REVIEW ARTICLE

Respiratory Failure in Intensive Care Unit Patients with Progressive COPD: Nursing Approaches to Patient Care

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a set of heterogeneous diseases characterized by a not entirely reversible and in most cases gradual restriction of expiratory flow. Tobacco smoke is a common risk factor for the development of COPD, but the effects of coming into contact with indoor air pollutants are also significant, and the exacerbation of COPD is one of the most significant reasons for intensive care unit (ICU) admission.

Registered nurses play a key role with regard to dealing with the adverse events associated with respiratory dysfunction. They track the condition of patients through their physiologic activity. The clinical treatment provided by nurses depends on the cause and type of insufficiency present in patients with respiratory failure. The aim of this study was to understand how emergency nurses could better develop their roles in ICU patients with respiratory failure due to COPD. There are many different interventions for different causes of respiratory failure. The clinical measures of respiratory dysfunction, such as changes in respiratory velocity, and occurrence of dyspnea, hypoxemia and acidosis are significant factors in the diagnosis of respiratory dysfunction and the evaluation of the risk for adverse events. Thus, nursing aims include avoiding hypoxia, reducing hypercapnia-associated acidosis and reducing complications and agitation in patients with respiratory failure due to COPD. (*Altern Ther Health Med.* 2022;28(1):52-57).

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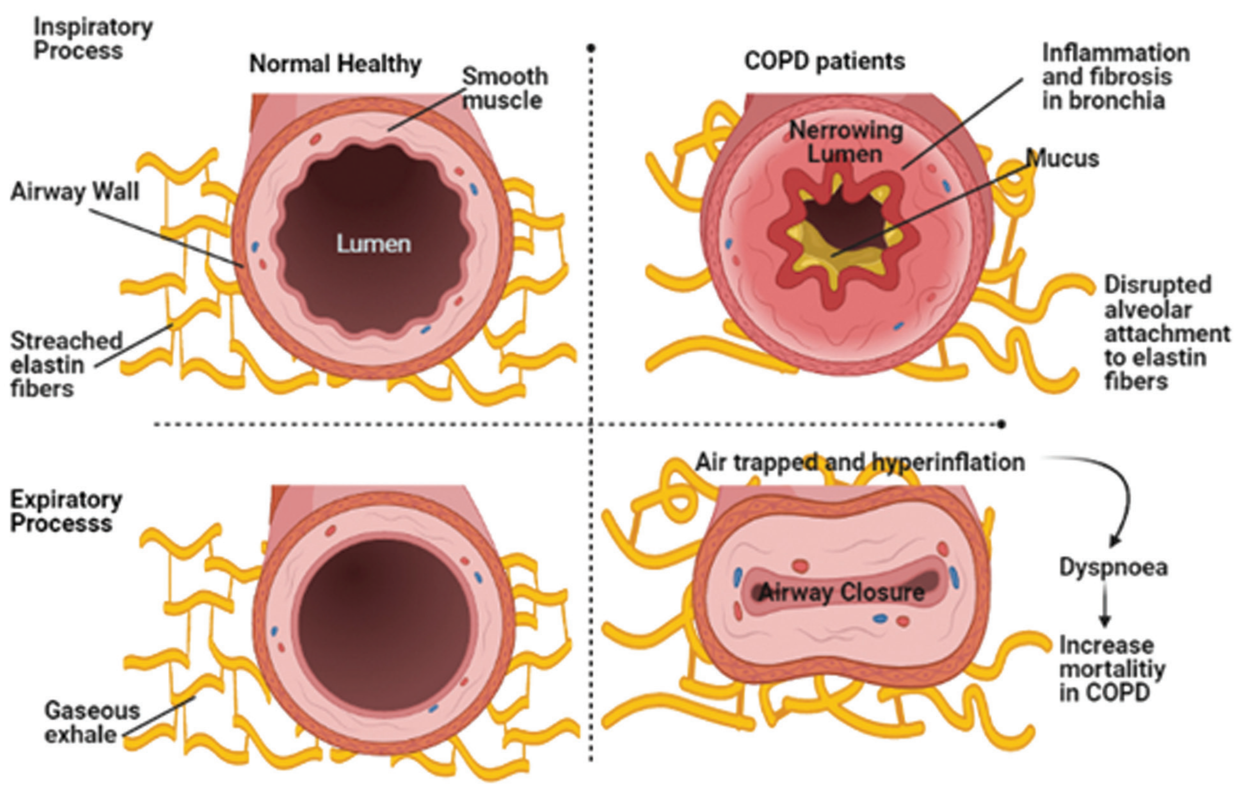
INTRODUCTION

