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APPROACH TO PATIENT WITH ACUTE DYSPNEA IN THE EMERGENCY DEPARTMENT

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Abstract:

Introduction: *Acute dyspnea is considered one of the major causes for admission to the emergency department (ED) [1] There are several ways to evaluate patients with acute decompensated heart failure (ADHF) include but not limited to the history taking, performing physical examination, chest imaging, 12-lead electrocardiography (ECG), and measurement of brain natriuretic peptide (BNP) or N-terminal pro-BNP. The physical examination, is usually not accurate, starting "dual therapy" for ADHF and chronic obstructive pulmonary disease (COPD) is considered to be harmful.****Aim of work:*** *In this review, we will discuss the most recent evidence regarding the approach to dyspnea in the emergency department, presentation, and management.****Methodology:*** *We did a systematic search for approach to dyspnea in the emergency department using PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com). Our search also looked for presentation, and management of diseases presents with dyspnea. All relevant studies were retrieved and discussed. We only included full articles.****Conclusions:*** *The diagnosis and management of patients presenting with acute dyspnea is one of the most important and challenging for clinicians in emergency department (ED). A right diagnosis is frequently delayed and difficult to confirm, explaining the need for fast diagnosis and a management plan. Acute dyspnea could present to the ED and it is imperative that emergency physicians be prepared to stabilize patients' oxygenation and ventilation, which requires careful and efficient consideration of the differential diagnosis. Using many cues from the history and physical examination, practitioners may guide the work-up and treatment to identify a parenchymal, obstructive, circulatory, or compensatory cause of dyspnea. The early use of bedside testing, including ultrasonography, may limit unnecessary tests and save time in determining the best treatment course.*

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INTRODUCTION:

Acute dyspnea is considered one of the major causes for admission to the emergency department (ED)[1]. Doctors working in the ED usually have to make a fast diagnosis and develop a management plan based on limited clinical evaluation [2] Quick and correct diagnosis and treatement can be lifesaving for patients with acute dyspnea [3], but making a differential diagnosis and choosing early management for patients with acute dyspnea in the ED is a huge and difficult challenge that needs complex decision-making to achieve hemodynamic stability, improve functional capacity, and avoid mortality and morbidity and the length of hospital stay [4]. There are several ways to evaluate patients with acute decompensated heart failure (ADHF) include but not limited to the history taking, performing physical examination, chest imaging, 12-lead electrocardiography (ECG), and measurement of brain natriuretic peptide (BNP) or N-terminal pro-BNP. The physical examination, is usually not accurate, starting "dual therapy" for ADHF and chronic obstructive pulmonary disease (COPD) is considered to be harmful.

In this review, we will discuss the most recent evidence regarding the approach to dyspnea in the emergency department, presentation, and management.

METHODOLOGY:

We did a systematic search for approach to dyspnea in the emergency department using PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com). Our search also looked for presentation, and management of diseases presents with dyspnea. All relevant studies were retrieved and discussed. We only included full articles.

The terms used in the search were: dyspnea, emergency department, COPD, asthma, CHF, presentation, diagnosis, management, and treatment.

Approach to Adult Patient with Dyspnea:

History

Acute dyspnea, or commonly in layman terms shortness of breath, is considered one of the major chief complaints in the ED. The differential diagnosis includes but is not limited to many disorders that could be divided based on obstructive, parenchymal, cardiac, and compensatory features. A detailed history taking can help to narrow this wide differential. In addition to common symptoms, considering risk factors such as past medical and family history, trauma, travel, medications, and exposures is extremely recommended.

Schwartzstein and his colleagues [5] used the analogy of a machine to detect different causes of dyspnea based on pathophysiologic data. Dysfunctions of the respiratory system could be due to faulty controllers, ventilatory pumps, or gas exchangers. Using guidelines makes it easier to understand the causes of shortness of breath related to respiratory risk factors.

The cardiovascular causes of dyspnea present with dyspnea by causing disruptions of the system that functional pumps oxygenated blood to tissues and then transports the carbon dioxide back to the pulmonary system. Likewise, the decreased oxygen carrying capacity seen anemia has a major role in its presentation with dyspnea.

