COVID-19: role of the NLRP3 inflammasome

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Abstract

Severe acute respiratory syndrome coronavirus 2 (SARS CoV-2) pandemic represents a very serious health public concern. This pathogen causes Coronavirus Disease-19 (COVID-19). It is a clinical condition, asymptomatic or paucisymptomatic in most of the patients, although, in a variable number of cases, it can be characterized by the development of very serious complications and by death. The pathogenetic mechanisms, responsible for this syndrome, are not fully understood and further studies are needed. However, according to our knowledge, after the early phase of viral infection, interactions between SARS-CoV-2 and host take place, and a state of immune system response dysregulation and hypercoagulability occurs. The individuals affected by SARS-CoV-2 infection experience an inflammatory response of different magnitude. In this review, we examine the events originating during COVID-19 disease and focus mainly on the activation of NLRP3 inflammasome and IL-1β as well as on the generation of the excessive systemic inflammatory response with the development of endotheliitis, thrombosis, and micro-thrombosis, detectable in this syndrome. Furthermore, we describe in brief the process of immune system dysregulation associated with SARS-CoV-2 infection and the onset of some life-threatening conditions, such as Acute Lung Injury (ALI), Acute Respiratory Distress Syndrome (ARDS), and ventilator-induced lung injury (VILI), which are observed in some patients with COVID-19.

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