

Acute Respiratory Distress Syndrome (ARDS) in adults

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Abstract

Background: Acute respiratory distress syndrome (ARDS) is a life-threatening condition characterized by poor oxygenation and non-compliant or "stiff" lungs. The disorder is related with capillary endothelial harm and diffuse alveolar harm. Once ARDS develops, patients ordinarily have changing degrees of aspiratory artery vasoconstriction and may along these lines create pulmonary hypertension. ARDS carries a tall mortality, and few viable restorative modalities exist to enhance this dangerous condition. This movement audits the clinical introduction, assessment, and administration of intense respiratory trouble disorder and highlights the significance of facilitated interprofessional cooperation in caring for patients with this condition. **Objective:** To know more about acute respiratory distress syndrome. Knowing the characteristics of patients with acute respiratory distress syndrome. Know how to treat patients with acute respiratory distress syndrome. **Design and Method:** Literature-review. **Conclusion:** ARDS proceeds to be associated with a high mortality. In spite of multiple randomized controlled trials, as it were lung defensive ventilation strategies, neuromuscular blocking operators, and inclined ventilation have been appeared to diminish mortality. Numerous trials are underway looking at nebulized heparin, aspirin, stem cell therapy, growth factors, interferon- β , and vascular endothelial growth figure. The unused Berlin definition of ARDS may help future trials of novel treatments by making strides diagnostic reliability and allowing more exact stratification of patients according to severity.

Keywords: Acute respiratory distress syndrome; adult; diagnosis; management of ARDS;

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Introduction

Acute Respiratory Distress Syndrome (ARDS) is a rapidly progressive disorder and initially manifests clinically as shortness of breath (dyspnea and tachypnea) which then rapidly progresses to respiratory failure. ARDS may also be characterized by bilateral pulmonary infiltrates and severe hypoxemia in the absence of cardiogenic pulmonary edema (Force et al., 2012). Generally ARDS is caused by sepsis, in addition to systemic disease factors, and lung injury (Bakhtiar & Maranatha, 2018; Force et al., 2012). Based on American statistics conducted by the National Institutes of Health (NIH) study, the annual frequency was recorded as 75 cases per 100,000 population, followed by research from Utah showing an incidence of 4.8-8.3 cases per 100,000 population. It is estimated that the condition of ARDS in America is 190,600 cases each year and an estimated death of 74,500 inhabitants. In addition, the latest study stated that 7.1% of cases admitted to the ICU and 16.1% of cases using ventilators had ARDS (Bakhtiar & Maranatha, 2018).

In Indonesia, ARDS is still underdiagnosed. the study of Bellani and colleagues showed that clinicians recognized or diagnosed as many as 60.2% of patients with ARDS. ARDS can cause sepsis and multiorgan failure that can lead to death so, in its management, clinicians must be faster and more responsive in dealing with ARDS (Andisari et al., 2020).

Method

This article was written using various sources from scientific journals and the World Health Organization (WHO). Source searches are carried out on online portals for journal publications such as Google Scholar (<https://scholar.google.co.id/schhp?hl=id>), National Center for Biotechnology Information/NCBI (<https://www.ncbi.nlm.nih.gov/>), and Medscape (<https://emedicine.medscape.com/>) with the keyword search used is "Acute Respiratory Distress Syndrome".

Literatur Review

Definition

Acute respiratory distress syndrome (ARDS) is an acute and diffuse inflammatory lung injury, resulting in increased pulmonary vascular permeability, increased pulmonary resistance, and loss of air-filled lung tissue, with hypoxemia and bilateral opacity on imaging, which is associated with increased shunting, increased physiologic dead space, and reduced lung compliance. ARDS may also be characterized by bilateral pulmonary infiltrates and severe hypoxemia in the absence of cardiogenic pulmonary edema (Force et al., 2012).

Epidemiology

Based on American statistics conducted by the study The National Institutes of Health (NIH) recorded an annual frequency of 75 cases per 100,000 population, followed by research from Utah showing an incidence of 4.8-8.3 cases per 100,000 population. It

is estimated that the condition of ARDS in America is 190,600 cases each year and an estimated death of 74,500 inhabitants (Force et al., 2012).

