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Letter to Editor

What Can We Learn From Kawasaki Disease To Treat COVID-19 Patients?

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There is currently no treatment proven to effectively treat COVID-19 disease, or to prevent the deleterious complications that occur in some patients, up to the time of writing this letter (11th April 2020). The pathogenesis is poorly understood and is based on our understanding of the pathogenesis of previously identified SARS-CoV and MERS-CoV. The viral antigen is presented after entry through the ACE-2 receptors to the antigen presenting cells, which is responsible for activation and production of specific IgM and IgG, indicating that IgG antibodies play a protective role [1].

The most common immunopathological event known so far for SARS-CoV-2 is the cytokine storm, which is known to release large amount of pro-inflammatory cytokines, as a result of triggering a violent attack of the immune system. Consequently, acute respiratory distress syndrome (ARDS) is developed [1].

While the immune response is critical for the body's response against the virus, viral antigen-host interaction may produce a set of immune mediators against the virus [2]. A report described COVID-19 pneumonia corpse, indicated that the disease triggered an inflammatory response in the lower airway and led to lung injury, then infected other cells, and stimulated the release of series of immune mediators and the production of cytokine storm, which characterizes the critical illness of some COVID-19 patients [3].

On the other side, Kawasaki disease (KD) is an acute febrile illness predominating in children, characterized by systemic inflammation in all medium-sized arteries and multiple organs and tissues. The main symptom include fever for 5 days and disease onset starts in 5 - 7 days after fever onset. The trigger is unknown, but data suggest an infectious antigen, mainly a virus that enters through the respiratory tract, triggering a cascade of innate and adaptive immunity, which enhance circulatory pro-inflammatory cytokine release, including marked elevation in IL-6 and regulatory T-cells [4]. Cytoplasmic viral inclusion bodies were found in the bronchial epithelium of KD patients, similarly found in a COVID-19 critical patient with pneumonia [5].

The efficacy of high dose IVIG in KD is well recognized to resolve the fever and the systemic manifestations induced by the pro-inflammatory cytokines, and to decrease mortality. Possible identified mechanism of IVIG is to modulate the pro-inflammatory cytokine production, and to augment the activity of regulatory T-cell population [4]. Steroids were found to benefit patients with refractory KD and pneumonia.

In conclusion, there are many similarities in the pathogenesis of COVID-19 and KD, we propose that high dose IVIG (at 2 g/kg/dose infused over 10 - 12 hours) is considered in the treatment of high risk COVID-19 patients, especially those with no improvement or worsening of symptoms.

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