Physical Examination:

Performing an extensive physical examination could provide important information. Respiratory rate and oxygen saturation are measured with vital signs. The doctor must evaluate the patient's work of breathing, looking for any tripoding or retractions. Crepitance in the chest could mean subcutaneous air and pneumothorax. Lung sounds including wheezing, rales, and rhonchi could help the differential. Other findings including decreased sounds, hyperresonance, or egophony could as well add additional information. Jugular venous distension, S3 gallop, and peripheral edema indicate that a patient has fluid overload. Conjunctival pallor, capillary refill, and temperature of extremities can provide clues about blood volume and general circulation. Pulses must also be assessed.

Testing:

There are many available tests that help to make the right diagnosis and narrow down the differential diagnosis for patients presented with acute dyspnea. When physicians use laboratory tests, they must weigh the information regarding risks involved in performing the tests Ultrasonography can add valuable details about the origin of symptoms, and, usually, it is the initial assessment of an acutely dyspneic cases. These images could be obtained during or shortly after initial assessment, greatly faster than laboratory tests or other imaging modalities. The Bedside Lung Ultrasonography in Emergency (BLUE) protocol is very helpful in offering one approach to differentiate many causes of respiratory failure [6].

There are other available protocols that help in assessments to assess for other cardiac causes of dyspnea.[7] [8] Focused evaluation of global left ventricular function, diastolic function, right ventricular size, and any pericardial effusion helps with rapid assessment for massive myocardial infarction, cardiac tamponade, and massive pulmonary embolism at the bedside. Additionally, inferior vena cava measurement could be used to check for right-sided heart failure and to calculate central venous pressure. Computed tomography (CT) use to assess acute dyspnea has increased in the last decade.[9] Risks could include contrast allergic reaction and nephropathy as well as cancers.[10] The American College of Physicians recently recommends advocate avoidance of CT as an initial test to evaluate patients at low risk for pulmonary embolism (PE) [11]. Furthermore, about one-fourth of patients undergoing CT for PE evaluation have significantly higher incidental findings. Though CT could add vital diagnostic detail, physicians should not only consider the scan's necessity but also plan appropriate follow-up for any clinically important incidental findings [12] It is recommended to constantly consider whether CT is a must or whether less risky methods, such as chest radiograph or ultrasonography, will provide an answer.

DIFFERENTIAL DIAGNOSIS FOR ACUTELY DYSPNEIC PATIENTS

Obstructive Dyspnea:

Expert physicians can detect respiratory distress from across the room. Symptoms and signs like diaphoresis , breathing extremely rapidly with minimal air movement should initiate an aggressive treatment, probably it is a severe asthma exacerbation, starting with BIPAP ventilation with continuous nebulized albuterol and order adjunct therapies including but not limited to intravenous steroids, intravenous magnesium, and intramuscular epinephrine. After twenty minutes, she could be able to breathe more comfortably with the BIPAP machine and repeat auscultation reveals diffuse wheezing and improved air movement.

Wheezing, or musical respiratory sounds, typically could result from partial airway obstruction [13]. Because this obstruction can result from inflammation, secretions, or even a foreign body, patients with noisy or whistling breathing need close evaluation to determine whether the noise is inspiratory or expiratory, and whether it is from the lower airways or the upper airways. Stridor from a swollen airway, foreign body, or other airway obstruction is extremely dangerous. Though patients in anaphylaxis could benefit from the nebulized beta-agonist medications used to treat an asthma exacerbation, it is not sufficient to save their lives. As opposed to wheezing, which is a lower airway expiratory sound, stridor is an upper airway sound transmitted when there is obstruction to the inflow of air during inspiration. The obstruction may be fixed or inflammatory (anaphylaxis), but in any situation must be emergently managed.

The national and world organizations define asthma by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation [14]. The reversibile airflow obstruction is the main distinguishing feature asthma from other obstructive respiratory disorders. While, chronic obstructive pulmonary disease (COPD)/ emphysema can be defined as persistent airflow limitation that is usually progressive and associated with enhanced chronic inflammatory responses in the airways and the lungs[14].

Asthma is considered as an obstructive disease caused by elevated airway resistance. It is a reversible condition, however if recurrent chronic inflammatory disorder that characteristically causes severe dyspnea, wheezing, and coughing.[15] There are two major issues in asthma: chronically inflamed airways and hyperresponsive airways. Intermittent airflow obstruction in symptomatic patients could result in lowered ability to expire, which lead to hyperinflation, stenting open the alveoli, and increasing the work of breathing. Early in an exacerbation, symptoms are bronchospastic secondary to smooth muscle contraction. As an episode progresses, inflammatory changes in the airways could cause increased airway resistance and lead to VQ mismatch The severity of an exacerbation could be measured clinically and should determine how aggressively a patient is managed.

