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Diagnosis and Treatment of Postoperative Dyspnea: Literature Review

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ABSTRACT

Postoperative dyspnea is one of the most common complaints in patients after undergoing surgery. Especially in those who underwent surgery in the thoracic area. The presence of postoperative dyspnea indicates clinical signs of postoperative pulmonary complications. There are several types of diseases that can cause shortness of breath in patients after surgery. Several postoperative lung diseases can cause complaints of dyspnea such as postoperative pleural effusion, atelectasis, pneumothorax, bronchospasm, aspiration pneumonitis, pneumonia, acute respiratory distress syndrome, pulmonary edema, and pulmonary embolism. The pathophysiology of postoperative dyspnea can occur due to changes in homeostasis due to anesthesia and the surgical procedure itself. Clinical symptoms arise due to compensatory efforts of the cardiovascular and respiratory systems. The appearance of dyspnea is the result of an imbalance in the interaction of multiple signals and receptors in the central nervous system, peripheral chemoreceptor receptors, and mechanoreceptors in the upper airway, lungs, and chest wall. In diagnosing the cause of dyspnea, the clinician must ascertain what is the source of the cause of the dyspnea. The management of postoperative dyspnea is carried out by treating the causes of dyspnea.

Keywords: postoperative; dyspnea; lung

INTRODUCTION

Post-surgical dyspnea is one of the complaints that often occurs in patients after undergoing surgery. Especially in patients who undergo surgery on the thoracic area. The presence of post-surgical dyspnea is a clinical symptom of post-surgical pulmonary complications. The term post-surgical pulmonary complications covers almost all complications affecting the respiratory system after anesthesia and surgery. These complications are heterogeneously defined, occur commonly, have major adverse effects on patients, and are difficult to predict [1].

The presence of post-surgical dyspnea indicates the presence of pulmonary complications which are a major cause of post-surgical morbidity and mortality. The incidence of post-surgical pulmonary complications has been reported to vary between 5% and 80%. The incidence varies between hospitals. Patients undergoing thoracic surgery are usually high-risk patients. Especially in patients who are elderly, have concurrent medical comorbidities, and have physical status problems due to malignancy, malnutrition, and pre-existing primary disease.

Pulmonary problems that can cause post-surgical dyspnea include bronchiectasis, bronchitis, asthma, pleural effusion, pneumothorax, pulmonary edema, bleeding, atelectasis, and other pulmonary problems. Non-lung problems can also cause anemia, trauma, obesity, ascites, acidosis, panic disorder, and others. Most dyspnea patients requiring thoracic area surgery are smokers and have occupational exposures that put them at higher risk of pulmonary complications. These problems arise due to the patient's lower baseline lung function. Pulmonary complications can manifest in the operating room itself or the post-anesthesia care unit, intensive care unit (ICU), and also in the surgical ward [2].

Post-surgical dyspnea may occur 48-72 hours after surgery. Post-surgical dyspnea may result from conditions affecting the respiratory tract that may affect the clinical course of the post-surgical patient. These conditions are pulmonary abnormalities that occur in the post-surgical period and can be identified as clinically significant and adverse disease or dysfunction [3].

A major cause of perioperative morbidity and mortality in the thoracic surgery population is respiratory complications. Major respiratory complications include atelectasis, pneumonia, and respiratory failure. These complications occur in 15-20% of patients and also cause 3-4% of expected deaths. Deaths occur due to various things such as late diagnosis and treatment. Based on research, it is known that patients who experience post-surgical lung problems experience 24% mortality compared to patients who do not experience 1.2% post-surgical lung problems within 90 days [1,2].

Seeing the incidence and complications of post-surgical dyspnea, the author is interested and there is a need for a review that discusses more deeply post-surgical dyspnea caused by pulmonary and respiratory system complications.

DEFINITION OF POST-SURGICAL DYSPNEA

The American Thoracic Society defines dyspnea as the subjective experience of respiratory discomfort consisting of qualitatively distinct sensations that vary in intensity. The experience stems from the interaction between various physiological, psychological, social, and environmental factors and may lead to secondary physiological and behavioral responses [3].

Post-surgical dyspnea is one of the complaints that often occurs in patients after surgery, especially in patients who undergo surgery on the thoracic area. Post-surgical dyspnea can occur 48-72 hours after surgery. Pulmonary problems that can cause post-surgical dyspnea include bronchiectasis, bronchitis, asthma, pleural effusion, pneumothorax, pulmonary edema, bleeding, atelectasis, and COPD. Problems outside the lungs can also cause shortness of breath, namely anemia, trauma, obesity, ascites, acidosis, panic disorder, and others [1,2].

