Chronic obstructive pulmonary disease

Introduction

It is not easy to explain to someone of what COPD is. COPD stands for Chronic Obstructive Pulmonary Disease. (Brown and Edwards, 2012) COPD, as stated by Brown and Edwards (2012), is a general term which involves both chronic bronchitis and emphysema. COPD can be defined as a respiratory condition that is progressive and unable to be cured. It is characterised by airflow limitation and irreversible obstruction (Brooke, 2013). Current statistics provided by Brown and Edwards (2012) indicate that approximately 2. 1 million Australian have some form of COPD, however, the figure will rise up to about 4. 5 million in 2050. This essay will provide a thorough explanation on how COPD develops, what risk factors that contribute to the exacerbation of COPD and the interventions, both pharmacological and non-pharmacological that help addressing its exacerbation.

Pathophysiology of COPD

Chronic bronchitis, as explained by McCance, Huether, Brashers & Rote (2010), is an excessive production of mucus and prolonged productive cough that lasts for at least 3 months of the year for at least 2 consecutive years. Hypersecretion of mucus is usually caused by inspired irritants. The irritants play an important role in increasing the size and number of mucus glands and goblet cells (responsible for mucus secretion) in airway epithelium. The mucus produced is thicker and more tenacious than normal becoming a reservoir for bacteria to rapidly grow. In chronic bronchitis, the cilia (tiny hairs that line the airways) are destroyed resulting in impaired ability of the

lungs to expel mucus. (Kaufman, 2013a) The rapid reproduction of bacteria, together with impaired ciliary function, increases vulnerability to pulmonary infection and injury. As a consequence, the bronchial walls further become inflamed and thickened due to oedema or fluid overload caused by excessive mucus production and the accumulation of inflammatory cells. Recurrent infection, multiplied by persistent inflammation, leads to bronchospasm (an abnormal contraction of the smooth muscle of the bronchi) and permanent narrowing of the airways. (McCance et al., 2010) Furthermore, bronchospasm and the narrowing of the airways lead to obstruction, especially during expiration. The obstruction of the airways leads to ventilation (the process by which gases are moved into and out of the lungs)-perfusion (the passage of blood flow through the areas of the lungs) mismatch with hypoxaemia (an abnormal deficiency of oxygen in arterial blood). Air trapping results from the airways collapse early in expiration causing the rib cage to expand. This puts the respiratory muscles at a mechanical disadvantage leading to a decrease in tidal volume (the amount of air inhaled and exhaled during normal ventilation), hypercapnia (greater than normal amounts of carbon dioxide in the blood) and hypoventilation (uneven distribution of inspired air). (McCance et al., 2010)

Emphysema is “ abnormal permanent enlargement of gas exchange airways accompanied by destruction of alveolar walls without obvious fibrosis” (McCance et al., 2010, p. 1288). Its major mechanism is loss of elastic recoil and usually initiated by inflammatory oxidants. One of the potential oxidants is cigarette smoke. It contains toxins which irritate airway epithelium and trigger the inflammatory response. The affected site is then infiltrated with

white blood cells such as, neutrophils, macrophages and lymphocytes. During the inflammatory period, enzymes such as proteases are released by neutrophils causing the breakdown of elastin in the alveolar walls. (Kaufman, 2013a) The breakdown of elastin leads to ventilation-perfusion mismatch and hypoxaemia. It also reduces elastic recoil of the airways meaning the inspired air cannot be completely expelled from the lungs. In similar to chronic bronchitis, air trapping occurs and causes hyperexpansion of the chest putting respiratory muscles at a mechanical disadvantage. This leads to an increase in work of breathing and the development of hypoventilation and hypercapnia in later stage. (McCance et al., 2010)

Clinical Manifestations

Brown and Edwards (2012) stated that " clinical manifestations of COPD typically develop slowly around 50 years of age after 20 pack-years of cigarette smoking" (p. 697). A diagnosis of COPD should be considered in any patient who presents with symptoms such as, cough, sputum production and dyspnoea (difficulty breathing), as well as, any patient who have a history of smoking and exposure to air pollution. (Brown and Edwards, 2012)

Apart from smoking and exposing to air pollution, occupational environments in which there is intense and prolonged exposure to irritant fumes, dust and gases, together with an inherited deficiency of the glycoprotein alpha $_1$ -antitrypsin, are also associated with the development of COPD. (Kaufman, 2013a) Alpha $_1$ -antitrypsin helps protecting lung tissue from the enzymes, called proteases, produced by inflammatory cells, neutrophils in particular. (Kaufman, 2013a)

A chronic and intermittent cough usually occurs in the morning. This may or may not be associated with the production of mucus. Dyspnoea usually occurs with exertion, such as exercising, and gradually interferes with activities of daily living. The more elastin in the alveolar walls collapse, the more air is trapped in the lungs. Overextending lungs cause the flattening of a diaphragm increasing anterior-posterior diameter of the chest. As a consequence, the barrel chest is formed following with an ineffectiveness of abdominal breathing. This helps explain why patients with COPD become more of a chest breather and rely greatly on the intercostal and accessory muscles. (Brown and Edwards, 2012)

Wheezing and chest tightness may be present and may vary depending on time of the day. Weight loss and anorexia, though adequate kilojoule intake, are common in patients with advance COPD. Fatigue plays a key role in affecting patients' activities of daily living. Furthermore, haemoptysis can occur during respiratory tract infections. (Brown and Edwards, 2012)

Impaired ciliary function and excessive production of mucus are the indications of a prolonged expiratory phase of respiration wheezes or decreased breath sounds, which are noted in all lung fields during physical examination. Difficulty breathing can be clearly manifested when patients use accessory muscles on inspiration, purse lips on expiration and assume tripod positioning. Hypoxaemia may develop later in the disease with hypercapnia. As the body tries to compensate for chronic hypoxaemia, more red blood cells are rapidly produced resulting in bluish-red colour of the skin. (Brown and Edwards, 2012)

Nursing interventions

According to the case study, Mrs. Neumann presents with 3 days of worsening dyspnoea and increased frequency of coughing which is now productive of green, purulent sputum. The interventions, with prioritisation, are provided in order to address ineffective airway clearance and impaired gas exchange.

