Journal of Respiratory Medicine

Review Article Open Access

Covid-19 and Acute Respiratory Distress Syndrome (ARDS) a Clinical Review

Shuofeng Yuan*

Department of Microbiology, University of Hong Kong, Hong Kong

Abstract

The COVID-19 pandemic is a serious problem of the new era. Acute respiratory distress syndrome (ARDS) and respiratory failure are the major lung diseases in COVID-19 patients. Although a COVID-19 vaccine is now available, there is still an urgent need to find potential treatments to reduce the impact of her COVID-19 on already sick patients. Several experimental drugs have been approved by the FDA, but their efficacy and possible side effects are unknown. An increasing number of studies worldwide investigating potential COVID-19-related therapies may help identify effective ARDS treatments. This review article first provides an overview of the immunopathology of ARDS. He then outlines the management of his COVID-19 patient requiring intensive care unit (ICU), focusing on current treatment strategies being evaluated in clinical trials for patients with COVID-19-induced ARDS.

Keywords: Respiratory; Alveolar-capillary; Chest trauma; Population

Introduction

Acute respiratory distress syndrome (ARDS) is a common but complex syndrome that occurs in critically ill patients. Clinically, ARDS presents as acute hypoxemia and the presence of bilateral pulmonary infiltrates that are not entirely due to heart failure or fluid overload. This flooding of alveoli with protein-rich edema is associated with the collapse of alveolar-capillary units and, in many cases, the influx of neutrophils and other immune cells into the airspace [1, 2]. These immune cells, along with activated epithelial and endothelial cells, release numerous proinflammatory mediators that propagate a wide range of inflammatory responses and various cytotoxic species that damage the lung parenchyma. Although this damage to endothelial and alveolar cells due to fluid and cellular exudation has been termed diffuse alveolar injury (DAD), data from biopsy and autopsy studies meet the clinical definition of ARDS shows that only half of the person has his DAD [3]. The presence of DAD is associated with increased mortality in ARDS. Despite over 50 years of research, ARDS is still poorly recognized and no specific effective treatments are available. Mortality from ARDS remains high, with most estimates pointing to mortality rates in the 30-50% range [4]. ARDS is a heterogeneous disease process that can be caused by a variety of direct or indirect lung injuries, including pneumonia, aspiration, unproductive ventilator, chest trauma, sepsis, and acute pancreatitis. ARDS can affect people of all ages, but increasing age is a common risk factor and is associated with increased mortality in ARDS patients. This relationship has been underscored by the current Covid-19 pandemic, in which the causative agent of ARDS, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has shown significant mortality in elderly patients [5, 6].

The world population is aging. Those aged 65 and older are projected to make up one in six of hers in the population by 2050, up from one in her 11th in 2019 [7, 8]. There is a strong association between aging and increased incidence of diseases, including respiratory diseases such as chronic obstructive pulmonary disease and acute infections. Good understanding age-related lung changes and how these influence disease risk and severity is critical to effectively managing the health of older adults [9]. Structural changes in the lungs with aging mainly include alveolar space enlargement, decreased elasticity and progressive decline in lung function. Accumulation of senescent cells in the lungs is observed in the elderly population.

Clinical Characteristics of Aging in ARDS

Various etiologies can cause the clinical syndrome of ARDS. These were divided into direct and indirect causes of disease. Direct causes include those that cause temporary damage to the lung epithelium, such as: Examples: pneumonia, aspiration, poison inhalation, drowning. Indirect causes include those that cause lung injury in the context of systemic inflammation and diffuse damage to the pulmonary vascular endothelium [10, 11]. These include, but are not limited to, extrapulmonary sepsis, noncardiogenic shock, trauma, blood transfusion, pancreatitis, drug overdose, and vasculitis [12]. ARDS occurs after one of these attacks elicits a dysregulated systemic host inflammatory response in the lung, usually within the first 12–48 hours after exposure. Diffuse damage to the alveolar-capillary membrane leads to void and interstitial edema with the development of protein-rich neutrophilic exudate that impairs gas exchange and reduces lung compliance [13, 14].

The hallmark of ARDS is diffuse alveolar damage (DAD) has been described as a sign of damage to the alveolar mucosa and endothelial cells with characteristic findings of hyaline membranes [15]. Pathologic findings early in the disease course include capillary congestion, intraalveolar edema, atelectasis, and hemorrhage [16]. In later stages of the disease, mononuclear cell infiltrates invade the alveolar space, resulting in interstitial fibrosis. Patients with direct and indirect ARDS have different pathological findings [17]. Lung samples from patients with direct pulmonary causes of ARDS were found to have higher levels of her DAD than samples from patients with non-pulmonary causes [18]. Investigators examining postmortem lung specimens from patients with ARDS found that patients with direct causes of ARDS had more alveolar collapse, fibrous exudate, and alveolar wall damage compared with those with indirect causes of ARDS [19]. We found that

***Corresponding author:** Shuofeng Yuan, Department of Microbiology, University of Hong Kong, Hong Kong, E-mail: yuan.shoufeng12@hotmail.com

Received: 01-Sep-2022, Manuscript No: jrm-22-74754; **Editor assigned:** 05-Sep-2022, Pre-QC No: jrm-22-74754 (PQ); **Reviewed:** 19-Sep-2022, QC No: jrm-22-74754; **Revised:** 22-Sep-2022, Manuscript No: jrm-22-74754 (R); **Published:** 30-Sep-2022, DOI: 10.4172/jrm.1000142

Citation: Yuan S (2022) Covid-19 and Acute Respiratory Distress Syndrome (ARDS) a Clinical Review . J Respir Med 4: 142.

edema predominates. Another similar analysis of autopsy specimens from patients in the early stages of disease showed higher collagen content in specimens from patients who had a direct pulmonary cause of ARDS, suggesting that the early stage of the extracellular matrix was consistent with remodeling [20]. Animal models show that direct lung injury is associated with greater alveolar inflammation and is associated with more damage and impaired lung function [21, 22]. Understanding the differences in epithelial and endothelial damage between each type of ARDS injury will help align new goals of treatment beyond current ventilator methods [23].

