic air-flow limitation which poses risk to life as seen in longitudinal studies on a large number of working men in Britain

[3]. Therefore, chronic bronchitis may be classified as "obstructive" depending upon the absence or presence of airways obstruction [4].

Some of the types of emphysema such as paraseptal or periacinar variety, centriacinar type with dust deposition disease (coal worker's pneumoconiosis), emphysema associated with check-valve obstruction are not associated with any significant airways-obstruction. On the other hand, primary or essential emphysema of panacinar variety and that associated with alpha-1 antitrypsin deficiency is characterized by airways obstruction. There is progressive breathlessness. It is a crippling and often fatal disease [1,2].

Diagnosis

History and examination

Though the history of smoking and respiratory symptoms are common to both, the predominant disorder can often be identified. Asthma is differentiated from COPD by the episodic nature, presence of symptoms from the childhood, family history of atopy and significant relief following bronchodilator administration. In advanced stages of chronic-bronchitis, a classical "blue-boater" has cyanosis, oedema and features of CO₂ retention. A "pink-puffer" is often seen in the late stages of emphysema, has normal or low PCO₂. In clinical practice, a patient may often show overlapping features.

Pulmonary function testing

Spirometric measurements are of immense value in the differential diagnosis of COPD. While vital capacity (VC) is normal in earlier stages, FVC is often reduced in emphysema. FEV₁ is an accurate estimate of the risk of premature morbidity and mortality from COPD [5]. Reduction of the FEV₁/FVC ratio is useful in the diagnosis of mild disease [6]. Most of the expi-

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ratory flows are low in all the obstructive disorders. Reduction of only the midexpiratory flow rates (i.e. FEF₂₅₋₇₅, FEF₅₀) but normal FEV₁ and FEV₁/FVC ratio may indicate mild or peripheral airways disease.

Other lung volumes such as RV, functional residual capacity (FRC) and total lung capacity (TLC) are increased in COPD as well as asthma. Measurement of lung compliance and distensibility (K) have poor correlation with pathological changes of emphysema. Diffusion capacity (DLCO) is almost always reduced in emphysema, sometimes in chronic bronchitis but normal (or elevated) in asthma [7,8].

Early small airways obstruction can be measured with the help of sensitive tests such as closing volume, density dependent (helium-oxygen) flow curves and frequency dependency of compliance. But they bear no relationship with clinical course of the disease.

Measurement of arterial PO₂ and PCO₂ are important in later stages of COPD for management and recognising patients for long term oxygen therapy.

Chest roentgenography

In chronic-bronchitis, chest X-ray may be normal or show non-specific signs, such as linear and nodular densities, peribronchial cuffing or thickened walls (seen as tram-lines). Chest radiographic accuracy in diagnosis of asthma has been different in different reports [9,10]. The signs of hyper-inflation include hyper-translucency, low flat diaphragms, tubular-heart and an increase in retrosternal air-space in a lateral view film. Radiographic demonstration of lung destruction, bullae formation and vascular attenuation i.e. rapid tapering and narrow angle branching are more specific findings of emphysema. Signs of pulmonary hypertension and right ventricular failure are present when there is chronic cor pulmonale development.

Other imaging modalities

Computer tomography (CT) scan is quite sensitive in diagnosing emphysema [11] there is reduced tissue density, "pruning" of pulmonary vessels and bullae formation. The physical density of "pixels" forming the lung images may be quantitated and the image-density computed for the diagnosis especially in high resolution CT. Air wall thickening and inflammation may also be demonstrable in high resolution CT.

Magnetic resonance imaging (MRI) offers no help in the diagnosis of COPD. Radioisotope scans are sometimes useful to demonstrate bullae but their role is also limited.

Management

There are five important aspects in managing COPD. These are tobacco cessation; pharmacologic therapy; pulmonary rehabilitation; longterm oxygen therapy and surgery.

Tobacco Cessation

Since most of COPD patients are smokers, quitting smoking is the most important step in management. Any form of therapy proves futile in the presence of continued smoking. Counselling is generally helpful although nicotine substitution may be required in some patients. Nicotine chewing gum and patches are available as the substitutes. Professional psychiatric help may occasionally be required. Failure to quit is frequent but fortunately the failure decreases with each new effort at quitting.

