Pathophysiology of Long-COVID

The mechanism behind the causation of Long-COVID syndrome may be multifactorial. Immune response, antibody generations, direct effects of the virus, complications of the critical illness, psychosocial factors, and post-intensive care syndrome, post-traumatic stress, and oxidative stress may be operative mechanisms. Cardiac deconditioning may also be a factor.

The mechanism of heart failure involving pro-inflammatory cytokines with interleukin 1 and interleukin 6 tumor necrosis factors may cause prolonged effects. Redox imbalance linking COVID-19 and chronic fatigue syndromes and systemic inflammation and neuroinflammation have also been postulated. Oxidative phosphorylation may be operative in a hyper inflammatory state with altered cardiorespiratory function. It is thought that viral infections cause a shift in mitochondrial energy system contribution from ATP synthesis to innate immune signaling occurring in order to eradicate pathogens, promote inflammation, and eventually restore tissue homeostasis. An increased rate of glycolysis and downregulation of oxidative phosphorylation are seen. Oxidative stress has been implicated in many acquired myocardial disorders and may lead to significant autonomic dysfunction.

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