Clinically, mortality rates in patients with direct and indirect ARDS have been shown to be similar (28% and 31%, respectively)

Impact of Age on Treatment Strategies and Specific Biomarkers

Biomarkers are important tools in diagnosis and patient stratification, but to date, no single specific ARDS biomarker has been validated. Analysis of systemic inflammatory mediators and endothelial activation markers in plasma from adults with ARDS revealed that in elderly patients, inflammatory mediators and endothelial activation markers (interleukin (IL)-6, IL-8, IL-10) [24], revealed relatively low levels of interferon. Young patients receive gamma, fractalkine, intracellular adhesion molecule (ICAM)-1, E-selectin), and high concentrations of platelet factor-4 and tissue plasminogen activator (tPA). Furthermore, aging was found to be independently associated with increased plasma levels of tPA and decreased plasma levels of fractalkine and her E-selectin. However, only tPA was associated with outcome and accounted for 10% of the association between age and outcome. These findings suggest a possible link between aging, fibrinolysis, and mortality, but further studies are needed to determine the role of tPA. Recently, Schouten et al. [25] We analyzed age-dependent differences in inflammatory and endothelial activation markers in BALF from ARDS patients of different ages. Neutrophil marker levels increased with age, with increasing age and decreases in MPO, IL-10, P-selectin, and ICAM-1, although no differences were found between adult and elderly cohorts. The association with was still significant thereafter. These data suggest that plasma may be more useful in identifying age-related differences in her ARDS patients, but to shed light on pulmonary and pulmonary pathophysiology, Longitudinal analysis of her BALF and plasma from the same patient cohort may be required. Systemic responses and how they interact across the spectrum of ARDS phenotypes. As with other diseases, variations in published data demonstrate the difficulty in identifying biomarkers associated with disease severity and outcome in this heterogeneous syndrome, suggesting a broader 'omic' (such as proteomics) approaches may be required [26]. Identifying specific biomarkers can help identify those most at risk in the elderly population and determine potential treatment options, but this is a difficult endeavor.

There is no conclusive evidence of age-related differences in treatment response. However, there is evidence from both in vivo and computational studies to suggest that ventilator-induced lung injury (VILI) may be more common in the elderly due to increased lung compliance and stiffness [27]. Conservative fluid management and the use of low tidal volume ventilation (LTVV) have been shown to mitigate the age-related increase in ventilator-related mortality in vivo. Clinically, the benefits of LTVV are uncertain and adherence among physicians is low. Further research on the clinical benefits of LTVV, especially in the elderly subpopulation, would be worthwhile. Extracorporeal membrane oxygen therapy (ECMO) has emerged as a valuable treatment to aid recovery from ARDS [28]. Data on his use of ECMO in older adults are limited, with most data coming from retrospective studies. Current data suggest that age is not a contraindication for his ECMO. Rather, usage should be determined on a case-by-case basis. Potential predictors to consider before initiating ECMO support include the presence of cardiogenic shock, APACHE II, and SAPS II scores. Accumulation of senescent cells has been proposed as a mechanism for increased mortality in the aged Her ARDS population [29]. Therefore, the use of senolytics may be an interesting therapeutic modality to improve outcomes in elderly patients with ARDS. Hemolytic agents have not yet been evaluated clinically in ARDS, but have shown benefit in preclinical fibrosis models. In the future, identifying individuals at the highest risk of developing ARDS, as well as identifying the most effective treatment strategies for phenotypically and demographically diverse patient populations, will help reduce mortality in the older her ARDS population is most important in reducing.

Conclusions

Previous studies have highlighted the significant analytical challenges posed by ARDS due to its complex etiology. Aging may alter the immune system, vasculature, and airway epithelium to facilitate the pathogenesis of ARDS, but much more work is needed to elucidate these mechanisms, and this is likely due to aging. is important for understanding the impact of on improving the risk of developing ARDS and its severity. Although further studies are needed, increased oxidative damage due to age-related changes, defects in autophagy, and senescent cell accumulation associated with increased vascular permeability may play a role. A better understanding of the contribution of these individual factors, along with the potential impact of comorbidities, may help guide future treatment strategies. A clear understanding is important to reduce mortality in this subpopulation. Cell therapy is a promising therapeutic option for ARDS, and these may be of particular importance in the elderly population, where aging and immunosenescence render host cells ineffective and the lungs exhibit age-related structural changes. Treatment with mesenchymal stem cells, endothelial progenitor cells, or Tregs can reduce age-related pathologies. Alternatively, it may be worth slowing the aging process to study ARDS decline with age. Indeed, caloric restriction slowed the rate of aging and reduced the risk of ARDS in preclinical models. Other potential therapies targeting aging are also being investigated in ARDS, such as rapamycin, mTOR inhibitors and metformin. I can do it. These therapies have been recently reviewed and represent many potential avenues for developing more targeted therapies for older ARDS patients. Stratification into phenotypes has revolutionized the clinical perspective. Studies involving multiple ARDS cohorts have identified distinct subphenotypes based on plasma inflammatory markers, degree of shock, and metabolic acidosis. These subphenotypes appear to respond differently to treatment, and although age does not differ significantly among subphenotypes in most of these cohorts, the role of age as a determinant of the ARDS subphenotype may be questioned. Focused further research may provide useful strategies for direct therapy. Alternatively, identifying subphenotypes within the elderly ARDS patient population may also prove useful, allowing stratification of these patients.

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