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Removal of Endotoxins and Cytokines by Plasmapheresis Filtration with Plasma Exchange Therapy (TPE) Could Benefit Patients with Covid-19 in Critical Condition

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Abstract

Background: The COVID-19 pandemic originated in Wuhan, China is rapidly and continuously spreading globally and can result in significant severe respiratory morbidity and mortality. The severe damage of the lung tissue can result in acute respiratory distress syndrome (ARDS), which can further precipitate septic shock. These two complications are the major contributors to the intensive care unit (ICU) care and mortality from COVID-19 in patients older than 60 years, with smoking history, and comorbid medical conditions. The possible high mortality rate of COVID-19 can be connected to the increasing of Th17 cells activity mainly stimulated by IL-6 and IL-23.

Discussion: The mitigation of morbidity risk in patients with COVID-19 infection in the progression of acute respiratory distress syndrome (ARDS) related to severe respiratory tract damage with the high blood level of inflammatory markers as Cytokines IL-6 is an essential goal of contemporary treatment these patients in ICU environment. The Plasma Exchange therapy (TPE) with continuous hemofiltration (CHF) reduced level IL-6 and other cytokines in patients with different critical pathologies as fulminant liver damage, autoimmune inflammation, neurological and infectious diseases.

Conclusion: CHF with TPE treatment can be one of the ways of concomitant therapy in the standard of care for COVID-19 infection patients with developing respiratory distress syndrome. However, further investigations are necessary in order to corroborate this successful therapy.

Keywords: COVID-19; Cytokines storm; IL-6; ARDS; Plasmapheresis; TPE;Plasma exchange therapy; Hemofiltration

Abbreviations: ASFA: The American Society for Apheresis; COVID-19: Coronavirus Disease 2019; ICU: Intensive Care Unit; ARDS: Respiratory Distress Syndrome; CRS: The Systemic Inflammatory Response; TPE: Therapeutic Plasma Exchange; CHF: Continuous Hemofiltration; CHDF: Continuous Hemodiafiltration; PF: Plasmafiltration; IL-16: Interleykin-16; IL-23: Interleykin-23; Th17 , Th-0: T- Helper Lymphocytes-17(0)

Introduction

The COVID-19 pandemic originating in Wuhan, China, is rapidly and continuously spreading globally and can result in significant severe respiratory morbidity and mortality [1]. The responsible agent, SARS-CoV-2, is an enveloped RNA virus of the Coronaviridae virus family. Human-to-human transmission occurs through respiratory droplets or contaminated surfaces [1]. The average incubation period is five days but ranges from 1–14 days. The pathogenesis of COVID-19 has been examined since the beginning of the viral pandemic. However, a lot of unclear parts of the knowledge remains and are waiting for the rigorous research and analysis. The goal of the world Medical Research Institutions is to mitigate the vast spread morbidity of the Coronavirus and possibly prevent the high mortality and complications of the virus in a wide variety of high-risk patients [2].

Pathology of the COVID-19

The infection caused by the virus SARS-CoV-2 is termed as Coronavirus Disease 2019 (COVID-19). The symptomatology of COVID-19 was extensively discussed in WHO-China joint report on

COVID-19 (n = 55,924) [3]. Patients with COVID-19 present with pyrexia in 85% of cases during their illness course, but only 45% are febrile on early presentation [4]. Moreover, cough is seen in 67.7% of patients, and sputum is produced by 33.4%. Respiratory symptoms such as dyspnea, sore throat, and nasal congestion present in 18.6%, 13.9%, and 4.8% of cases, respectively [4]. Constitutional symptoms such as muscle or bone aches, chills, and headache are seen in 14.8%, 11.4%, and 13.6% of the cases, respectively [4]. Gastrointestinal (GI) symptoms such as nausea or vomiting and diarrhea are seen in 5% and 3.7% of the cases, respectively. These clinical manifestations of COVID-19 were consistent in other similar studies on COVID-19 patients in China (n = 41, n = 81, n = 99, n = 138) [5-8]. The severe damage of the lung tissue can result in acute respiratory distress syndrome (ARDS), which can further precipitate septic shock. These two complications are the major contributors to the intensive care unit (ICU) admittance and mortality from COVID-19 in patients older than 60 years, with smoking history, and comorbid medical conditions [1]. The first COVID-19 pathology found bilateral diffuse alveolar injury with the cytomyxoid fiber exudate, and disproportional increase in Th17 cells count with the reduction in CD4 and CD8 lymphocytes in the peripheral blood [9]. Th17 cells are helper T cells differentiated from Th0 cells, mainly stimulated by IL-6 and IL-23 [10]. The hyper-Th17-emia is related to the high level of cytokines as IL-6 and IL-23 in plasma of COVID-19 patients. It is The Systemic Inflammatory Response (CRS) that can be followed by ARDS's development [10-13]. CRS is more common in immune system-related diseases or immune-related therapy, such as CAR-T cell therapy, organ transplantation sepsis [14], and virus infection. The SARS-CoV-2 binds to alveolar epithelial cells, then the virus activates the innate immune system and adaptive immune system, resulting in the release of a large number of cytokines, including IL-6. Also, due to the role of these pro-inflammatory factors, vascular permeability increased, a large number of fluid and blood cells into the alveoli, resulting in dyspnea and even respiratory failure [15-18]. The highest risk in the mortality rate is in the group of patients older than 60 years, with smoking history, and comorbid medical conditions, or predisposed COPD, cardiovascular pathology, or Diabetes [19,20,4,6]. Cardiovascular injury is significantly associated with fatal outcome of COVID-19, while the prognosis of patients with underlying CVD but without myocardial injury is relatively favorable. Myocardial injury is associated with cardiac dysfunction and arrhythmias. Inflammation may be a potential mechanism for myocardial injury. Aggressive treatment may be considered for patients at high risk of myocardial injury [19,20]. There are several reports underline the importance of mitigation of liver dysfunction in COVID-19 patients [2]. Different Therapeutic approaches are used in the mitigation of the CRS and Cytokine Storm. There are several directions in the prevention of CRS as a leading cause of mortality and cardiovascular and respiratory truck complications. Some researchers use a pharmacological way to inhibit IL-6 activity. Growing information also suggests that virus-induced cytokine storms in the lungs may drive severe pathogenesis and provide potential therapeutic targets, for example, anti-IL6 or anti-IL-1 approaches [21]. The FDA has recently approved the use of hydroxychloroquine in COVID-19 patients, but the efficacy remains to be determined. Another possibility to reduce the cytokine storm consequences is to pharmacologically target the Notch signaling as a mediator of viral activity [22]. The efficacy and safety of this therapeutic approach should be demonstrated in followed studies.