Pharmacologic therapy [12]

The aim is to relieve symptoms of cough, expectoration and breathlessness and treat the reversible factors, such as infection, heart failure, or cardiac arrhythmias.

Bronchodilators are the mainstay for symptomatic relief. For early stages, oral theophylline and/or inhalational beta agonists are generally adequate. Long acting preparations are more convenient. Oral sympathomimetics alone or in combination, can be administered, but may cause tremors and other side effects. For moderately advanced cases, same drugs are used, often together, in increased dosage and frequency-schedule. Ipratropium, if available, may be added alongwith. Cough suppressants are used for unproductive, irritating cough.

For unrelieved bronchospasm, oral (or occasional parenteral) steroids may be required. After an initial dose of 40 to 60 mg for a few days, it is tapered to 7.5 to 15 mg a day. Inhaled steroids may be used to substitute maintenance oral dose. For acute exacerbation, parenteral or nebulized bronchodilators, mucokinetic drugs (e.g. acetylcysteine or bromhexine) and antibiotics are used. The choice is general for a broad spectrum antibiotic depending upon the clinical setting. For chronic cor pulmonale, continuous O₂ therapy, pulmonary vasodilators and diuretics are used. Low dose digoxin is useful if there is evidence of left heart failure. Respiratory stimulants are used for progressive hypercapnia to buy time.

Narcotics and sedatives should be avoided. Occasionally, these drugs may be used in reduced dosages for symptomatic relief of unalleviated cough or dyspnoea. Other psychoactive and anxiolytic drugs are re-

quired in patients with depression, anxiety and sleep disturbances. Extreme caution should however, be maintained with the use of these drugs.

Nutritional support

There is an altered metabolic stage and increased energy demand in COPD. This results in malnutrition and weight loss resulting in a wasting syndrome. Caloric and protein supplementation is extremely important during exacerbations as well as the maintenance phase.

Pulmonary rehabilitation

Rehabilitation comprises a multidisciplinary individually-tailored approach, consisting of accurate diagnosis and treatment, education of the patient and family members, respiratory and physical therapy, exercise-conditioning, psychological support and vocational-rehabilitation [13]. The programme help in reduction of symptoms and improving the quality of life. patient's life-style and independence are maintained as near to normal as possible. Hospitalization needs are decreased and return to work is made possible [14]. There is also a possible prolongation of survival.

The comprehensive respiratory therapy includes the use of aerosols and oxygen at home, breathing and relaxation exercises, maintaining bronchial hygiene by chest percussion, postural drainage, forced coughing and intensive spirometry. The experience with mechanical ventilators at home for assisted ventilation is now available in this country as well, though it remains a costly proposition [15].

Long-term oxygen therapy

The two landmark oxygen therapy trials, one each from U.K. and U.S.A. have clearly shown an improved quality of life and an increased survival of COPD patients from low flow, domiciliary O₂ therapy [16,17]. The specific goal is to achieve a PaO₂ of about 60 mmHg with an FiO₂ of about 24-28 percent. The total daily duration should be determined keeping in mind the precept that survival is poor with no oxygen, better with some oxygen and best with continuous (or near continuous i.e. >18 hrs/day) oxygen. Enough experience on domiciliary oxygen is now available in this country [18].

There are three sources of oxygen for home use: i) compressed gas cylinders (most common and cheaper, but the mechanics are cumbersome), ii) liquid oxygen-costly but portable iii) oxygen concentrator-high initial cost but most useful for long term home use. Continuous-flow nasal cannulae or ventimasks are adequate for delivery. To conserve oxygen, vari-

ous devices such as reservoir cannulae, demand-valves and transtracheal catheters have been used [19].

Surgical therapy

Bullectomy is done for large bullae especially when there are signs of compression. Bullae can also be resected with lasers via thoracoscopic procedures [20]. More recently, lung volume reduction surgery (LVRS) is being practised at several centres where bilateral resection of lung apices is done to reduce lung volume and provide mechanical advantages of breathing [21]. It is essentially a palliative form of surgery. air leaks following LVRS pose the greatest problem in management.

Lung and occasionally both lungs and heart transplantations are done for end stage disease. Single lung transplantation has been shown to be both feasible and more practical [22]. This however, is a complex issue involving ethical, economical, technical and social angles. It is yet to evolve as a form of therapy in this country.

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