Chronic pulmonary obstructive disease (COPD) has a significant impact on health worldwide and globally kills more than 3 million people annually.[1] The World Health Organization (WHO) complies the global burden of the disease based on the most detailed statistics on the global distribution of COPD. The greatest percentage of COPD deaths occur in East and South Asia; these 2 areas have the highest age-standard mortality rates from COPD. Despite the smaller numbers of patients with severe and extremely severe COPD relative to its milder manifestations, the early stages of the disease require the vast majority of healthcare services[2] due to excessive hospitalization, exacerbations and mortality rates.[3]

The significant percentage of adverse outcomes is apparently followed by a time in which the patient has distinctly irregular clinical symptoms. Respiratory failure is also a significant complication of COPD and hospitalization, with a poor prognostic predictor for an acute episode. In extreme cases, these patients will deteriorate rapidly and must be closely monitored. Mechanical ventilation is an effective tool for reducing blood carbon dioxide levels. Patients in intensive care units (ICUs) with serious breathing problems should be treated in a compassionate way, according to Surviving Sepsis Campaign recommendations.[4] The ICU and emergency room (ER) nurses have the most contact with the affected patients, and will focus on the cause and type of impairment present in patients with respiratory failure.

The aim of this study was to understand how ER nurses could better develop their roles in the ICU with regard to respiratory failure due to COPD. There are many different interventions for different causes of respiratory failure. The clinical measures of respiratory dysfunction, such as changes in respiratory velocity and occurrence of dyspnea, hypoxemia and acidosis are significant factors in the diagnosis of respiratory dysfunction and evaluation for risk of adverse events.[5] Thus, the clinical goals of nurses are to prevent

Figure 1. Exploring the air-trapping process in COPD that increases mortality in ICU patients.



hypoxia, reduce hypercapnia-associated acidosis and improve symptoms and anxiety of patients in respiratory failure due to COPD.

LITERATURE REVIEW

Technically significant results were obtained from English-language publications in Springer ScienceDirect, Medline, Google Scholar, PubMed and Mendeley. Multiple keywords were used: chronic obstructive pulmonary disease, epidemiology of respiratory failure pathology in COPD, involvement of cytokines in COPD, factors associated with COPD, pathophysiology of COPD, approaches to treat COPD, changes lung morphology in immunomodulatory reaction and nursing interventions in the healthcare treatment of ICU patients with respiratory failure. Reference lists were also screened for corresponding articles not included in the initial search.

PATHOPHYSIOLOGY OF RESPIRATORY FAILURE IN COPD

While cigarette smoking is the primary environmental risk factor for COPD, nearly one-third of patients with COPD worldwide are non-smokers. Other environmental substances such as biomass fuel used for cooking and heating are also important environmental risk factors for COPD in many parts of the world,[6-8] and there is evidence that dusty environments are associated with a risk for COPD.[9] The next most important risk factor is a history of tuberculosis.[10] In addition, comorbid abnormalities in patients with COPD are particularly common, but mostly not significant in pulmonary function.[11] Thus, COPD can be regarded as the pulmonary portion of a systemic and multidimensional disorder.[12]

Although the pathways leading to COPD remain unclear, the condition is typically associated with corticosteroid-resistant chronic inflammation. Furthermore, COPD indicates rapid pulmonary aging and an irregular repair process driven by oxidative stress. Acute exacerbations are significant since they are related to a poor prognosis, and are often caused by viral or bacterial infections.[13-15] Thus, it is important to better understand the complex disease pathways that contribute to COPD and lead to respiratory failure (see Figure 1).

