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Lymphocytes in COVID-19 Patients

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ABSTRACT

Coronavirus disease 2019 (COVID-19) has so far caused over 108.2 million confirmed cases and over 2.3 million deaths all over the world as of February 14, 2021. Among in-hospital patients with COVID-19, the mortality was approximately 28%, though, the percentage increased to over 60% among critically ill patients, and over 80% among those who require mechanical ventilation. Treatment of these severe patients is becoming one of the major challenges. It has been hypothesized that a cytokine storm is the main cause of disease progression which leads to acute respiratory distress syndrome and organ failure. For this reason, corticosteroids and/or immunomodulatory drugs have been lengthily used during the SARS-CoV-2 pandemic. However, the clinical efficacy of corticosteroids remains controversial. Moreover, corticosteroid therapy was described to stay the clearance of SARS-CoV-2, and a high dose of corticosteroid was found to be related with death in severe COVID-19.3 Therefore, to classify patients most likely benefit from corticosteroid and give precise corticosteroid therapy is essential for the management of severe COVID-19 and saving lives.

Keywords: SARS-CoV-2; Lymphocytes; Patients

INTRODUCTION

Patients were defined as severe cases when they met any of the following criteria: Respiratory distress, respiratory rate>30/min; Mean oxygen saturation ≤ 93% in the resting state; Arterial blood oxygen partial pressure (PaO2)/oxygen concentration (FiO2) ≤ 300mmHg; Lung involvement on imaging>50% within 24-48h. Patients who died within the first 48h of admission were excluded because it was difficult to evaluate the effectiveness of corticosteroids.

COVID-19 patients and stratified by factors including age, hypertension, C-reactive protein (CRP), D-dimer, high sensitivity cardiac troponin I, and lymphocyte count. Incessant variables were rehabilitated to categorical variables. Communication effects were also examined between outcomes and selected variables. The dosage and period of corticosteroids used for the patients were selected according to age, clinical symptoms, comorbidities, and lab tests. There was no significant difference in age, blood pressure, heart rate, respiratory rate, symptoms, comorbidities between the two groups. However, patients with corticosteroid treatment had significantly lower levels of platelet, lymphocyte, monocyte, albumin, hs-cTnI, and significantly higher neutrophil, transaminase, CRP, and D-dimer levels. In consistent with previous studies, older age, leukocytosis, cardiac injury, high levels of CRP and D-dimer, and lymphopenia were associated with the death of severe COVID-19.

The improved outcomes associated with corticosteroid in patients with lymphocytopenia is intriguing and maybe clinically important. In severe COVID-19, lymphocytes were significantly reduced, and lymphopenia was considered a potential indicator for disease severity, therapeutic response, and disease outcome. It was speculated that high levels of proinflammatory cytokines in cytokine storm syndrome such as tumor necrosis factor (TNF)-α and interleukin-6 could induce lymphocyte deficiency. Improved outcomes with corticosteroids in this population may indicate benefit in this hyperinflammatory situation. Patients with higher lymphocyte counts were less likely to have CSS and may experience more harm than benefit when receiving corticosteroids. Furthermore, it has been suggested that early low-dose corticosteroid therapy (within 14 days from onset of symptoms, 40 mg) accelerates the recovery of CD8+T cells in critical COVID-19 patients

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CONCLUSION

Lymphocyte count could be a potential indicator for the identification of severe COVID-19 patients who may benefit from corticosteroid therapy. Future studies on the role of these clinical indicators in guiding corticosteroid therapy and to predict clinical response are needed.

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