UMassMemorial Health Care

COVID-19 Clinical Care Council

CLINICAL PRACTICE GUIDELINE: CARDIOVASCULAR COMPLICATIONS, EVALUATION, AND MANAGEMENT
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I. Background and Purpose

SARS-CoV-2, which causes COVID-19 has been noted to impact multiple organ systems, particularly the respiratory system. While acute respiratory distress syndrome (ARDS) is a feared complication related to pulmonary involvement, preexisting cardiovascular disease (CVD) or direct cardiovascular (CV) manifestations of COVID-19 are responsible for significant morbidity and mortality.

This document aims to review some of the important CV considerations related to COVID-19 infection and to provide clinical practice guidelines based on best evidence or consensus. Since COVID-19 information is continuing to evolve, so too will this document.

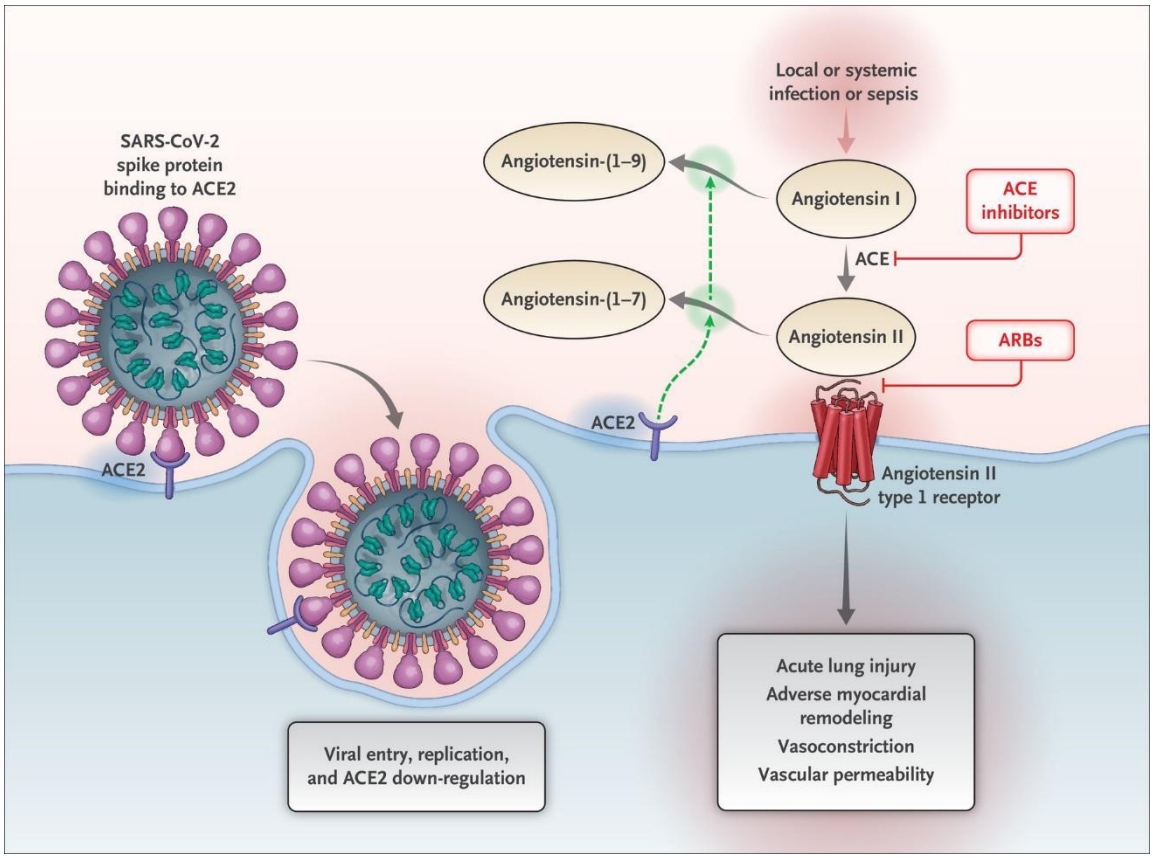
II. Pre-existing Cardiovascular Disease

Pre-existing CV disease such as hypertension (HTN), coronary artery disease (CAD), or cardiomyopathy (CM) is associated with those who have severe or critical illness with COVID-19 and is associated with significantly worse mortality in COVID-19. Those with pre-existing CVD and COVID-19 have approximately a 10% risk of mortality versus around 1% for those without comorbid conditions.