The cell-based therapies are another direction of COVID-19 treatment. These utilize a range of different cell sources, doses, dosing strategies, and targeted patient populations. A range of approaches have been utilized for dose, dosing, and MSC source with MSCs of bone marrow (BM), adipose, umbilical cord, cord blood, and the placenta being investigated. A recent systematic review indicated that BM and UC-MSCs were more effective than adipose tissue-derived MSCs in reducing mortality in pre-clinical acute lung injury models [23,24]. However, there is no apparent pre-clinical data to support the rationale for any of these approaches [25]. The extracorporeal blood purification therapy as plasmapheresis (Continuous Hemofiltration- CHF) with- or-without plasma exchange therapy (TPE) is a possible way to reduce the Cytokine storm outcomes.

Another therapeutic way to reduce the Cytokines storm could be a physical removal of the cytokines from the bloodstream by using the extracorporeal blood purification therapy as Plasmapheresis with- or- without plasma exchange therapy (TPE). As the American Society of Apheresis (ASFA) states, the Plasmapheresis is performed by two fundamentally different techniques: centrifugation or filtration [26]. With centrifugation apheresis, whole blood is spun so that the four major blood components are separated into layers by their different densities. With filtration plasmapheresis, whole blood passes through a filter to separate the plasma components from the more significant cellular components of red blood cells, white blood cells, and platelets. Heparin is usually used for the prevention of early and late coagulation adverse effects. Historically, centrifugation apheresis is commonly performed by blood bankers. A significant advantage is that there is no limit on the size of the molecules being removed. Its disadvantage is that it usually requires a consultation with another service such as a blood banker. Nephrologists and intensivists commonly perform Continuous Hemodiafiltration- (CHDF) or Filtration Plasmapheresis (PF) with Hemodialysis. It is a significant advantage that a larger pores filter can be added to the existing continuous veno-venous hemodialysis circuit without much interruption to patient care. The size of the molecules removed is limited by the size of the pores of the filter [26,27]. Continuous Hemofiltration- (CHF) is the extracorporeal procedure of plasma separation from the blood in a centrifuge with the following plasma filtration through the pored plastic filter that washes out molecules, proteins, and toxins with specific molecular weight and size. It is possible to choose the small,

