NSAIDs Immunomodulation in COVID-19 Might Inhibit SARS CoV-2 ORF Proteins Induced Caspase Activation, Necroptosis and Endoplasmic Reticulum Stress.

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Abstract

We have previously suggested numerous immunomodulatory and anti-inflammatory benefits when NSAIDs are administered to manage COVID-19 and in this commentary, we add other potential benefits related to SARS CoV-2 ORF proteins dependent activation of caspases with subsequent mitochondrial dysfunction, endoplasmic reticulum stress and necroptosis that were described with complicated COVID-19 as NSAIDs are known to be caspase inhibitors. Moreover, NSAIDs might independently inhibit other COVID-19 associated downstream pathological signaling mechanisms. We also postulate that CARD-14, a caspase recruitment domain-containing protein, polymorphisms might play a role in development of severe and critical COVID-19. We believe that it is very unfortunate that for more than one year of relentless struggle, our recommendation to adopt NSAIDs as first choice COVID-19 therapy has not adopted while lives are lost are succumbed every day.

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