Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the Coronavirus Disease 2019 (COVID-19) Pandemic

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Prevalence of CVD in Patients with COVID-19

The lack of widespread testing, national surveillance and standardized data collection, as well as the potential sampling bias in sicker, hospitalized patients with more comorbidities such as CVD has complicated efforts to accurately estimate the prevalence of CVD in patients with COVID-19. Moreover, there is marked variation in testing by country. A number of studies in the available literature suggest an association between preexisting CVD and severe COVID-19, which are summarized in Tables 1 and 2. A meta-analysis of six studies inclusive of 1,527 patients with COVID-19 examined the prevalence of CVD and reported the prevalence of hypertension, cardiac and cerebrovascular disease, and diabetes to be 17.1%, 16.4%, and 9.7%, respectively (4). Patients who required intensive care unit (ICU) admission were more likely to have these comorbidities compared to non-ICU patients. Increased case-fatality rates in the previously referenced analysis of 44,672 confirmed COVID-19 cases from Wuhan, China were noted in patients with CVD (10.5%), diabetes (7.3%), hypertension (6.0%), all notably higher than the overall case-fatality rate of 2.3% (15). Several smaller cohort studies have yielded similar results suggesting higher risk for adverse events in patients with CVD who contract COVID-19, although biases related to testing and standardized data apply here as well (1,19,25-28). Notably, while reports outside of China are limited, data from Italy suggest similar mortality rates and an elevated risk for death in patients with comorbidities (29). As emerging international data become available, analysis from multinational cohorts can help inform risk stratification for severe disease especially for patients with prior CVD.

COVID-19 Outcomes and CVD: Potential Mechanisms of Increased Risk

Mechanisms that lead to CVD are increasingly recognized to overlap with pathways that regulate immune function. For instance, age is the strongest risk factor for CVD and the effect of aging on immune function may be equally important for COVID-19 suceptibility and severity. Exemplary of this, the effect of age on the immune system is exemplified by low protective titers among 50% of adults older than 65 who receive the influenza vaccine (30,31). Other traditional CVD risk factors such as diabetes and hyperlipidemia impact immune function, and conversely, dysregulated immunologic status corresponds with elevated risk of incident CVD (32-35). Thus, prevalent CVD may be a marker of accelerated immunologic aging/dysregulation and relate indirectly to COVID-19 prognosis. An increased frequency of adverse CVD events post COVID-19 infection might also play a role in prognosis, similar to other viral infections such as influenza with mechanistic underpinnings which are complex, multi-factorial, and bi-directional (36,37). In addition, COVID-19 infection may trigger pathways unique to this pathogen which contribute to outcomes in CVD patients. For instance, higher expression of ACE2 in patients with hypertension and CVD has been postulated to enhance susceptibility to SARS-CoV2, although the data are conflicting and without clear suggestion for treatment (Figure 1) (5). Additional study is needed to understand the potential mechanistic relationships between CVD and COVID-19 outcomes.