a. Renin-Angiotensin-Aldosterone System (RAAS) Inhibition in COVID-19:
Angiotensin converting enzyme 2 (ACE2) receptors have been shown to be the entry point into human cells for SARS-CoV-2 and ACE2 receptors are widely expressed in the heart and other areas not just in Type II alveolar cells. The American College of Cardiology (ACC), American Heart Association (AHA), and the Heart Failure Society of America (HFSA) released a statement on March 17, 2020 advising providers to continue medications that inhibit the RAAS if a patient is currently prescribed those medications such as angiotensin converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARB), angiotensin receptor blocker neprilysin inhibitor (ARNI) or mineralocorticoid antagonists (MRA). On an individualized basis, these medications can be initiated or stopped based on the clinical situation. The ACC/AHA/HFSA acknowledge the lack of evidence for which to offer concrete guidance. Clinical trials are ongoing to assess administration of recombinant human ACE2 and Losartan (ARB) in COVID-19.

Key points:

- *Continue RAAS inhibition where possible*
- *Initiate RAAS inhibition when appropriate for the individualized clinical situation (e.g heart failure with reduced ejection fraction with hemodynamic stability)*



b. Case Fatality Rate (CFR): Elderly people with comorbidities like CVD are more likely to be infected with SARS-CoV-2 and more likely to have severe disease. From the Chines meta-analysis of 44,672 patients, CFR was higher in patients with pre-existing CVD and risk factors, compared to the overall CFR of 2.3%.

- CVD – 10.5%
- Diabetes – 7.3%
- Hypertension – 6.0%

III. Cardiovascular Complications

Approximately 7.2% of patients in 138 hospitalized patients in a cohort from Wuhan, China had elevated troponins, new electrocardiographic (ECG) abnormalities, or new echocardiographic abnormalities. 22% of patients who were critically ill had one or more of these three cardiac findings. 11.8% of people

without CVD had elevated troponin or cardiac arrest during the hospitalization. Myocardial injury, as indicated by elevated troponins, is common in COVID-19 infection.

Potential Cardiovascular Complications related to SARS-CoV-2:

1. Acute myocardial injury
2. Acute coronary syndrome
3. Acute or Fulminant Myocarditis
4. Arrhythmias

1. Acute Myocardial Injury: Acute myocardial injury related to COVID-19 infection can occur by multiple mechanisms including critical illness/sepsis, acute myocarditis, exuberant inflammatory response, or acute coronary syndrome. Two more unusual mechanisms by which SARS-CoV-2 is hypothesized to directly cause acute myocardial injury include cytokine release/inflammation and acute myocarditis.
Definition: Troponin-I >99th percentile (see below), abnormal ECG or echo findings.

UMMS, Memorial, and Marlborough 99th percentile: 0.04 ng/mL

HealthAlliance and Clinton 99th percentile: 0.1 ng/mL

Incidence: Troponin elevation effects over 7% of COVID-19 patients who are admitted. Among the critically ill, the incidence of troponin elevation is >20%.
Natural History: Troponin elevation may be a late manifestation of COVID-19, with a steady rise from 4-22 days seen in non-survivors. Troponins may not be elevated on presentation and may rise later in the hospital course. In non-published reports from the United States, troponin elevation can be observed in the critically ill COVID-19 patient even as ARDS improves.
Prognostic implications: Troponin elevation is common in patients requiring ICU admission and is associated with increased mortality.
Diagnosis/Testing:

Troponin: Check troponin on admission and trend to peak to detect myocardial injury. Consider repeat ad hoc testing with new concerning symptoms for ischemia (chest pain, chest pressure, dyspnea) of heart failure (HF). Consider intermittent testing of troponin for critically ill patients particularly if their clinical status changes. The detection of elevated troponins may prompt further cardiac evaluation to detect myocardial dysfunction and elevation in troponins can assist in prognostication.

- If Troponin-I is >0.2 ng/mL: Obtain a 12-lead ECG, start telemetry, obtain point-of-care ultrasound (POCUS) if cohorted providers are trained in echocardiography.
- If there is an abnormality on POCUS or ECG, consider a formal echo (see cardiovascular imaging section). If there are no ECG, telemetry, or echo abnormality, continue daily monitoring.

High inflammatory markers: Significant elevations in CRP, ESR, IL-6, and Ferritin may occur when cytokine release/inflammation is the pathophysiologic mechanism of myocardial injury.
Management: Management will be dependent on the cause of the troponin elevation (see below).