In Indonesia, ARDS is still underdiagnosed. the study of Bellani and colleagues showed that clinicians recognized or diagnosed as many as 60.2% of patients with ARDS. in addition, mild ARDS ranged between 51.3% and 78.5% for severe ARDS. Based on the condition ARDS can occur in all age groups, from children to adults. The incidence of ARDS increases with age, ranging from 16 cases per 100,000 per year in the age range 15-19 years and increasing to 306 cases per 100,000 per year in the age range 75-84 years (Andisari et al., 2020).

Pathophysiology

ARDS occurs due to acute inflammation that affects the surface of the alveolus. This causes an increase in membrane permeability thereby activating the action of neutrophils and other acute inflammatory mediators to the lung alveoli. The exudate resulting from acute inflammation leads to surfactant inactivation resulting in collapse and consolidation of the distal airspaces and progressive loss of lung gas. This condition will be compensated by pulmonary vasoconstriction which will cause hypoxia. If the inflammatory process continues, it will allow the blood to be deoxygenated. This process can lead to severe hypoxemia and eventually to respiratory failure due to hyperventilation (Griffiths et al., 2019).

One of the factors causing ARDS is sepsis. In the early stages of sepsis, there is an inflammatory state, upregulation of TNF- and IL1 β , and activation of inflammatory cells such as neutrophils. Among the proinflammatory cytokines, TNF α , IL-1 β , interleukin 6 (IL-6), and IL-8 are elevated in ARDS patients. The initial stage of inflammation is followed by decreased immunity, making the patient susceptible to nosocomial infections (Bakhtiar & Maranatha, 2018; Geetha, 2014).

Coagulation disorders that often occur in sepsis are thought to influence the onset of ARDS because in ARDS there is intra-alveolar, interstitial, and intravascular fibrin deposition. As a result of the systemic inflammation and coagulation that occurs in sepsis, there are significant changes in microcirculation, vascular reactivity, platelet aggregation, and white blood cell adhesion to the endothelium. Changes in the vascular endothelium and interactions between white blood cells and red blood cells result in peroxidase of the red blood cell membrane, changes in the pump in the red blood cell membrane, and calcium influx into the red blood cell. This leads to increased red blood cell aggregation and microvascular occlusion that favors organ failure (Bakhtiar & Maranatha, 2018).

Criteria and Diagnostic Steps

Diagnostic criteria and diagnostic steps (Force et al., 2012). The criteria for the diagnosis of ARDS are based on the Berlin Criteria, namely:

- a. Time. the onset of ARDS occurs within 1 week
- b. chest X-ray. Bilateral opacities not due to effusion, atelectasis, or pulmonary nodules
- c. Source of edema. The cause is respiratory failure, not heart failure or overloaded fluid

- d. hypoxemia:
 - Mild: $200 \text{ mmHg} < \text{PaO}_2/\text{FiO}_2 \leq 300 \text{ mmHg}$ with PEEP or CPAP $\geq 5 \text{ cmH}_2\text{O}$
 - Moderate: $100 \text{ mmHg} < \text{PaO}_2/\text{FiO}_2 \leq 200 \text{ mmHg}$ with PEEP $> 5 \text{ cmH}_2\text{O}$
 - Weight: $\text{PaO}_2/\text{FiO}_2 \leq 100 \text{ mmHg}$ with PEEP $\geq 5 \text{ cmH}_2\text{O}$

The steps for diagnosing ARDS start from anamnesis, physical examination, and supporting examinations (Geetha, 2014).

- a. History, characterized by acute shortness of breath after trauma, sepsis, drug overdose, massive transfusion, pancreatitis, and aspiration.
- b. Physical examination, characterized by tachypnea, tachycardia, and increased FiO_2 requirements. In addition, hypotension and signs of peripheral vasoconstriction such as chills and peripheral cyanosis may be found. On auscultation of the thorax, bilateral wet crackles were found. The temperature of patients with ARDS is febrile or hypothermic (John Hopkins, 2021).
- c. Investigations are characterized by chest radiographs in the form of bilateral opacification and symmetrical or asymmetrical consolidation accompanied by an air bronchogram.

