Cardiovascular Morbidity Among Patients Infected With Coronavirus (COVID-19)

Dr. N John Camm, Klinikum Nuremberg Hospital, Germany

Introduction
Coronavirus COVID-19 was first reported in late December 2019 in Wuhan, China. COVID-19 is a betacoronavirus, like SARS and MERS, presenting as viral pneumonia with a wide range of acuity. As of February 12, there are 45,204 confirmed cases and 1,117 confirmed deaths across 28 countries; COVID-19 have greater infectivity and a lower fatality rate when compared to SARS and MERS. Majority of all cases are in mainland China, where despite aggressive containment efforts, case counts continue to rise rapidly.

Early Findings
Early case reports suggest patients with underlying conditions are at higher risk for complications or mortality from COVID-19; up to 50% of hospitalised patients have a chronic medical illness. 40% of hospitalised patients with confirmed COVID-19 patients have cardiovascular or cerebrovascular disease. In a recent case report on 138 hospitalised COVID-19 patients, 19.6% of patients developed acute respiratory distress syndrome:

I. 16.7% of patients developed arrhythmia; 7.2% developed acute cardiac injury
II. 8.7% of patients developed shock; 3.6% developed acute kidney injury
III. Rates of complication were universally higher for ICU patients

The first reported death was a 61-year-old male, with a long history of smoking, who succumbed to acute respiratory distress, heart failure, and cardiac arrest. Early, unpublished first-hand reports suggest at least some patients develop myocarditis.
Cardiovascular implications

In all influenza pandemics other than the 1918 flu, cardiovascular events surpassed all other causes of mortality, including superimposed pneumonia. Viral illness is a well-known destabilising factor in chronic cardiovascular disease, a general consequence of the imbalance between infection-induced increased metabolic demand and reduced cardiac reserve. The viral infection along with superimposed pneumonia will directly and indirectly affect the cardiovascular system:

I. Both coronary artery disease and heart failure patients are at increased risk of acute events or exacerbation; viral illness can potentially destabilise coronary plaques through several mechanisms including systemic inflammatory responses which have been recently documented with COVID-19.

II. Multiple co-morbidities (DM, obesity, HTN, COPD, CKD) further increase risk. Although published literature on CV implications of SARS/MERS is limited, in the absence of more detailed reporting on COVID-19, it may prove instructive:

I. 60% of MERS cases had one or more pre-existing comorbidity, resulting in a poorer prognosis; expert guidance suggests patients with diabetes, CVD, or renal disease should be prioritised for treatment.

II. Both SARS and MERS have been linked to acute myocarditis, acute myocardial infarction, and rapid-onset heart failure

III. Reversible, sub-clinical diastolic LV impairment in acute SARS even among those without underlying cardiac disease appears common, likely the result of systemic inflammatory immune response and is not unique to SARS; however, lower EF upon admission was predictive of later mechanical ventilations

IV. In one study of cardiovascular complications of SARS in 121 patients:

A. 71.9% of patients developed persistent tachycardia, including 40% with continued tachycardia during outpatient follow-up.

B. 50.4% of patients developed sustained asymptomatic hypotension during hospitalisation; one patient required inotropic support.

C. 14.9% of patients developed transient bradycardia.

D. 10.7% of patients developed transient cardiomegaly, without signs or symptoms of heart failure.

E. One patient experienced transient paroxysmal AF, with spontaneous resolution.
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F. Cardiovascular complications appeared statistically uncorrelated with oxygen desaturation or ICU admission.

Clinical guidance
1. COVID-19 is spread through droplets and can live for substantial periods outside the body; containment and prevention using standard public health and personal strategies for preventing the spread of communicable disease remains the priority.
2. In geographies with active COVID-19 transmission (mainly China), it is reason-able to advise patients with underlying cardiovascular disease of the potential increased risk and to encourage additional, reasonable precautions.
3. Older adults are less likely to present with fever, thus close assessment for other symptoms such as cough or shortness of breath is warranted.
4. Some experts have suggested that the rigorous use of guideline-directed, plaque stabilising agents could offer additional protection to CVD patients during a widespread outbreak (statins, beta blockers, ACE inhibitors, ASA); however, such therapies should be tailored to individual patients.
5. It is important for patients with CVD to remain current with vaccinations, including the pneumococcal vaccine given the increased risk of secondary bacterial infection; it would also be prudent to receive influenza vaccination to prevent another source of fever which could be initially confused with corona-virus infection.
6. It may be reasonable to triage COVID-19 patients according to the presence of underlying cardiovascular, respiratory, renal, and other chronic diseases for prioritised treatment.
7. Providers are cautioned that classic symptoms and presentation of AMI may be overshadowed in the context of coronavirus, resulting in underdiagnosis.
8. For CVD patients in geographies without widespread COVID-19 em-phases should remain on the threat from influenza, the importance of vaccination and frequent hand washing and continued adherence to all guideline-directed therapy for underlying chronic conditions.
9. COVID-19 is a fast-moving epidemic with an uncertain clinical profile; providers should be prepared for guidance to shift as more information becomes available.

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