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Cardiovascular Post-Acute-COVID-19-Illness Sequelae

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At 60 days of the following-up, chest pain was present around 20 % of the COVID-19 survivors [1, 2], whereas at 6 months following-up in the post-acute-COVID-19 Chinese study revealed ongoing chest pain and palpitations in 5 % and 9 % of the COVID-19 survivors, respectively [3]. Ongoing myocardial inflammation may occur at the rates as high as 60 % more than two months after the diagnosis by magnetic resonance imaging (MRI) [4]. The perpetuated mechanisms in post-acute-COVID-19-illness cardiovascular sequelae include SARS-CoV-2 (COVID-19) viral invasion, the immunologic response and inflammation affecting the structural integrity of the cardiac conduction system, pericardium, and myocardium, and downregulation of ACE 2. Autopsy studies in 39 COVID-19 cases (62.5 %) revealed SARS-CoV-2 (COVID-19) viral particles in the cardiac tissues [5] that may contribute to the cardiomyocyte death and fibro-fatty displacement of desmosomal proteins that is critical for cell-to-cell adherence [6, 7]. Persistently increased cardiometabolic demand may be occur in recovered COVID-19 patients that may be related to decreased cardiac reserve, dysregulation of the renin-angiotensin-aldosterone system (RAAS) [8]. SARS-CoV-2 (COVID-19) can induce heightened catecholaminergic state due to cytokine storming from particular cytokines, such as IL-1, IL-6, and TNF-α, that can prolong ventricular action potentials by modulating cardiomyocyte ion channel expression [9], in addition to the induction of resultant cardiomyopathy from SARS-CoV-2 (COVID-19) infection, and myocardial scarring or fibrosis that can contribute to re-entrant cardiac arrhythmias [10]. After SARS-CoV-2 (COVID-19) illness, autonomic dysfunction can result in inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome, that has been demonstrated as a resulting adrenergic modulation [11, 12]. Abstinence from aerobic activities or competitive sports for 3-6 months until resolution of myocardial inflammation by normalization of the troponin levels or cardiac MRI and serial echocardiogram, electrocardiogram, and cardiac MRI may be considered in competitive athletes with post-acute-COVID-19-related cardiovascular complications [13, 14] and in those with persistent cardiac symptoms [15, 16]. In a previously retrospective study among 3,080 COVID-19 patients revealed that withdrawal of cardiac-guidelines-directed medical treatment was related to higher mortality in the acute to post-acute-COVID-19 illness phases [17]. Potential harmfulness may be occur in the abrupt cessation of the use of RAAS inhibitors [18]. A low-dose beta blocker for decreasing adrenergic activity and heart rate management and anti-arrhythmic drugs (such as amiodarone) are recommended with attention in post-acute-COVID-19-illness patients with postural orthostatic tachycardia syndrome [19] and with pulmonary fibrotic changes following COVID-19 illness [20], respectively.

In conclusion, more follow-up is needed to determine risk-over-time resolution, particularly cardiovascular risk in patients with pre-existing conditions due to sustained- and increased-clinical-sequelae risk is frequently identified from 4 weeks to 4 months after the acute-COVID-19- illness phase.

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