PATHOPHYSIOLOGY OF POST-SURGICAL DYSPNEA

The pathophysiology of post-surgical dyspnea may be due to changes in homeostasis caused by anesthesia and the surgery itself. Anesthesia can cause respiratory distress, either in patients breathing spontaneously or with the aid of mechanical ventilation. Thus, most anesthetics lead to a decrease in functional residual capacity caused by loss of muscle tone. Pulmonary ventilation function may be altered by the use of anesthesia prior to a surgical procedure. This can occur through several mechanisms, such as increased shunt dead space, changes in ventilation distribution to non-dependent lung regions, changes in pulmonary circulatory distribution where there is increased perfusion to the dorsal part of the lung, and hypoxic pulmonary vasoconstriction [1,3].

These mechanisms can cause changes in internal homeostasis through several pathways. Physiologically, there is awareness of respiratory motor commands that affect the ventilatory muscles. This awareness has a different regulation of breathing compared to the involuntary ventilation mechanism regulated by the brainstem and voluntary ventilation regulated through the motor cortex and sensory cortex. Such awareness is important in shaping the auxiliary respiratory effort and is induced by physiological changes, such as respiratory muscle weakness due to loss of muscle tone (changes that occur due to the use of anesthesia). In addition, changes in respiratory homeostasis due to anesthesia may also affect the function of receptors in the thoracic wall, vagal receptors, and chemoreceptors. These functions can be affected through afferent signaling pathways that shape breathing patterns [4,5].

Clinical symptoms result from compensatory efforts of the cardiovascular and respiratory systems. Dyspnea may also result from metabolic disorders, neuromuscular disorders, or psychogenic conditions. It results from increased respiratory effort or air deprivation, caused by pulmonary ventilation that does not match the effort to breathe. Dissociation between pulmonary ventilation and respiratory effort arises from a mismatch between afferent receptors in the airways, lungs, and chest wall structures, and the motor activity of the respiratory center [6].

POST-SURGICAL PULMONARY COMPLICATIONS

There are several types of diseases that can cause complaints of shortness of breath in post-surgical patients. Some post-surgical lung diseases that can cause complaints of dyspnea include post-surgical pleural effusion, atelectasis, pneumothorax, bronchospasm, aspiration pneumonitis, pneumonia, acute respiratory distress syndrome (ARDS), pulmonary edema, and pulmonary embolism.

It is estimated that more than 230 million major surgeries occur worldwide each year. The incidence of dyspnea after major surgery ranges from 1 to 23%. Some studies show dyspnea with pulmonary complications is more common than cardiac complications, and post-surgical respiratory failure is the most frequent. Mortality increases in both the short and long term in patients who develop dyspnea and progress to pulmonary complications. One in five patients who have pulmonary complications will die within 30 days of major surgery compared to 0.2-3% without pulmonary complications. 90-day mortality has been shown to be significantly increased in patients with pulmonary complications [1].

Morbidity is also increased due to dyspnea and pulmonary complications. Hospital length of stay has been shown to lengthen by 13-17 days. The presence of dyspnea with pulmonary complications also increases healthcare costs, especially as a result of increased length of stay. For example, pneumonia or respiratory failure in a tertiary hospital in Canada resulted in increased costs of 41 and 47%, respectively [1].

Pleural Effusion

Post-surgical pleural effusion is the accumulation of fluid in the pleura after surgery. Pleural effusion can occur in 41 to 89% of patients undergoing procedures such as CABG especially in the first 7 days after surgery [8]. Accumulation of excess fluid can occur if excessive production, decreased absorption, or both exceed normal homeostatic mechanisms. In the post-surgical period, there may be disturbances in hydrostatic pressure and homeostasis resulting in pleural effusion. The most common type of pleural effusion is transudate [7,8,9].

A common surgical procedure that causes pleural effusion is coronary artery bypass grafting (CABG). Pleural effusions can have an unfavorable prognosis and slow healing after surgery. Often post-surgical pleural effusions require additional interventions such as fluid drainage and risk causing other post-surgical complications. In today's modern era, the increase in patient comorbidities and complexity of procedures has led to an increase in the incidence of post-surgical pleural effusion [10].

Atelectasis

Atelectasis can be defined as incomplete lung expansion and can lead to lung collapse. It is caused by partial or complete reversible collapse of small airways, resulting in impaired exchange of CO2 and O2. The incidence of atelectasis in patients under general anesthesia is 90%. The incidence of atelectasis is unaffected in both genders, and there is no increased incidence in patients with COPD, asthma, or geriatrics [11].

Atelectasis is one of the most common complications of surgery, especially cardiac and abdominal surgery. The most common types of postoperative atelectasis are postoperative atelectasis and obstructive atelectasis [11]. Atelectasis can occur during surgery due to several factors such as induction from general anesthesia using neuromuscular ganglion blockers that induce muscle paralysis, increased intra-abdominal pressure (laparoscopy), and can be triggered due to surgical positions such as the Tredelenburg position. The most common lung area is the basal area. Atelectasis can reach 15-20% of the lung area and can be more extensive in obese patients. Management may include encouraging deep breathing, early ambulation, incentive spirometry, use of acapella devices, chest physiotherapy, tracheal suctioning (in intubated patients), and/or positive pressure ventilation has been shown to reduce atelectasis [12,13].