Differential Diagnosis

ARDS can have non-specific symptoms so doctors need to consider various diseases that are the differential diagnosis, such as heart disease, infection, and other diseases based on the previous history including comorbidities, medications, or other exposures (Fishman et al., 2008; Force et al., 2012).

The following is a table for the differential diagnosis of ARDS (Force et al., 2012):

Conditions	Diagnostic clues
Asthma	Cough, wheezing
Chronic obstructive pulmonary disease	Decreased respiratory movement, prolonged expiratory phase
Congestive heart failure	Jugular venous distention, peripheral edema, presence of a third heart sound (S3)
Pneumonia	Productive cough, fever, pleuritic chest pain

In most cases, ARDS appears to be differentiated from pneumonia and congestive heart failure. The following is a table of differences in several factors in ARDS, CHF and pneumonia (Force et al., 2012):

Factor	ARDS	CHF	Pneumonia
Symptoms			
Dyspnea	+	+	+
Hypoxia	+	+	+
Pleuritic chest pain	+/-	-	+
Sputum production	+/-	-	+
Tachypnea	+	+	+
Signs of			
Edema	-	+	-
Fever	+/-	-	+
Jugular venous distension	-	+	-
Rales	+	+	+
Third heart sound	-	+	-
Study			
Bilateral infiltrates	+	+/-	+/-
Enlarged heart	-	+	-
Increased brain natriuretic peptide level	+/-	+	-
Hypoxemia	+	+	+
Localized infiltrates	-	-	+
PAO ₂ /FIO ₂ ratio \leq 300	+	-	-
Pulmonary wedge pressure \leq 18 mmHg	+	-	+
Response			
Antibiotic	-	-	+
Diuretic	-	+	-
Oxygen	-	+	+

Congestive heart failure

Heart failure is a condition in which the heart works less efficiently than usual. Signs and symptoms that can result from this condition include shortness of breath, fluid retention, irregular heartbeat, dizziness, fatigue, and feeling weak. Congestive heart failure is usually caused by conditions that aggravate the function and work of the heart muscle such as coronary heart disease, hypertension, cardiomyopathy, arrhythmias, abnormalities in heart valves, and congenital heart disease. In addition, other causes can cause congestive heart failure, namely anemia, excessive consumption of alcoholic beverages, hyperthyroidism, and pulmonary hypertension (John Hopkins, 2021).

The difference between congestive heart failure and ARDS is that congestive heart failure is characterized by fluid overload, while ARDS does not have a fluid overload. In addition, congestive heart failure found edema, jugular venous distension, S3 gallop, increased brain natriuretic peptide, and responds well to the use of diuretic drugs. While the ARDS did not find these signs (Bakhtiar & Maranatha, 2018).

Pneumonia

Pneumonia is an acute infection of the respiratory tract, especially in the alveolar structures. When a person has pneumonia, the alveoli are filled with pus and fluid, making it painful to breathe and restricting oxygen intake. The most common causes of pneumonia from viral pathogens in adults are human rhinovirus and influenza, while in bacterial pathogens *Streptococcus pneumoniae* is found and there are also bacteria and other viruses (Fishman et al., 2008).

Pneumonia is known to be the most common cause of ARDS so distinguishing ordinary pneumonia and pneumonia with ARDS is a challenge to make a diagnosis, but some of the differences are in patients with ordinary pneumonia, systemic symptoms and pulmonary inflammation are shown in the form of fever, fatigue, chills, sputum production, hypoxia, chest pain. local and multifocal pleuritic and infiltrates. If symptoms such as hypoxia do not improve, it can be suspected that the patient has ARDS (Bakhtiar & Maranatha, 2018).