The traditional theory is that smoking triggers an abnormal inflammatory reaction in vulnerable people[16] that damages the airways (bronchitis, bronchiolitis) and alveoli (emphysema). Physiologic lung function is worsened and contributes to limited airflow and recurrent respiratory symptoms that are difficult to reverse and may reoccur regularly as exacerbations.[17-19] The intensity of COPD airflow restrictions is related to the extent to which neutrophils, macrophages and lymphocytes infiltrate the lung tissues. In extreme COPD, tertiary lymphoid lymphocytes are produced, indicating an adaptive immune response. The numbers of CD4+ T helper 1 and CD8+ cytotoxic T cells in lung tissue increase during this adaptive immune reaction.[20] The number of CD4+ T helpers 17 cells is increased in the lungs, and neutrophilic inflammation can be further amplified.[21] In COPD, high levels of some inflammatory mediator—such as lipid and peptide mediator and cytokines and chemokines—are stored and inflamed and circulatory cells are recruited into the lungs.[22] The vast array of inflammatory mediators is regulated by pro-inflammatory transcription factors, including NF-κB and MAPK, and the mitogen-activated protein kinase (MAPK) family, with a particular emphasis on p38 MAPK.[23-25] Airway remodeling involves thickening of the airway walls (epithelium, lamina propria, smooth muscle and adventitia) of airways smaller than 2 mm in diameter. Researchers using Micro-CT have observed that terminal and transitional bronchioles are decreased by 40% in mild-to-moderate COPD and by 80% in extreme-to-very severe COPD.[26] Loss of alveolar spaces—resulting in emphysema—is another significant characteristic of COPD. The main pathogenic mechanism in emphysema is initially a distortion of protease and antiprotease activity caused by pulmonary infiltration via active neutrophils, reduced antiprotease activities, or both; the classic example is alpha 1 antitrypsin deficiency.[27] In addition, numerous protease-destroying elastin fibers are eliminated from neutrophil, macrophage and epithelial airway resident cells in patients with COPD. In larger airways, elastases from neutrophils induce hypersecretion of mucus, while in the lung tissue, MMP9 and MMP12 may be essential in the elastolysis that has been observed in patients with emphysema. In recent years, more variables such as accelerated apoptosis and pulmonary maintenance impairment, as well as oxidative stress, autoimmunity, malnutrition or a combination have been suggested.[28-30] Patients with COPD also undergo invasion of the lower respiratory tract with bacteria like *Streptococcus pneumoniae* and *Haemophilus influenzae*. This chronic bacterial colonization is related to a defect in bacterial uptake by macrophages (phagocytosis).[31]

Autoimmune mechanisms can also play a role in the durability of bacterial infections, and there is evidence of the existence, at least in serious illnesses, of autoantibodies in the lungs of individuals with COPD such as endothelial cell antibodies and antibodies to carbonyl-modified proteins. In general, the smoking-induced injury repair process begins with coagulation system activation, which initiates a damage control response required to stop bleeding.[32] In addition, the inflammatory immune cells, mostly neutrophils or macrophages, are infiltrated to avoid infection from injury and take part in a demolition process that destroys the dead and injured tissue.[32] Subsequently, myofibroblasts, endothelial precursor cells and fibroblasts appear and create a provisional matrix in order to allow the microvascular network to reconnect and support the restoration of the epithelial surface.