Key points

- *Myocardial injury as indicated by elevated troponin is common in COVID-19, especially in critically ill patients.*
- *Determining the underlying mechanism of myocardial injury can be challenging, and the mechanisms include ACS, Inflammatory/cytokine release, or acute myocarditis.*
- *Troponins should be checked on admission and should be checked serially to detect myocardial injury.*

2. Acute coronary syndrome (ACS)

Incidence: Since increased risk of ACS is seen in patients with influenza (6-fold increase within 7-days of influenza diagnosis), there may be an increased risk of ACS with COVID-19. Elevations in troponin and ECG abnormalities are common, but existing cohorts suffer from selection bias as ischemic evaluation by cardiac catheterization has not been routinely performed. The true incidence of ACS versus other mechanisms of myocardial injury is not known.

Diagnosis/Testing: Elevated troponin-I or ECG changes alone will not allow for discrimination between ACS, sepsis, or acute myocarditis. Higher consideration for ACS should be based on all the clinical evidence available:

Symptoms: New chest pain, anginal equivalent, or dyspnea.

Uncharacteristic symptoms: Pleuritic pain, middle or lower abdominal pain, pain localized to one spot on the chest, pain reproduced with movement or palpation, pain lasting seconds or less, pain radiating to lower extremities.

ECG: Dynamic ECG changes (new T-wave inversions, ST depression or elevation).

Troponin-I: Rise in troponin over the 99th percentile and subsequent fall.

Echo: New regional wall motion abnormality in a typical coronary distribution.

Management: Medical management is preferred in the absence of ST segment elevation due to the potential spread of SARS-CoV-2 in the catheterization lab and uncertain benefit of percutaneous intervention in the setting of COVID-19. Cardiology consultation is recommended to clarify appropriateness of an invasive strategy.

STEMI: Initiate response team activation. Ensure IV access. Administer aspirin 325 mg. Repeat 12-lead ECG within 5 minutes. The STEMI team will follow previously defined protocols for personal protective equipment (PPE).

NSTEMI: Discuss with the cardiology consult team to help determine if a Type 1 myocardial infarction is suspected. If Type 1 MI is suspected:

- Full-dose aspirin initially followed by Aspirin 81 mg PO once daily, heparin gtt with ACS target PTT, oxygen (if hypoxemic), statin (may be beneficial with anti-inflammatory effects)
- Nitrates (if hypertensive or with persistent chest pain) and opiates (if persistent pain despite nitrates)

- Beta blockers within the first 24 hours in the absence of new heart failure, a low-output state, or cardiogenic shock.

Key points

- *NSTEMI's are preferably treated with medical therapy.*
- *STEMI's should result in the usual STEMI activation protocols.*

3. Acute or Fulminant Myocarditis

Definition: Myocarditis is an inflammatory disease of the myocardium. It has traditionally been defined by histologic, immunologic, and immunohistochemical criteria. SARS-CoV-2 appears to cause acute (new onset) or fulminant (rapid and aggressive) myocarditis.

Incidence: The true incidence of acute or fulminant myocarditis is uncertain in COVID-19. Myocardial injury is common as manifested by elevation in troponin values >99th the upper range of normal. However, this myocardial injury may be a result of sepsis, ACS, inflammation, or may indicate direct myocardial injury related to SARS-CoV-2. A definitive diagnosis of myocarditis requires an endomyocardial biopsy. Most cases or myocarditis in the literature are suspected based on clinical features and other diagnostic imaging.

Natural History: There are rare reports of cardiac dominant presentations with less evidence of acute lung injury and more prominent cardiogenic shock related to acute/fulminant myocarditis. In some instances, myocarditis appears late in the hospital course.

Pathophysiology: The pathophysiology of myocarditis is uncertain but is postulated to be related to direct viral injury due to the presence of abundant ACE2 receptors in the heart.