Pneumothorax

Pneumothorax is a condition where air is trapped in the pleural space. Pneumothorax can occur spontaneously or secondary to surgery. The incidence of pneumothorax is quite high, based on an epidemiological study from 2011-2015 there were 10,500 cases in Germany due to pneumothorax [14].

Iatrogenic pneumothorax occurs due to complications of medical or surgical procedures.

Postoperative pneumothorax may occur after barotrauma due to mechanical ventilation, spontaneously due to a ruptured bleb, due to invasive catheter insertion, or due to bronchial branching damage [15,16]. Several surgical or medical procedures can lead to intra- or post-surgical pneumothorax. Acupuncture, orthopedic injections, CT scan-guided lung biopsy, bronchoscopy, central venous catheter (CVC) insertion, or thoracocentesis may cause damage to the pleura visceralis and respiratory air to enter the pleural cleft, resulting in a pneumothorax [14].

Diagnosis is generally made through clinical evaluation supported by examination with radiologic modalities. Chest X-ray is the most commonly used modality, but chest radiography, ultrasonography, or CT can also be used for diagnosis. The radiographic finding in a pneumothorax patient of a 2.5 cm air space is equivalent to 30% of pneumothoraces. An occult pneumothorax may be diagnosed with CT but is usually not clinically significant [15].

Management may be conservative, observational, or interventional with needle aspiration and chest tube insertion.[14] The choice of treatment is tailored to the patient's presentation and clinical condition. For patients with associated symptoms and signs of instability, needle decompression is the primary management option [15].

Bronchospasm

Bronchospasm is a type of airway hyperreactivity triggered by an underlying respiratory disorder. Bronchospasm is common during post-surgery. Bronchospasm can be identified by prolonged expiration, wheezing, and dyspnea. The causes of post-surgical bronchospasm are aspiration, histamine release caused by drugs such as opiates and atracurium, or allergic reactions to drugs. The sudden onset of bronchospasm after induction of anesthesia is associated with various physiological changes, cardiovascular changes, and skin signs that clinically suggest a drug-induced anaphylactic response. Bronchospasm commonly occurs during the perioperative period after induction of anesthesia or intubation. It can be triggered by various factors, such as immediate hypersensitivity reactions including IgE-mediated anaphylaxis, non-pharmacologic mechanisms, or pharmacologically induced by histamine-releasing drugs in patients with uncontrolled airway hyper-reactivity [16,17].

Acute exacerbation of chronic lung conditions such as asthma or chronic obstructive pulmonary disease (COPD) can cause bronchospasm. Reflex bronchial smooth muscle constriction is triggered by tracheal stimulation resulting from secretions, suctioning, endotracheal intubation, or other surgery that stimulates the trachea. Management may involve the administration of medications that cause relaxation of the bronchial muscles and close observation [2,18].

Aspiration Pneumonitis

Aspiration pneumonitis is an inflammatory condition of the bronchi to the alveolus due to the ingress of irritants. Pneumonitis presents with symptoms of sudden hypoxia, fever, tachycardia, dyspnea, and chest X-ray abnormalities caused by the microaspiration of harmful fluids. Often the fluid is sterile gastric contents or it could be bile or other material from the stomach. The risk of aspiration pneumonitis at the time of surgery comes from aspiration of acidic gastric contents during perioperative. Clinical symptoms may vary with some cases resolving completely but may also progress to secondary infection. Diagnosis is made through clinical evaluation, especially in critically ill hospitalized patients. Once confirmed through clinical symptoms, management should be given immediately without waiting for imaging results or other supporting examinations. If aspiration is suspected, the patient should be closely monitored for the next 24 to 48 hours. Management may include antibiotics especially if infection is suspected [2,19].

Pneumonia

Pneumonia is an inflammation of the lung parenchyma caused by microorganism infection. Post-surgical pneumonia is quite common in patients with gastric area surgery. The incidence rate in patients undergoing total gastrectomy ranges from 4.2 to 14% [20]. Postoperative pneumonia can be defined as Hospital Acquired Pneumonia (HAP) which is defined as pneumonia that develops 48-72 hours after hospital admission or Ventilator-Associated Pneumonia (VAP), pneumonia that develops 48-72 hours after endotracheal intubation) that occurs in postoperative patients. Currently, postoperative pneumonia is the third most common complication of all surgical procedures and is associated with increased patient morbidity and mortality [21].