Management

- a. Recognizing hypoxemic respiratory failure when a patient with respiratory distress is failing oxygen therapy. There is a possibility that the patient has an increased work of breathing or is hypoxemic despite being given facemask oxygen with a reservoir bag (10-15 L/min, Fios between 60-100) (Saguil & Fargo, 2012). Respiratory failure in ARDS results from a ventilation-perfusion mismatch and usually requires mechanical ventilation (Beckerman J, 2020). Ascending ventilation can be in the form of non-invasive ventilation with mask ventilation with support or invasive ventilation via endotracheal or tracheostomy. Non-invasive ventilation (NIV) is not recommended in patients with hypoxemic respiratory failure except for cardiogenic pulmonary edema and postoperative respiratory failure. Because this can cause delays in intubating, the volume is not high, and there is barotrauma (Grief & Loza, 2018).
- b. Ventilation failure is usually accompanied by a decrease in functional residual capacity, which is the volume of air that remains in the lungs at the end of an abnormal expiration due to inadequate respiratory muscles (Kemenkes, 2019). Mechanical ventilation using low tidal volume (4-8 ml/kg BW), and low inspiratory pressure (plateau pressure <30 cm H₂O) Adequate PEEP with FiO₂ safe, avoiding barotrauma (airway pressure < 35 cm H₂O), and adjust the ratio of inspiration: expiration (Force et al., 2012).

- c. Ventilation tidal volume is the volume of air delivered by mechanical ventilation with each breath (Saguil & Fargo, 2012). The tidal volume starts with a low of 6 mL/kg BW which is better to use than a tidal volume of 10-15 mL/kg BW. The literature recommends starting ventilation with a low tidal volume. However, the use of low tidal volumes is associated with a higher partial pressure of carbon dioxide (PaCO₂), but in some cases hypercapnia is allowed a PaCO₂ of 50 mmHg or more. However, the patient should be monitored closely and adjustments should be made while balancing the preference for low tidal volume with the risk of increased PaCO₂. The use of low tidal volumes is considered as supportive therapy for ARDS patients because it can reduce mortality and reduce ventilator time (Kemenkes, 2019).
- d. In patients with moderate or severe ARDS it is recommended to use a higher Positive End Expiratory Pressure (PEEP), PEEP titration is required based on the benefit (reduced atelectrauma and increased alveolar recruitment) and risk (excess pressure at the end of inspiration leading to lung injury). PEEP as a mechanical ventilation model to prevent refractory hypoxemia in ARDS patients and aims to prevent lung damage due to repeated opening and closing of the bronchioles and alveoli so as to prevent lung collapse. Positive End Expiratory Pressure (5-20 cmH₂O) is an important part of ARDS patients, can decrease intrapulmonary shunt and increase arterial oxygenase (Kemenkes, 2019).
- e. In severe ARDS patients, prone position >12 hours per day can be performed. applying the prone position to ARDS patients in adults and children although it is not generally recommended but in 70% of ARDS patients prone position can improve oxygenase, increase PaO₂ significantly, and can be considered if the patient requires PEEP > 12 cm H₂O and FiO₂ > 0.60 and best performed on ARDS with onset < 36 hours. Prone position is recommended for patients with severe ARDS and for patients with moderate ARDS with a PaO₂/FiO₂ ratio of less than 150. If this technique is used the position should be maintained for at least 12-16 hours/day (Force et al., 2012).

Conclusion

Acute Respiratory Distress Syndrome is a rapidly progressive disorder with clinical manifestations such as shortness of breath which then changes rapidly to respiratory failure. ARDS is characterized by bilateral pulmonary infiltration and severe hypoxemia in the absence of pulmonary edema. ARDS is caused by sepsis, systemic disease, and lung injury. ARDS results from acute inflammation affecting the alveolar surface. This causes an increase in membrane permeability thereby activating the action of neutrophils and other acute inflammatory mediators to the lung alveoli. One of the factors causing ARDS is sepsis. ARDS has non-specific symptoms so it is necessary to be more careful in considering or making a diagnosis. There are several differential diagnoses such as asthma, COPD (Chronic Obstructive Pulmonary Disease), Pneumonia, and heart failure.

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