Diagnosis/Testing: A definitive diagnosis of myocarditis requires endomyocardial biopsy but this is no longer routinely performed or necessary. Therefore, most diagnoses are presumptive based on the totality of clinical evidence. This can include:

- Elevation in Troponin indicative of myocardial injury
- Elevation in BNP or NT-proBNP levels
- High inflammatory markers such as CRP, ESR, IL-6, and Ferritin may occur when cytokine release/inflammation is the mechanism of injury but will often be elevated in myocarditis also.
- LV and/or RV systolic dysfunction on cardiac imaging such as echocardiography with regional wall motion abnormalities in a non-coronary distribution or global dysfunction.
- Cardiogenic Shock or signs and symptoms of new onset heart failure.
- Cardiac magnetic resonance imaging (cMRI) consistent with myocarditis (Abnormal late gadolinium enhancement and Abnormal T2 signal indicating edema). Radiology and Cardiology consultation is required if considering cMRI.
- Endomyocardial Biopsy consistent with lymphocytic myocarditis. Requires discussion with heart failure consult team.

Management

- Cardiology consultation is indicated if a diagnosis of myocarditis is suspected or confirmed.
- Enrollment in a clinical trial for novel therapies should be strongly considered in the setting of myocarditis due to poor survival. Please note that efficacy of novel therapies in acute myocarditis with SARS-CoV-2 is unproven. Administering dual therapy with an anti-viral and anti-inflammatory agent (if inflammatory markers are elevated) are strategies other institutions have reported in discussion with the United States consortium experiences and in case reports from China.
- Treatment of Cardiogenic Shock is per usual care, and as outlined in clinical practice guidelines available elsewhere, for non-acute MI cardiogenic shock.
- If cardiogenic shock requires mechanical circulatory support (MCS), the decision must be made in conjunction with the Cardiology team, Heart Failure team, CT surgery, and/or Intensivists. There are limited data on the efficacy of percutaneous options (Impella RP/CP, IABP) and surgical options (Impella 5.0, ECMO, CentriMag). Data from China suggest poor outcomes with predominantly veno-venous ECMO although there are criticisms that the centers deploying ECMO were inexperienced. Anecdotal experience from Columbia hospital indicates better survival than observed in China. Utilization of Impella Connect for Impella devices is recommended to minimize risk of exposure. Cardiology will contact the nurse manager and resource nurse in the Coronary Care Unit to establish appropriate bedding of the patient with MCS.
- For hemodynamically stable, acute decompensated systolic HF without cardiogenic shock, consideration of goal directed medical therapy must be individualized for the patient. However, standard heart failure therapies are recommended.

Key points

- *The diagnosis of myocarditis as the cause of myocardial injury in COVID-19 can be challenging due to risk of exposure during invasive procedures/imaging.*
- *Treatment involves usual standard of care for HF and/or cardiogenic shock.*
- *Novel pharmacologic therapy involving an anti-viral agent and anti-inflammatory agents should be considered under a clinical trial.*

4. Arrhythmias

Incidence: Unspecified arrhythmias in 17% of hospitalized patients with COVD-19 (n = 23 of 138) with higher rates in ICU patients (44%) versus non-ICU (7%). Ventricular tachycardia (VT) and ventricular fibrillation (VF) may be seen as late manifestations in anecdotal reports.

Diagnosis/Testing:

- Telemetry, 12-lead ECG, electrolytes, troponin-I, BNP or NT-proBNP, thyroid studies
- POCUS to assess LV and RV function.
- Formal echo if arrhythmia confirmed on 12-lead ECG or telemetry
- Cardiology consultation for refractory ventricular arrhythmias or difficult to control supraventricular arrhythmias particularly with associated hemodynamic compromise.

Management:

1. **Atrial fibrillation/atrial flutter**
   Management Options:
   Beta blocker: in the absence of HF or shock
   Non-dihydropyridine calcium channel blockers (diltiazem, verapamil): in the absence of HF or shock
   Digoxin: consider as an add on agent to beta blocker or calcium channel blocker
   Amiodarone: in the absence of hypotension or shock
   DC cardioversion: appropriate when the patient is hemodynamically unstable. Deliver biphasic synchronized 200 Joules shock.
2. **Ventricular tachycardia (VT):** Sustained (≥30 seconds) VT has been reported in cases of acute/fulminant myocarditis in COVID-19. VT can also be seen in patients with underlying structural cardiovascular disease.
   Unstable (SBP < 90 mm Hg)/pulseless: ACLS Resucitation
   Stable: Cardiology consultation and Procainamide (start with 100 mg IV over 2 minutes) or Amiodarone (start with 150 mg IV over 10 min).
3. **Polymorphic VT:** In the setting of QT prolongation, this is known as torsades de pointes (TdP). This is often non-sustained and self-terminating but can recur if correction or removal of precipitating factor not addressed. Anti-arrhythmic medications, antibiotics (including azithromycin), anti-malarials (chloroquine, hydroxychloroquine), narcotics, electrolyte abnormalities, congenital defects, and other medication classes can cause TdP. Polymorphic VT is usually poorly tolerated and can degenerate into ventricular fibrillation.
   Management: Magnesium sulfate IV 2 g over 2 minutes, immediate electrical cardioversion, increase pacing rate if pacemaker present, discontinue precipitating drugs, and consult cardiology.