There are several studies linking general surgical procedures with the development of postoperative pneumonia. In a retrospective study involving 555 patients undergoing partial hepatectomy, Pessaux et al reported an incidence of postoperative pneumonia of 13%. Similarly, Nobili et al retrospectively reviewed 555 hepatectomy patients and found that the incidence of postoperative pneumonia was 13%.[22] Hepatic surgery patients are prone to VAP. Siniscalchi et al reviewed 242 orthotopic liver transplant patients and determined a VAP incidence of 7.4%. The mortality rate was 22% and 4% in patients with and without VAP, respectively [23].

There are several studies that link cardiothoracic surgical procedures with the development of postoperative pneumonia. In an observational study by Strobel et al, 16,084 patients underwent coronary artery bypass grafting (CABG), and 3.3% of patients developed postoperative pneumonia [24]. Ventilator use may be a risk factor for postoperative pneumonia, based on a retrospective study by Allou et al that evaluated 7,439 cardiac surgery patients and reported a postoperative VAP incidence of 3.5% [25].

Similarly, Poelaert et al retrospectively studied 136 cardiac surgery patients and reported a postoperative pneumonia incidence of 32% [26]. A prospective observational study of 105 cardiothoracic surgery patients by Stephan et al diagnosed 54.3% with non-ventilator intensive care unit (ICU) acquired postoperative pneumonia [27]. A retrospective study by Simonsen et al looked at 7479 lung cancer surgery patients, finding 3.6% of patients had postoperative pneumonia [28]. Similarly, a retrospective study by Wang et al studying 511 patients undergoing lung resection for cancer found 2.9% of patients had postoperative pneumonia [29].

Post-surgical pneumonia is a major complication that often occurs in patients after elective surgery. Especially in immunocompromised populations such as geriatrics. Increased age, chronic obstructive pulmonary disease, emergency surgery, decreased postoperative albumin, prolonged ventilation, and longer duration of bed rest were identified as significant risk factors independently associated with postoperative pneumonia. Postoperative pneumonia is most commonly caused by gram-negative pathogens and multidrug-resistant bacteria accounted for approximately 16.99% of cases [30]. The incidence of post-surgical pneumonia will greatly affect the quality of life and also affect the outcome of the procedure performed.

Management with antibiotics and regular monitoring. Because patients can fall into sepsis and death if not treated properly. Antibiotic selection for each patient should be based on risk factors for MDR pathogens. Broad-spectrum empirical antibiotic therapy must be accompanied by a commitment to reduce antibiotic use, based on serial clinical and microbiological data, to limit the emergence of resistance in the hospital [20].

Pulmonary Edema

Pulmonary edema that occurs during general anesthesia is a rare complication, pulmonary edema can be either cardiogenic or non-cardiogenic. The most frequent causes of iatrogenic pulmonary edema in surgical patients are circulatory overload and the use of anesthetics that depress the myocardium, especially when used in conjunction with strong vasoconstrictors [31]. Post-surgical pulmonary edema is a complication of upper airway obstruction. Post-obstructive pulmonary edema is a life-threatening clinical condition in which pulmonary edema occurs immediately and develops after upper airway obstruction [32].

Several etiological factors have been found to cause postoperative pulmonary edema with much evidence suggesting that most patients have a history of pre-existing cardiac disease. Excessive intravenous fluid administration during the perioperative period can trigger cardiac dysfunction which can be fatal if it progresses to ischemia. Besides fluid overload, there are other pathogenic mechanisms that can lead to the clinical presentation of pulmonary edema.

Neurogenic pulmonary edema is one potential cause for the development of this pathology. Acute respiratory distress syndrome (ARDS) is considered an important differential diagnosis, especially in critically ill patients and trauma poly patients. Other clinical conditions such as head injury, hyponatremia, adrenal tumors, sepsis, pneumonitis, and so on are also involved in causing postoperative pulmonary edema [33].

The therapeutic targets in pulmonary edema are to improve oxygenation, maintain adequate blood pressure for the perfusion of vital organs, and reduce excess extracellular fluid. The underlying cause should be identified and be the primary therapeutic target. Diuretics are indicated for patients with fluid overload. The most commonly used drug is furosemide which should be given by intravenous injection slowly. Routine use of morphine is not recommended due to its side effects. Oxygen should only be given in cases of hypoxemia. Inotropic drugs should only be started if there is hypotension and evidence of reduced organ perfusion. In these cases, dobutamine is usually the first-line treatment [34].

Pulmonary Embolism

Acute pulmonary embolism is a fatal complication, and the exact mechanism that triggers embolism is still unknown. Pulmonary embolism can occur within hours to days after surgery. Early diagnosis and appropriate treatment are key to saving the patient. Delaying diagnosis and treatment from the time symptoms appear may increase mortality rates. The urgency of managing acute pulmonary embolism is also emphasized from the data that 18 patients died within 4 hours of symptoms [35].