COPD as described by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) is persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases [14]. The pathophysiology usually a mixture of lung parenchymal destruction, as described in emphysema, and small airway inflammation with airway obstruction, or obstructive bronchiolitis [13]. An exacerbation of COPD presents as dyspnea, cough, and increased sputum production. In the emergent setting, physicians have to manaeg the airflow limitation. As with asthma, monitoring of

pulse oximetry, degree of respiratory distress, and hemodynamic stability can help clinicians anticipate the degree of severity of a particular exacerbation. More specific testing could play a role, as radiographs and electrocardiograms may help differentiate other causes of shortness of breath from a COPD exacerbation. Additionally, an increase in sputum production or the presence of purulent sputum should be treated with antibiotics, regardless of other infectious symptoms [16]. Anaphylaxis occurs as a sudden, potentially fatal, allergic reaction and involve multiple organ systems [17,18].

Parenchymal Dyspnea:

Acute dyspnea is the most important symptom of patients with heart failure exacerbation. [19] around 80% of patients with acutely decompensated heart failure present through the ED with a chief complaint of dyspnea [20]. This symptom is associated with pulmonary as well as systemic fluid overload and also low cardiac output. The American College of Emergency Physicians clinical policy makes level B recommendations that standard clinical judgment can be improved with the use of a single B-type natriuretic peptide (BNP) or N-terminal pro–B-type natriuretic peptide measurement to rule in or out the diagnosis of CHF [21]. However, the true utility may be in patients with dyspnea not expected to have a CHF exacerbation, when finding a positive BNP could modify the treatment plan and allow a more rapid initiation of the managemen [22].

Carpenter and his colleagues [22] concluded that the classic constellation of symptoms such as jugular venous distension, peripheral edema, rales, and S3, were no more predictive of patients with both pulmonary edema on chest radiograph and an increased BNP level greater than 500 pg/dL than any individual finding alone. Though rales were the most sensitive finding tested for either outcome, they had specificity of only around fifty percent each. Jugular venous distention and S3 gallop were the individual findings most predictive for pulmonary edema on imaging or increased BNP level. Ultrasonography of the inferior vena cava could also improve diagnostic accuracy versus BNP and chest imaging soley [23].

The most common complaints in community-acquired pneumonia include but not limited to fever, cough, pleuritic chest pain, and sputum production, along with dyspnea. But, these clinical criteria could have sensitivity as low as fifty percent compared with a chest imaging [24]. On examination, several patients have crackles or evidence of consolidation. Guidelines from the Infectious Diseases Society of America and the American Thoracic Society, recommend chest radiograph in patients with suspected pneumonia, which may show lobular consolidation, interstitial infiltrate, or cavitation [25]. Though infiltration with suggestive symptoms makes the diagnosis, infiltrate may not be visible initially on patients with volume depletion. It is appropriate to treat empirically for twenty-four to forty eight hours in these patients.

The treatment plan of pneumonia requires history to allow classification based on the setting in which the illness was acquired. The Infectious Disease Society of America and American Thoracic Society define the types of pneumonia as follows [26]. Hospital acquired pneumonia (HAP) is which is a pneumonia that occurs forty eight hours or more after admission which was not incubating at the time of admission. Community-acquired pneumonia is not acquired in any of these situations. These classifications detect typical pathogens and guide appropriate initial management.

Circulatory Dyspnea

Pulmonary embolism (PE) could interfere with both ventilation and perfusion. Thus, it causes circulatory collapse due to obstruction of right ventricular outflow eventually causing elevated pulmonary artery pressure and failure of the right then left ventricles. Echocardiography could provide highly important findings: signs of right ventricular (RV) strain, including dilatation of the right ventricle, RV hypokinesis, paradoxic septal wall motion, McConnell sign (hypokinesis of the free RV wall with sparing of the apex), and tricuspid regurgitation [27].

Dresden and his colleagues [28] studied the use of ultrasonography in moderate-risk to high-risk patients to check whether the patients were good candidate for anticoagulation while awaiting definitive imaging. It was found that early anticoagulation is recommended to improve mortality and there is evidence to support anticoagulation before diagnosis in patients with a Wells score greater than four who will have a delay to diagnosis of more than one hour and fourty minutes [29,30].