Injuries caused by repeated damage such as from smoking induce a more complex tissue repair process that combines tissue destruction with scar formation.[33] However, a shift in the small pulmonary arteries caused by inflammation and vein constriction in COPD is common; such changes can also be caused by oxygen deficiencies in the arteries, which may contribute to smooth muscle proliferation and thickened internal vasculature.[34] Pulmonary hypertension is typically not marked in COPD, and is primarily seen in a small number of patients with disproportionate pulmonary hypertension who may experience right heart failure.[35]

Finally, peripheral lung inflammation found in COPD can "spill" into systemic circuitry and lead to systemic COPD inflammation associated with various comorbidities, such as respiratory failure.[36] The respiratory system has pathophysiologic features and consists of 2 primary compartments: the lung as a gas exchange unit and the ventilatory pump, which controls the device. Functional lung dysfunction (type I respiratory failure) contributes mostly to arterial hypoxemia along with average or lower $PaCO_2$ levels owing to compensatory enhanced ventilation. In contrast, ventilation pump failure (type II [hypercapnic]), due to an elevated PCO_2 level, sometimes synonymous with hypoxemia, is a consequence of mechanical disadvantage (such as lung hyperinflation in COPD), central nervous system anomalies or respiratory muscle malfunction.[36-38]

FUNDAMENTAL TREATMENT STRATEGY FOR RESPIRATORY FAILURE IN COPD

Symptoms

An individual with COPD experiences long-term, progressive damage to their lungs affecting airflow, sometimes called emphysema or chronic bronchitis. Symptoms include fast, shallow breathing, as if the patient just performed intense exercise; coughing; shortness of breath at rest or with minimal activity, such as walking from one room to another; feeling excessively sleepy or confused; having lower oxygen levels than normal; noticing increasing amounts of mucus, which is often yellow, green, tan, or even blood-tinged; and wheezing more than usual.