Due to the variable nature of the presentation of pulmonary embolism, evaluation depends largely on the symptoms of suspected pulmonary embolism and the stability of the patient. There are scoring systems to aid in the determination of possible pulmonary embolism and thromboembolic events. Diagnostic scoring systems such as the Wells criteria and Geneva score are frequently used. In addition to the Wells and Geneva systems, which clinicians use to help determine thrombosis, the PE Rule-Out Criteria (PERC) can help rule out pulmonary embolism in low-risk emergency department patients.

Imaging modalities such as computed tomography pulmonary angiography (CTPA) or ventilation-perfusion (V/Q) scans are able to visualize pulmonary vasculature and perfusion patterns for the diagnosis of pulmonary embolism [36].

Pulmonary embolism mostly results from a blood clot that breaks off in the deep veins of the legs traveling to the lungs and blocking the pulmonary arteries. In addition to the predisposing risks before surgery, there is a theory that explains how pulmonary resection can cause pulmonary embolism minutes to hours after surgery. In patients undergoing pneumonectomy, if the pulmonary artery stamp is left too long after ipsilateral pneumonectomy, it may predispose thrombus formation and cross-embolism to the contralateral artery. Damage to the vessel wall during surgery is one accepted theory for blood clot formation that precedes the development of pulmonary embolism [37].

DIAGNOSIS

Pre-surgical evaluation is an important thing to do. Several risk prediction methods can be used to identify patients at high risk of complications to enable optimal perioperative management. One widely used method is the ARISCAT method which develops a seven-variable regression model, categorizing patients into low, intermediate, and high risk groups. The independent variables are low preoperative peripheral oxygen saturation (SpO2; <96%), respiratory tract infection in the past month, age, preoperative, anemia (<10 g dl or acute lung injury/ARDS), intrathoracic/upper abdominal surgery, procedure duration (>2 hours), and emergency surgery. Therefore, these conditions are important to evaluate and stratify the risk [1]

In establishing the diagnosis of the cause of dyspnea, the clinician must ascertain what the source of the dyspnea is. Dyspnea can stem from lung, heart or metabolic problems. So it must be explored in depth whether the dyspnea that occurs after surgery is due to abnormalities in the lungs, heart, or metabolism. If the cause of the dyspnea has been determined, the next step is to conduct a follow-up evaluation, as shown in Figure 1. To be able to make a proper diagnosis, it is necessary to have a proper history, physical examination, and support [1,37].

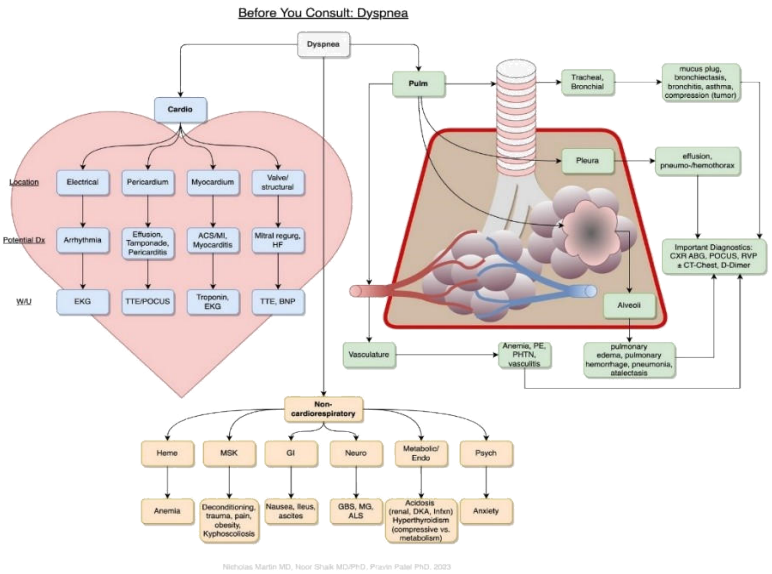

FIGURE 1: Differential diagnosis of dyspnea [37].

The history and physical examination aim to determine the presence of underlying cardiovascular, pulmonary, musculoskeletal, or psychiatric disease. Key components of the history that should be collected include onset, duration, and whether it occurs during rest or activity. The presence of a cough may indicate asthma, chronic obstructive pulmonary disease (COPD), or pneumonia. A severe sore throat may indicate epiglottitis. Pleuritic chest pain may indicate pericarditis, pulmonary embolism, pneumothorax, or pneumonia. Orthopnea, nocturnal paroxysmal dyspnea, and edema suggest a possible diagnosis of congestive heart failure [38].