First of all, ineffective airway clearance is commonly related to a number of factors include, increased size and number of mucus glands, increased mucus production, thick secretions, decreased ciliary function, alveolar wall destruction, decreased energy and fatigue, impaired exhalation, bronchospasm and smoking. (Gulanick and Myers, 2011) The administration of both bronchodilators and corticosteroids, accompanied by the use of effective coughing techniques, is the intervention for ineffective airway clearance. The only way to relieve the exacerbation of COPD is to administer both short acting bronchodilators and corticosteroids, such as Prednisolone. These help open the air passage by relieving bronchoconstriction and reduce swelling and inflammation in the airways. As a consequence, it will be easier for Mrs. Neumann to breath. Moreover, coughing is the most helpful way to remove most secretion. Assisting Mrs. Neumann with effective coughing techniques help mobilise secretions from smaller airways to larger airways making coughing up sputum much easier to do. (Gulanick and Myers, 2011)

Secondly, impaired gas exchange is commonly related to the loss of lung tissue elasticity, residual volume has increased, the overproduction of secretions along bronchial tubes and bronchoconstriction. (Gulanick and

Myers, 2011) Promoting more effective breathing pattern is the intervention in improving impaired gas exchange. This can be done by instructing Mrs. Neumann to position herself correctly, teaching the patient to pursed-lip breathing and to use abdominal in combination with other accessory muscles. Sitting upright and high-fowler's position help expanding the lungs and push the diaphragm downward resulting in optimal breathing. Pursed-lip breathing promotes positive airway pressure during exhalation. This technique decreases carbon dioxide retention and breath rates, allowing tidal volume to increase and prolonged diffusion between oxygen and carbon dioxide, and increases alveolar ventilation. This technique also helps alleviate shortness of breath on exertion. In addition, the movement of diaphragm is facilitated by using the abdominal muscles and chest excursion can be increased by using the accessory muscles. (Gulanick and Myers, 2011)

Pharmacology Intervention

National Institute for Health and Care Excellence (2010) recommended the use of short acting beta $_2$ agonists, especially Salbutamol, as the first line bronchodilator therapy for the management of COPD. Short acting beta $_2$ agonists, in general, have its onset within five to ten minutes post inhalation and last for three to six hours. (Tiziani, 2010) Inhaled beta $_2$ agonists, as stated by Kaufman (2010), are, by far, the best way to provide immediate access to the airways. Kaufman (2013b) explained that Salbutamol has an effect on beta $_2$ receptors in bronchial smooth muscle and initiates the production of cyclic adenosine monophosphate. These effects not only help dilate the smooth muscle of the bronchi but also help relax the walls of the

airway smooth muscle. These further increase the amounts of air and oxygen to reach the alveoli. (Kaufman, 2013b) Symptoms such as difficulty breathing or breathlessness and exercise limitation in COPD can now be effectively relieved by the use of Salbutamol. The clearance of the mucus associated with the condition is also improved. (Kaufman, 2013b)

National Institute for Health and Care Excellence (2010) recommended the use of short acting muscarinic antagonists or anticholinergic agents, especially Ipratropium, as a substitute for, or in combination with, short acting beta $_2$ agonists in the management of COPD. Anticholinergic agents have its onset within thirty to sixty minutes post inhalation, which is slower than short acting beta $_2$ agonists, and last for approximately six hours. (Kaufman, 2013b) This type of drugs is given through the inhaled route in order to lessen the quantity of drug that reaches the systemic circulation and minimise side effects. (Kaufman, 2010) “ Combination therapies of short acting beta $_2$ agonists and anticholinergic agents have traditionally been given to control breathlessness in COPD” (Kelly, 2009, p. 6). Muscarinic or cholinergic receptors are found in airways smooth muscle. They are influenced by the neurotransmitter acetylcholine that is responsible for the resting tone of bronchial smooth muscle. Bronchoconstriction subsequently occurs when the resting tone has increased. (Kaufman, 2013C) Ipratropium, on the other hand, blocks the acetylcholine or cholinergic receptors in order to prevent bronchoconstriction. In addition, Ipratropium is slightly more effective than short-acting beta2 agonists in improving lung function and health status and reducing the need for oral corticosteroids. (Kaufman, 2013C)

It is recommended that the combination of short acting beta $_2$ agonists, given at high doses and increased frequency of use and up to two weeks of therapy, and oral corticosteroids, such as Prednisolone, is very helpful in managing the exacerbation of COPD. (Bullock and Manias, 2011) As mentioned in the pathophysiology section, COPD is greatly associated with chronic inflammatory response. Of that, corticosteroids, well known for its anti-inflammatory effects, are considered to be used as one of the first line treatments. This type of drugs inhibits the rupture of mast cells (responsible for inflammatory process), diminishes the synthesis of inflammatory mediators, stops the production of new antibody and suppresses the activity of immune cells, especially lymphocytes and macrophages. These result in the reduction of oedema, mucus production and bronchoconstriction. (Bullock and Manias, 2011)