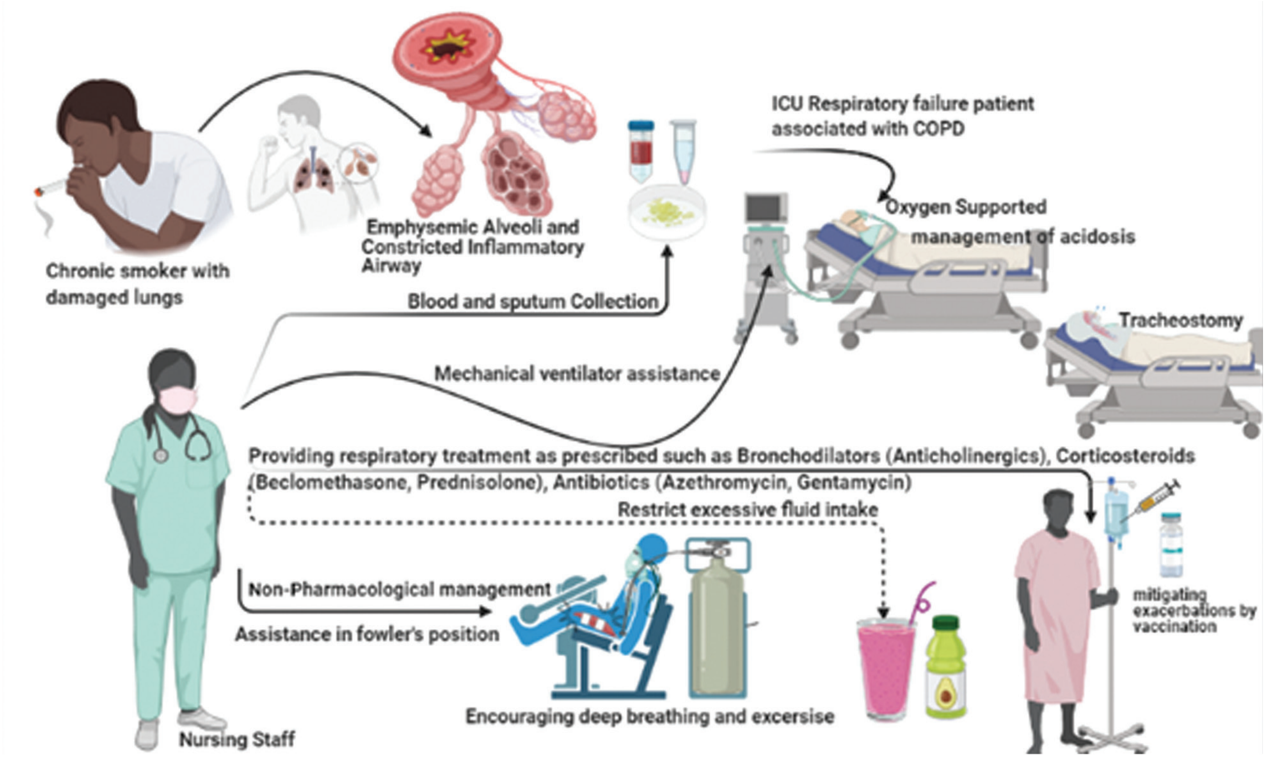
Prophylactic Treatment

The prophylactic approach for respiratory failure in COPD naturally includes stopping or minimizing worsening of illness and preventing or mitigating exacerbations via vaccination;[39] bronchodilators including adrenergic agonists, anticholinergic agents and xanthin derivatives;[40] anti-inflammatory therapy involving inhalation and systemic steroids;[41] or a combination.[42]

Exacerbations

Exacerbation management involves oral antibiotics including amoxicillin or doxycycline if evidence exists for an elevated purulence and amount of sputum.[43] Short courses of oral corticosteroids have also been used based on their individual strength, and new research indicates that shorter courses (5 days) of corticosteroids can be as beneficial as long courses, such as the more traditional 14 days.[44-46] Some data indicate that treatment with oral corticosteroids in patients with elevated blood eosinophils numbers during an exacerbation is more successful, but more studies are needed to validate these results.[47]

Figure 1. Nursing assistance for ICU patients with COPD-associated respiratory failure.



Corticosteroids

Inhaled corticosteroids (ICS) are commonly used for the treatment of asthma and COPD, although their effectiveness for the latter remains controversial. Nevertheless, these drugs are now widely used in COPD at high doses, with patients frequently receiving the equivalent of 1000 µg of fluticasone per day. High doses have been associated with significant systemic effects such as pneumonia, glaucoma, cataracts, adrenal suppression, accelerated bone turnover and diabetes. Patients who require prolonged high-dose corticosteroids are at risk for systemic adverse events, particularly immunosuppression and adrenal suppression. Patients who are using high-dose corticosteroids should be advised to inform the healthcare team responsible for their treatment if they become ill for any reason, as this may affect the dose required. An analysis suggested that 1000 mcg of inhaled fluticasone propionate was approximately equivalent to 10 mg of oral prednisolone and at this dose, half the patients were sufficiently suppressed to be unable to mount the necessary adrenal response to stress. Therefore, all patients taking an oral corticosteroid for more than 3 weeks or prolonged high-dose inhaled steroids should have the dose tapered gradually.

When respiratory failure is unavoidable or exacerbated by the previous situation, it is strongly advised that gas exchange be sustained or enhanced with additional non-pharmacologic alternatives, particularly the supply of oxygen. The aim of this process is essentially to increase the percentage of inspired and ingested oxygen to eliminate and reduce the occurrence of acute or chronic hypoxia. Ventilation assistance must be considered as a second major alternative in order to improve alveolar ventilation and/or alleviate mechanical stress. This can be done by either invasive or non-invasive procedures in the ICU intensive care unit and emergency room (see Figure 2).

NURSING CARE FOR MANAGEMENT OF RESPIRATORY FAILURE IN PATIENTS WITH PROGRESSIVE COPD IN THE ICU

The treatment of respiratory failure in COPD is etiologically distinct. Inhaled bronchodilators are central to COPD treatment, and it is becoming evident that certain patients may struggle to reach optimal peak inspiratory flow (PIF) with some inhalers. As a result, both the mode of delivery and the type of inhaler are important considerations in COPD treatment. Device selection should be informed by the patient's needs, preferences and abilities. For instance, patients who find it hard to reach optimal PIF may benefit from an inhaler that requires less inspiratory effort to activate. Primary care practices should use multiple educational and training methods (eg, verbal, visual, demonstration) to instruct patients on proper inhaler technique. Instruction and review should be repeated at every office visit to ensure effective inhalation and drug delivery to optimize therapeutic outcomes.