The physical examination should begin with a rapid assessment of the airway, breathing, and circulation. Once the patient is stabilized, a full physical examination can be performed. To determine the severity of dyspnea, observe respiratory effort, accessory muscle use, mental status, and speech. The tracheal deviation may indicate possible anatomical abnormalities or pneumothorax. Stridor sounds from auscultation of the upper airway suggest airway obstruction. Palpation of the chest can determine the presence of subcutaneous emphysema or crepitus.

Percussion of the lung lobes for dimness can determine the presence or absence of consolidation and effusion. Hyperresonance on percussion indicates possible pneumothorax or severe bullous emphysema.

Auscultation of the lungs may result in an absence of breath sounds suggestive of a pneumothorax or an area-occupying mass such as pleural effusion or malignancy. The presence of wheezing is highly consistent with the diagnosis of obstructive lung diseases such as asthma or COPD. However, wheezing may be associated with pulmonary edema or pulmonary embolism. Pulmonary edema and pneumonia may present with rales on auscultation. Cardiac auscultation aims to look for dysrhythmias, heart murmurs, or aberrant heart sounds. Lower limb edema is associated with congestive heart failure, and extreme swelling of the extremities suggests the possibility of deep vein thrombosis that may lead to pulmonary embolism. Pounding fingers are present in some forms of lung malignancy or severe chronic hypoxia. Cyanosis of the extremities suggests hypoxia [38].

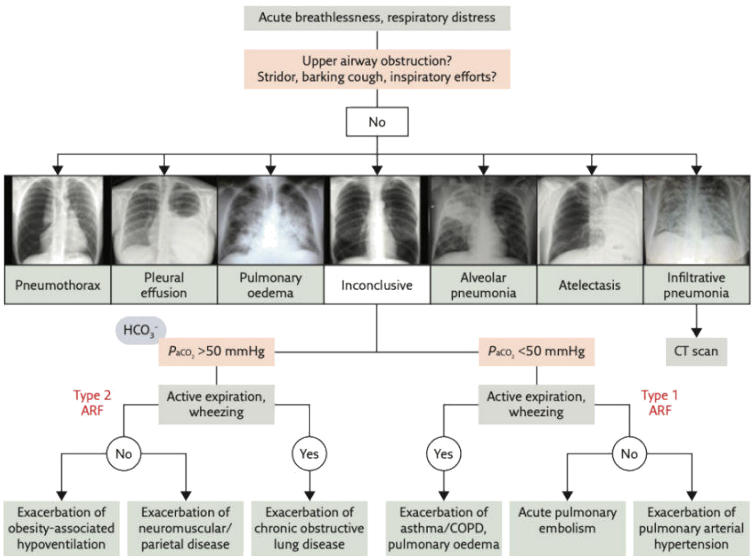

FIGURE 2: Diagnostic approach to postoperative dyspnea [38].

Clinical assessment, thoracic X-ray, and blood gas analysis are important in the evaluation of post-surgical dyspnea as shown in Figure 2. Chest x-ray is the first diagnostic test that should be used in evaluating dyspnea. If abnormal results are found, the problem is likely with the heart or primary pulmonary process. An echocardiogram is required to evaluate heart function and valve function. Blood gas analysis examination to determine the degree of hypokalemia, calculate a gradient, and assess the state of acidosis.

PRINCIPLES OF MANAGEMENT

In general, the principle flow of management of patients with dyspnea is to treat emergencies first by providing oxygenation and stabilization. A quick evaluation with good treatment will help reduce mortality [1,6].

1. Oxygen Therapy

The principle of post-surgical dyspnea management is to treat the cause of dyspnea. Supplemental oxygenation can reduce mortality in chronic hypoxemic patients with COPD. The beneficial effects of oxygen may be related to changes in chemoreceptor stimulation, changes in breathing patterns, and/or stimulation of receptors related to gas flow through the upper airway [39].

2. Causal therapy

Once the patient is stabilized, the cause of the dyspnea is evaluated, whether it is due to pulmonary, cardiovascular, or other problems. It is important to determine the severity and determine the location of follow-up care. Evaluation and surveillance in the intensive care unit are recommended. The focus of management is to treat the cause of the dyspnea itself [39].

3. Pulmonary rehabilitation

Pulmonary rehabilitation therapy is an integral component of the management of patients with chronic lung disease. Among the beneficial effects of pulmonary rehabilitation are a reduction in dyspnea during exercise and improved exercise tolerance, as well as a decrease in self-reported dyspnea with activity. Exercise is the main component of pulmonary rehabilitation responsible for these improvements, but it is less clear what the mechanism is that leads to improvements in dyspnea. According to the practice guideline recommendations of the American College of Chest Physicians and the American Association of Cardiovascular and Pulmonary Rehabilitation (ACCP/AACVPR), pulmonary rehabilitation is reserved for any stable patient with chronic lung disease that causes disability due to respiratory symptoms [40].

