The passepartout of Covid-19, Cytokine storm and Kounis syndrome: Pathophysiologic, Clinical and therapeutic considerations

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Abstract

Background: Coronavirus Disease 2019 (Covid-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), along with its cardiovascular, gastrointestinal, hematologic, mucocutaneous, respiratory, neurological, renal and testicular manifestations and further complications constitutes one of the deadliest pandemics in modern history. A common pathogenetic mechanism of these complications seems to be the Covid-19-induced excessive immune response of uncontrolled release of interleukins, chemokines, interferons, tumor necrosis factors and colony-stimulating factors, the so called cytokine storm syndrome. Severe anaphylactic reactions with profound hypotension or hypoxemia can be also associated with release of pro-inflammatory cytokines. Aim: Careful investigation for similarities in clinical manifestations and correlated multi-organ complications of Covid-19 with other viral infections including dengue and human immunodeficiency viruses together with the action of inflammatory cells inducing the Kounis syndrome could provide a better understanding on pathophysiolgy and trigger mechanisms, elucidating potential preventing and therapeutic strategies. Methods: A search was performed in Medline (via PubMed), for current literature on the pathophysiology, causality, clinical appearance, variance, prevention, and treatment of Covid-19, anaphylaxis with profuse hypotension and the Kounis anaphylaxis associated acute coronary syndrome. Results: Insights from research in allergy/anaphylaxis-associated cardiac syndromes and Covid-19, suggest that the same key immune cells are involved in cardiovascular complications of Covid-19 and the anaphylaxis-associated Kounis syndrome. The myocardial injury in patients with Covid-19 has been attributed to coronary spasm, plaque rupture and microthrombi, hypoxic injury or cytokine storm and shares the same patho-physiology with the 3 clinical variants of Kounis syndrome. Conclusion: The patho-physiology, etiology, clinical manifestations and therapeutic approaches of the severe Covid-19 and their associations might be proved ben-

eficial for future treatments. Early immunological interventions targeting inflammatory markers that are predictive of worse disease outcome would be more beneficial than those blocking late-appearing cytokine related storm. Individualized, tailored to each patient treatment approach is required in Covid-19 cases

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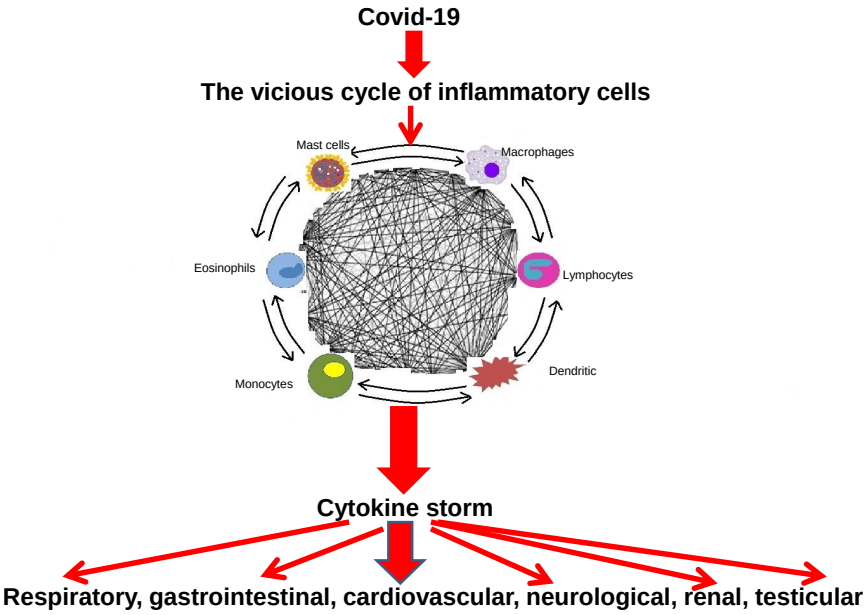

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