Nursing staff must understand respiratory physiology and the pathophysiologic mechanism of respiratory failure in order to treat these patients. ICU patients are vulnerable to risk for infection due to their lethality, compromised protective mechanisms and extended duration of hospitalization, particularly those patients undergoing intubation and requiring ventilation-supported breathing.[48]

Breathing System Regulation

The respiratory system has a vital role in retaining basic human mechanisms, which make breathing system regulation the primary indicator for successful treatment in the ICU.[49-51] Other than pharmacologic intervention, using oxygen and mechanical ventilation is a beneficial tool in recovery. Oxygen therapy is one of the major ICU medical therapies.[52] Respiratory treatment includes suction of the airways, oral care, oxygen breathing and ventilation and protection against ventilator-related pneumonia (VAP).[53] Many studies have revealed that ICU respiratory disabilities can cause comorbidities.[54] Nurses are more involved than most members of the medical profession in hospital and patient care systems, and precise patient respiratory treatment is one of the pillars of nursing in these units due to the fragile and critical nature of the ICU.[55]

EFFECTIVE NURSING

Effective nursing requires a number of specific conditions: discipline, knowledge and experience and engagement to provide adequate treatment and commitment to life-saving or end-of-life care,[52] and comprises a significant portion of the national healthcare workforce. Nurse healthcare has a direct impact on the quality of nursing care.[56] In chest physiotherapy, the nurse has a key role to play in a number of common operations such as thoracic squeezing, strengthening of the expiratory cage and chest walls, and special maneuvers such as positioning, manual hyperinflation and suction. In terms of respiratory symptoms, several research studies have demonstrated that chest physiotherapy has beneficial effects, including reduced ventilator-associated pneumonia, increased airway secretion removal, rapid mechanical ventilation weaning and accelerated ICU discharge.[57]

Nurses are necessarily not only responsible for proper oxygen management, but also for other essential health services that support these patients such as care, oxygen saturation monitoring and vital signs, to improve the ICU respiratory failure status of patients.[58-60] In this review, nurses are playing a key role in 5 areas: suction, hand sanitation, posture change involved various roles for the chest, chair-sitters and semi-sitting and sitting positions and endotracheal cuff treatment were used in the therapies), oral care and avoidance of respiratory equipment pollutants, and VAP prevention, and the highest total score applicable to the prevention of breathing equipment pollution. Of note, other options may also be used as effective adjuncts to the treatment of ICU patients, such as non-pharmacologic therapy, acupressure, psychological hypnotherapy, listening to music, etc.61 In addition, respiratory babies play an important role in patient education, improved self-control and management.[58]

CONCLUSION

This condition is still not well known by the general population or general medicine physicians and practitioners outside of pulmonary medicine, despite its high prevalence, morbidity and mortality. This lack of knowledge is attributed in part to an incomprehensible disease name and insufficient identification of the disease itself as many diseases can lead to fixed airway obstruction syndrome. Furthermore, a multidisciplinary, coordinated approach is required to treat acute respiratory failure and other respiratory illnesses. Nurses play a vital role in mitigating or avoiding adverse conditions that can occur due to respiratory failure. Nurses have the most important part to play in assessing respiratory distress risk and tracking and monitoring/managing a patient's condition during their hospitalization. The diagnosis and treatment of the fundamental cause of respiratory failure is difficult since different disorders can cause respiratory failure. Furthermore, it might be appropriate to consult with pulmonary medicine doctors. More research that explicitly explores the impact of nursing assessment and action on the medical condition of patients and the frequency and seriousness of respiratory failure is needed.

CONFLICT OF INTEREST

None.

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