CONCLUSIONS

Post-surgical dyspnea is one of the complaints that often occurs in patients after surgery, especially in patients who undergo surgery in the thoracic area.

The presence of post-surgical dyspnea is a clinical symptom of post-surgical pulmonary complications. Some post-surgical lung diseases that can cause complaints of dyspnea include post-surgical pleural effusion, atelectasis, pneumothorax, bronchospasm, aspiration pneumonitis, pneumonia, pulmonary edema, and pulmonary embolism. The incidence of dyspnea in major surgery ranges from 1 to 23%. The pathophysiology of post-surgical dyspnea may be due to changes in homeostasis caused by anesthesia and the surgery itself. In establishing the diagnosis of the cause of dyspnea, the clinician must ascertain the source of the dyspnea. Management of post-surgical dyspnea is done by addressing the cause of the dyspnea.

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REFERENCES

[1] Miskovic A, Lumb AB. Postoperative pulmonary complications. Vol. 118, British Journal of Anaesthesia. Oxford University Press; 2017. p. 317-34.

[2] Sengupta S. Post-operative pulmonary complications after thoracotomy. Vol. 59, Indian Journal of Anaesthesia. Indian Society of Anaesthetists; 2015. p. 618-26.

[3] Sarkar S, Pal J, Chakraborty S. Clinical Methods in Respiratory Medicine. 1st ed. Jaypee Brothers Medical Publishers; 2018. 66-75 p.

[4] American Thoracic Society Medical Section of The American Lung Association Dyspnea Mechanisms, Assessment, and Management: A Consensus Statement [Internet]. Available from: www.atsjournals.org

[5] Hedenstierna G, Edmark L. Effects of anesthesia on the respiratory system. Vol. 29, Best Practice and Research: Clinical Anaesthesiology. Bailliere Tindall Ltd; 2015. p. 273-84.

[6] Coccia CBI, Palkowski GH, Schweitzer B, Motsohi T, Ntusi NAB. Dyspnoea: Pathophysiology and a clinical approach. South African Medical Journal. 2016 Jan 1;106(1):32-6.

[7] Meriggi F. Dyspnea in Cancer Patients: A Well-Known and Neglected Symptom. Rev Recent Clin Trials. 2018 May 7;13(2):84-8.

[8] Schiefenhövel F, Poncette AS, Boyle EM, von Heymann C, Menk M, Vorderwülbecke G, et al. Pleural effusions are associated with adverse outcomes after cardiac surgery: a propensity-matched analysis. J Cardiothorac Surg. 2022 Dec 1;17(1).

[9] Roberts ME, Rahman NM, Maskell NA, Bibby AC, Blyth KG, Corcoran JP, et al. British Thoracic Society Guideline for pleural disease. Thorax. 2023 Jul 1;78: S1-42.

[10] Brookes JDL, Williams M, Mathew M, Yan T, Bannon P. Pleural effusion post coronary artery bypass surgery: Associations and complications. J Thorac Dis. 2021 Feb 1;13(2):1083-9.

[11] Grott K, Chauhan S, Dunlap JD. Atelectasis. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.

[12] Kreider ME, Lipson DA. Bronchoscopy for atelectasis in the ICU: A case report and review of the literature. Vol. 124, Chest. American College of Chest Physicians; 2003. p. 344-50.

[13] Kelkar KV. Post-operative pulmonary complications after non-cardiothoracic surgery. Vol. 59, Indian Journal of Anaesthesia. Indian Society of Anaesthetists; 2015. p. 599-605.

[14] Devecel G, Şenturan L. The Effect of Breathing Exercises on Preventing Postoperative Atelectasis: A Randomized Controlled Trial. Florence Nightingale Hemşirelik Dergisi. 2018 Oct 26;159-67.

[15] McKnight CL, Burns B. Pneumothorax. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.

[16] Kimmel ME, Zasik JM. Postoperative pneumothorax. Real or pseudo? Gastrothorax simulating postoperative pneumothorax. Chest. 1990;97(3):719-20.

[17] Garcia D, Kehar M, Khan ES, Mendonca R, Girshin M. Multiple Episodes of Severe Bronchospasm During General Anesthesia: A Case Report. Cureus. 2022 Jan 23.

[18] Schnell J, Beer M, Eggeling S, Gesierich W, Gottlieb J, Herth FJF, et al. Management of Spontaneous Pneumothorax and Post-Interventional Pneumothorax: German S3 Guideline. Vol. 97, Respiration. S. Karger AG; 2019. p. 370-402.

[19] Son YG, Shin J, Ryu HG. Pneumonitis and pneumonia after aspiration. J Dent Anesth Pain Med. 2017;17(1):1.

[20] Kunisaki C, Miyata H, Konno H, Saze Z, Hirahara N, Kikuchi H, et al. Modeling preoperative risk factors for potentially lethal morbidities using a nationwide Japanese web-based database of patients undergoing distal gastrectomy for gastric cancer. Gastric Cancer. 2017 May 1;20(3):496-507.