Angina pectoris is can because chest pain due oxygen demand is higher than myocardial oxygen supply; in this patient due to occlusion of coronary arteries. Though typical chest pain is a part of the presentation, dyspnea alone may be the initial complaint, termed an anginal equivalent. To help the diagnosis it is recommended to ask the patients whether they experienced shortness of breath were considered dyspneic. The subset with no prior known

coronary artery disease had more than four times the risk of sudden cardiac death versus asymptomatic patients and more than twice the risk of those with typical angina [31].

Physicians must take into consideration the past medical history and risk factors when assessing dyspnea for cardiac causes like acute myocardial infarction and acute coronary syndrome.

Pneumothoraces could be detected by ultrasonography, chest radiograph, or CT. Management could be helped by cause, severity, comorbidities, interventions like positive pressure ventilation, size of the pneumothorax, and patient's preference. Recently studies have shown that uncomplicated spontaneous pneumothorax in patients not undergoing positive pressure ventilation may be managed as successfully with needle aspiration as with other more invasive chest drains, regardless of size [32].

Tension pneumothorax is considered a dangerous that needs needle decompression to prevent the loss of cardiac output and arrest. But, recently studies have suggested that the classic presentation of tension pneumothorax with hypotension, absent breath sounds, and deviated trachea may not be immediately seen in patients with spontaneous, unassisted respiration [33]. Due to the slower development of the accumulation of air and pressure variations, spontaneously breathing patients may compensate much longer and present atypically. So, physicians must remain alert.

Compensatory Dyspnea:

It is important to also take into consideration that shortness of breath, tachypnea, or other typical symptoms of dyspnea could arise from change in metabolic demands. These cases could have respiratory distress; they may be tachypneic, tachycardic, even pale or diaphoretic. In these patients, the physician is responsible for the fixation the true problem in order to improve the respiratory symptoms.

In patient with severe anemia, their bodies have oxygen hunger, which is manifested as shortness of breath. Patients with dysfunctional hemoglobins secondary to irreversibly bound atoms or toxins may also be functionally anemic with the same symptoms. Both the rate and the depth of ventilation often increase, leading to both tachypnea and hyperpnea, at times referred to as Kussmaul respirations. This compensatory response is crucial for survival and should not be mistaken for dyspnea. It is highly recommended to realize that an increase in alveolar ventilation is not usually a compensatory response (to acidosis or to primary pulmonary disorders) and hypocapnia may cause primary respiratory alkalosis, from central nervous system compromise, toxins, anxiety, or pain [34]. In these patients, imaging might not reveals a source of dyspnea, however clinical suspicion based on history and examination, including signs such as the fruity breath of ketonemia, the pallor of anemia, or the cyanosis of toxic hemoglobinopathies, directs providers toward appropriate laboratory testing and management.

Diagnosis of Exclusion:

Additionally, some cases of dyspnea are not dyspnea. For example, in acute anxiety and panic disorder the patient presents as shortness of breath, tachypnea, or hyperventilation. Patients with panic disorder often describe symptoms similar to those of patients with true airway obstruction despite their normal pulmonary function. It has been estimated that these patients have abnormal proprioception, experiencing dyspnea without abnormal stimuli. [35] But, patients with a history of pulmonary disease can also have pure panic episodes. Arterial blood gas may be useful in diagnosing anxiety-related hyperventilation [36].

Severe pain can also lead to abnormal respiratory patterns. Like compensatory problems, pain and anxiety can be managed by managing their causes. Manage the pain. Reduce stress and anxiety with words, behaviors, even medications.

CONCLUSIONS:

The diagnosis and management of patients presenting with acute dyspnea is one of the most important and challenging for clinicians in emergency department (ED). A right diagnosis is frequently delayed and difficult to confirm, explaining the need for fast diagnosis and a management plan. Acute dyspnea could present to the ED and it is imperative that emergency physicians be prepared to stabilize patients' oxygenation and ventilation, which requires careful and efficient consideration of the differential diagnosis. Using many cues from the history and physical examination, practitioners may guide the work-up and treatment to identify a parenchymal, obstructive, circulatory, or compensatory cause of dyspnea. The early use of bedside testing, including ultrasonography, may limit unnecessary tests and save time in determining the best treatment course. So, ensuring both the best care for the patient and also the physician's ability to readily respond to the next case.

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