

MANAGEMENT OF COVID-19 IN PERSPECTIVE OF CARDIOLOGISTS

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Contribution

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After influenza Coronavirus Disease 2019 (COVID-19) is pandemic. The outbreak of the disease started in China and the number of cases exceeded in the world as of March 15, 2020 and the rate is multiplying tremendously.¹ COVID-19 has its effect on cardiovascular system increasing morbidity with underlying cardiovascular condition and causing myocardial infarction and dysfunction.²

COVID-19 is caused by severe acute respiratory syndrome Coronavirus-2 (SARS-COV-2). It is believed that SARS-COV-2 entered in humans after it shifted from cats to an intermediate host (Malayan Pangolin).³

SARS-COV-2 spread by respiratory droplets and can be found in stool. The secondary infection rates range from 0.5-5%.^{4,5} The most common symptoms are fever (88%), dry cough (67.7%), rhinorrhea (4.8%) less frequent is gastrointestinal symptoms (diarrhea 4-14%, nausea/emesis 5%).⁵ Chinese reports presented with more significant symptoms in which 14% presented with dyspnea, respiratory rate \geq 30 bpm blood oxygen saturation \leq 93%, PO₂ to fraction of impaired oxygen ratio $<$ 300 and/or lung infiltrates $>$ 50% within 24 to 48 hours and 5% with respiratory failure, septic shock, and/or multiple organ dysfunction or failure.⁶

COVID-19 caused myocardial injury as presented in Wuhan China with elevated high sensitivity troponin I (hs-CTNI) or new ECG changes in 7.2% and 2.2% required ICU care.⁷ There is an elevation of other inflammatory biomarkers (D-dimer, ferritin, interleukin-6, lactate dehydrogenase) reflects cytokine storm or secondary hemophagocytic lymphohistiocytosis. The presentation of cardiac problem misleads viral myocarditis or stress cardiomyopathy. There is left ventricular dysfunction (EF 27%, LVEDD 5.8cm) and elevated cardiac biomarkers.⁸

The exact mechanism of cardiac involvement is not known. It is postulate that myocardial involvement may be via ACE2 or cytokine storm with T helper cells or hypoxia induced excessive intracellular calcium leading to cardiac myocyte apoptosis.⁹⁻¹¹

Preventive measure are best strategy for COVID-19. Vaccines and monoclonal antibodies against SARS-CoV-2 are in investigation stages. Recombinant human ACE2 (APN01) has been proposed as treatment to prevent SARS-COV-2.¹²⁻¹³ Remdesevir and Chloroquine are other drugs that may add clinical benefits in reduction of pneumonia and hospital stay.¹⁴⁻¹⁷

In mild to severe cases of COVID-19 typical imaging findings are not different (e.g. ground-glass opacification with or without consolidative abnormalities, consistent with viral pneumonia, minimal or no pleural effusions).¹⁸

CT chest was performed in Chinese cohort, though one should avoid its use unless necessary. If it is mandatory to use as a diagnostic tool it should be balanced with risk to other patients and health care workers during process of patient transport and time that is spent in diagnostic area. An alternative approach on bedside is use of lung ultrasound that presents with thickening of the pleural line and B line supporting alveolar consolidation. Pleural effusions are unusual.¹⁹

In a study of 1014 patients in Wuhan who underwent reverse transcription polymerase chain reaction (RT-PCR) testing and chest CT for evaluation of COVID-19 a posterior chest CT for COVID-19 had sensitivity of 97% and specificity of 25% using PCR test as a reference.²⁰ Chest CT abnormalities has also been identified in development of symptoms and even prior to detection of viral RNA from upper respiratory symptoms.²¹

In cardiac medicine the view points as a cardiologist for the diseases and management are,^{7,22}

1. A relatively high percent of patients admitted with COVID-19 will have underlying coronary artery disease (CAD). For most, symptoms of CAD will not be present on admission
2. It is likely that COVID-19 directly and indirectly affects the cardiovascular system, causing acute and chronic coronary syndrome.
3. In patients with known or suspected COVID-19, approach for the diagnosis and management of ST-elevation myocardial infarction (MI) similar to those without. On occasion, it is reasonable to liberalize the use of fibrinolytic therapy relative to primary percutaneous coronary intervention in low risk patients in centers where PPE and COVID cath labs are not available. In centers where proper PPE kits are available and can manage appropriately keeping in view the benefit and transferability to patients and health workers primary PCI can be undertaken.
4. Try to delay elective revascularization procedures in patients for whom the indication is relief of symptoms. Such an approach protects the patient and health care workers from potential exposure. For patients who must have revascularization for reasons such as extremely poor quality of life or prolongation of life, as with significant left main CAD, test the patient for COVID-19 infection.
5. None of the usual medications for the management of CAD, such as aspirin, beta blockers, statins, and nitrates, have been associated with worse outcomes in the setting of COVID-19 infection.
6. Continue angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers.
7. Underlying medical comorbidities appear to significantly impact COVID-19 severity and mortality. Patients with underlying cardiovascular disease and hypertension have been reported to have significantly high-case fatality rates compared with patients without these underlying comorbidities (10.5% and 6% mortality, respectively, compared with 0.9% mortality without underlying comorbidities).²³

COVID-19 with view as a cardiac anesthetist are;²⁴

1. Incidence of deep vein thrombosis and pulmonary embolism is due to immobility, systemic inflammation and disseminated intravascular coagulation.
2. During the preoperative evolution, focus should be on respiratory compromise assessment of organ failure particularly signs of shock, liver failure, renal failure and assessment of airway and to formulate airway plan. If the patient is considered high risk then it should be brought in notice of surgeons on urgency of operation and delay if possible.
3. During perioperative period, approach to infection control for all health care workers and patient in pre and postoperative area according to hospital guideline is mandatory. In addition, droplet and contact precautions should be taken care to patients who are highly suspected and confirmed case of COVID 19.
4. Hospital management plays pivotal role in multidisciplinary team between anesthesiologist, medical team, surgical

team, infection control and intensive care to implement isolation practices.

5. According to WHO recommendations N95 mask eye protective goggles, gown, gloves, and caps are necessary for all care provider. Operations on confirmed positive cases should be performed in an airborne infection isolation room (AIIR). Existing operating rooms should be converted to AIIR and maintained negative pressure. To prevent cross infection, single use of all anesthetic equipment, utensils, and drugs for individual patients must be drawn.
6. Anesthesia devices to maintain airway such as laryngoscope blades, circuits, filter, respiratory balloons, and suction tubes should be discarded after single use. At the end of surgery in operating room thorough disinfection and sterilization should be carried out that includes anesthesia machine, operating room spray and mopping, and ultraviolet radiation.
7. All health care providers involved in procedure may transmit infections to others so strict hospital policies should be addresses regarding dispose of potential, contaminated clothing.

In conclusion chest CT findings may be characteristics of COVID-19. No funding can completely rule in or rule out the possibility of COVID-19. CT chest though can be used for screening or diagnosis of COVID-19 and should be reserved for hospitalized patients when needed for management. All measures for care of health care worker and patients in view of cardiologist and anesthetist should be appropriately followed.

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