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middle, or big pore filters to direct the process of filtration. TPE includes albumin infusion for the restoration of plasma volume and protein depletion, followed by the Plasmapheresis [26]. The anti-inflammatory effect of extracorporeal blood purification as Plasmapheresis is very well known and used in different morbid conditions like liver failure, progressive immune diseases as Corticosteroid-Refractory Eosinophilic Granulomatosis [28], Guillain-Barré Syndrome in Children [29,12] or drug-resistant bronchial asthma [30]. The positive results of the CHF with TPE in patients with severe inflammation with a high level of cytokines were published in 2015 [31]. The significant reduction in pre- vs. post-TPE plasma concentrations for sICAM-1 (517 ± 246 vs. 260 ± 159ng/ml, $p < 0.0001$), sTNF-R (8.1 ± 6.4 vs. 5.7 ± 3.9ng/ml, $p < 0.05$), and resisting plasma levels (14.3 ± 6.9 vs. 9.5 ± 4.7ng/ml, $p < 0.001$) using albumin as exchange fluid received in two consecutive TPE sessions [31]. Another study showed significant improvement in the level of adiponectin in patients with acute liver failure treated with plasma filtration with dialysis (CHDF) and plasma exchange therapy (TPE) [32]. The meta-analysis of several controlled studies was published in 2020 showed that high volume plasma exchange therapy (TPE) with CHF was used successfully in patients with acute-on-chronic liver failure (ACLF) with a high level of cytokines and endotoxins in the blood. The analysis showed improvement of survival at 30-and 90-d in nontransplant patients with fulminant hepatitis related to the use of TPE with CHF [33]. In some cases, despite the improvement of clinical representation after TPE, the concentration of IL-6 was not decreased after one treatment [34]. Plasma Exchange therapy (TPE) with continuous hemodiafiltration (CHDF) was clinically successful in patients with critical hypercytokinemia [34]. TPE, in combination with CHDF therapy, was given to 5 patients with acutely aggravated autoimmune diseases, two patients with hemorrhagic shock and encephalopathy syndrome, and three patients with thrombotic microangiopathy. All patients had clinical improvement after apheresis therapy. TPE alone was less effective than in combination with CHDF. The TNF-α and IL-8, but not IL-6 level were significantly lower after treatment in the TPE+CHDF group [34]. The plasma concentration of peptides, lipids, or other biochemical substrates can be higher after apheresis instead of a reduction in the first two weeks after the procedure. This phenomenon of "the rebound-effect" was described previously in some studies [35,36]. The concentration of antibodies to Adeno-associated Virus (AAV) in one study [35], and serum cholesterol in familial hypercholesterolemia case in other studies [36] was increasing during two after the first Plasmapheresis. Lately, the concentration of substrates reduced after followed additional Plasmapheresis [36]. We may suggest that the increasing concentration of biological substrates could be the result of the inflow of the peripheral plasma to the central bloodstream from other biological compartments as interstitial fluid and lymph.

The TPE has been beneficial in the treatment of the cytokine storm and to provide hematologic support in patients with primary and secondary Hemophagocytic Lymphohistiocytosis: Pathologic Hyperactive Inflammation (HLH) [37]. Several publications support this research results [38-41]. Nakakura et al. published a case report of successful treatment with plasma exchange for hemophagocytic syndrome associated with severe systemic juvenile idiopathic arthritis in an infant girl [42]. Sanada S, et al. published case report of the positive effect of plasma exchange on reactive hemophagocytic syndrome associated with toxic shock syndrome [43]. Nusshag K et al. reported a positive effect of Plasmapheresis on hemophagocytic lymphohistiocytosis syndrome in an adult kidney transplant recipient [44]. Investigators Song KS and Sung HJ reported a reduction of concentration IL-6 in plasma of patients with fatal hemophagocytic syndrome associated with ductopenia [45]. The same results reported in the case of the Hemophagocytic Lymphohistiocytosis Induced by Severe Pandemic Influenza A (H1N1) 2009 Virus Infection [46]. A recent small study found significantly improved survival in patients with secondary HLH who received plasma exchange, steroids, and IVIG ($n = 17$) versus those who received plasma exchange, steroids, and cyclosporine, and/or etoposide ($n = 6$). Currently, the ASFA has not commented on the use of TPE in HLH. Further research is warranted for this complicated therapeutic strategy [27].

Discussion

Follow the many reports stated that the Plasmapheresis was used last 30 years in different countries for patients in critical condition to remove the increased level of Cytokines and Toxins in the bloodstream to prevent developing an acute immune reaction and high rate of mortality of this category of ICU patients, we may suggest that using Plasmapheresis (CHF with TPE) in patients with COVID-19 can mitigate the cytokine storm and prevent developing of CRS as a leading cause of mortality and cardiovascular and respiratory tract complications. We know that the Interleukin-6 and 23(IL-6, IL-23) are members of the polypeptide cytokines with a four–α-helix structure and a molecular mass of 21 to 54.1 kDa. (From Rheumatology (Sixth Edition, 2015) [47]. As a practical recommendation, we can suggest that the medium/large-pore (50/100 kD) membrane the continuous Hemofiltration (CHF) [48] with followed albumin returning in plasma exchange therapy TPE can be presumably helpful in removing molecules of IL-6 and Il-23 from the COVID-19 patients' plasma. By the fact that the benefits of using apheresis (CHF with TPE) in patients with COVID-19 are hypothetical and are not well studied, we can recommend the use of this treatment by the individualized decision. It is reflected in the Category III of The American Society for Apheresis (ASFA) "Guideline on the Use of Therapeutic Apheresis in Clinical Practice-Evidence-Based Approach," 2016: "Optimum role of apheresis therapy is not established. Decision making should be individualized" [49].

Conclusion

The mitigation of morbidity risk in patients with COVID-19 infection in the progression of acute respiratory distress syndrome (ARDS) related to severe respiratory tract damage with the high

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blood level of inflammatory markers as Cytokines IL-6 and IL-23 is an essential goal of contemporary treatment these patients in ICU environment. Plasmapheresis (continuous hemofiltration CHF) with therapeutic plasma exchange (TPE) can be one of the ways of concomitant therapy to the standard of care for such a category of patients. However, further investigations are necessary in order to corroborate this therapy.

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