[21] Chughtai M, Gwam CU, Mohamed N, Khlopas A, Newman JM, Khan R, et al. The Epidemiology and Risk Factors for Postoperative Pneumonia. J Clin Med Res. 2017;9(6):466-75.

[22] Nobili C, Marzano E, Oussoultzoglou E, Rosso E, Addeo P, Bachellier P, et al. Multivariate analysis of risk factors for pulmonary complications after hepatic resection. Ann Surg. 2012 Mar;255(3):540-50.

[23] Siniscalchi A, Aurini L, Benini B, Gamberini L, Nava S, Viale P, et al. Ventilator associated pneumonia following liver transplantation: Etiology, risk factors and outcome. World J Transplant. 2016;6(2):389.

[24] Strobel RJ, Liang Q, Zhang M, Wu X, Rogers MAM, Theurer PF, et al. A Preoperative Risk Model for Postoperative Pneumonia After Coronary Artery Bypass Grafting. In: Annals of Thoracic Surgery. Elsevier USA; 2016. p. 1213-9.

[25] Allou N, Allyn J, Snauwaert A, Welsch C, Lucet JC, Kortbaoui R, et al. Postoperative pneumonia following cardiac surgery in non-ventilated patients versus mechanically ventilated patients: Is there any difference? Crit Care. 2015 Mar 11;19(1).

[26] Poelaert J, Haentjens P, Blot S. Association among duration of mechanical ventilation, cuff material of endotracheal tube, and postoperative nosocomial pneumonia in cardiac surgical patients: A prospective study. Journal of Thoracic and Cardiovascular Surgery. 2014 Oct 1;148(4):1622-7.

[27] Stéphan F, Zarrouki Y, Mougeot C, Imbert A, Kortchinsky T, Pilorge C, et al. Non-ventilator ICU-acquired pneumonia after cardiothoracic surgery: Accuracy of diagnostic tools and outcomes. Respir Care. 2016 Mar 1;61(3):324-32.

[28] Simonsen DF, Søgaard M, Bozi I, Horsburgh CR, Thomsen RW. Risk factors for postoperative pneumonia after lung cancer surgery and impact of pneumonia on survival. Respir Med. 2015 Oct 1;109(10):1340-6.

[29] Wang Z, Cai XJ, Shi L, Li FY, Lin NM. Risk factors of postoperative nosocomial pneumonia in stage I-IIIa lung cancer patients. Asian Pacific Journal of Cancer Prevention. 2014;15(7):3071-4.

[30] Xiang B, Jiao S, Si Y, Yao Y, Yuan F, Chen R. Risk Factors for Postoperative Pneumonia: A Case-Control Study. Front Public Health. 2022 Jul 8;10.

[31] Adriani J, Zepernick R, Harmon W, Hiern B. Iatrogenic Pulmonary Edema in Surgical Patients. Surgery Journal. 1967 Feb;61(2).

[32] Cho H, Tsuchida K, Iwasaki K, Maezawa Y. Risk factors of post-operative pneumonia in elderly patients with gastric cancer: A retrospective cohort study. Jpn J Clin Oncol. 2021 Jul 1;51(7):1044-50.

[33] Bajwa Sjs, Kulshrestha A. Diagnosis, prevention and management of postoperative pulmonary edema. Ann Med Health Sci Res. 2012;2(2):180.

[34] Purvey M, Allen G. Managing acute pulmonary oedema. Aust Prescr. 2017;40(2):59-63.

[35] More PG, Durve SR. Acute pulmonary oedema: A post-operative complication due to neostigmine and post obstructive pulmonary oedema in a case of tonsillectomy. Journal of Clinical and Diagnostic Research. 2015 May 1;9(6): UD05-6.

[36] Corrigan D, Prucnal C, Kabrhel C. Pulmonary embolism: the diagnosis, risk-stratification, treatment and disposition of emergency department patients. Clin Exp Emerg Med. 2016 Sep 30;3(3):117-25.

[37] Shonyela FS, Yang S, Liu B, Jiao J. Postoperative acute pulmonary embolism following pulmonary resections. Vol. 21, Annals of Thoracic and Cardiovascular Surgery. Japanese Association for Coronary Artery Surgery; 2015. p. 409-17.

[38] Brinkman JE, Sharma S. Physiology, Pulmonary. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.

[39] Sharma S, Hashmi M, Burns B. Alveolar Gas Equation. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.

[40] Shih AR, Uruga H, Bozkurtlar E, Chung JH, Hariri LP, Minami Y, et al. Problems in the reproducibility of classification of small lung adenocarcinoma: an international interobserver study. Histopathology. 2019 Nov 1;75(5):649-59.