Key points

- *Arrhythmias are common in COVID-19 and treatment is usual standard of care.*
- *Ventricular arrhythmias could be a result of myocarditis and should prompt further investigation.*

IV. General Laboratory Testing

Recommended laboratory testing for the evaluation of cardiac disease involves detecting myocardial injury, high filling pressures, and inflammation.

- Troponin-I trended until peak.
- If the admission troponin-I value is normal, consider daily assessment for up to 3 days to detect onset of myocardial injury as this may prompt imaging assessment for the development of indolent myocarditis. Troponin elevation is associated with poor outcomes and the need for ICU admission.
- If the patient is in the ICU, consider intermittent assessment of troponin particularly if the clinical status changes to detect new myocardial injury that may be secondary to myocarditis or cytokine release syndrome. Myocarditis may occur late in the hospitalization even as respiratory symptoms improve.
- BNP or NT-proBNP (NT-pro BNP must be ordered STAT for a 4 hr Quest turnaround) in those who are short of breath on admission may indicate poor prognosis and high intracardiac filling pressures suggesting underlying heart failure from myocarditis or other myocardial injury.
- Inflammatory markers such as CRP, ESR, IL-6 levels, and ferritin may be useful for identifying a subset of critically ill COVID-19 patients that may have cytokine release syndrome and a massive SIRS response. These patients may have myocardial dysfunction as a result. Clinical trials for IL-6 inhibitors are enrolling to assess safety and efficacy in this scenario. Tosilizumab is available at UMass, but at the present time, UMass is not currently participating in a clinical trial for Tosilizumab.

Key points

- *Check Troponin-I, BNP or NT-proBNP on all patients with COVID-19 at admission.*
- *Consider serial testing as warranted by clinical judgement.*

V. Non-invasive Cardiovascular Imaging

Cardiovascular imaging such as nuclear stress testing, transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), cardiac computed tomography (Cardiac CT) and cardiac magnetic resonance imaging (cMRI) may be relevant diagnostic tests that can greatly enhance the care of patients with COVID-19. However, performing these tests can potentially increase the spread of SARS-CoV-2 through contaminating equipment, direct patient contact with more providers and staff, and will involve the consumption of more personal protective equipment (PPE) that is in short supply.

a. Electrocardiogram: A 12-lead ECG and/or rhythm strip is recommended on admission and should be repeated as determined by the clinical team if abnormal. QTc can also be determined on telemetry strips.

b. Echocardiography:

For COVID-19 Suspected or Patients Under Investigation (PUI):

1. Patients with known heart disease may require comprehensive TTE.

2. If the patient is hemodynamically stable and/or without ischemic symptoms, it is preferable to defer the echo until COVID-19 is ruled out.

3. If hemodynamically unstable, dynamic ECG changes, arrhythmias, or significantly elevated/rising troponin, but the COVID-19 PCR assay has not resulted, POCUS by a capable provider or limited echo by a sonographer should be considered.

For COVID-19 Confirmed Patients:

1. There is no established role for routine echocardiography.

2. Recommend POCUS by a capable provider and/or cardiology consult if any of the following are present:

    1. Shock
    2. Significant elevation or rising troponin-I
    3. Decline in MVO2 or central venous O2
    4. New onset CM or clinical heart failure
    5. New arrhythmia
    6. Significant ECG changes

3. Limited echo by the Echo lab sonographer if point-of-care ultrasound is abnormal or if recommended in cardiology consultation.

c. Other Cardiac Imaging:

- Additional cardiac imaging must be considered on a case-by-case discussion with cardiology consultation and/or radiology.

Key points

- *POCUS is the preferred screening of cardiac structure and function if an Echocardiogram is desired prior to a formal echocardiogram.*

VI. Invasive Cardiac Evaluation

Invasive cardiac evaluations should be reserved for situations where it will greatly impact the care of the patient due to risks of spreading SARS-CoV-2 and consumption of PPE. The risks and benefits must be weighed in each situation. Cardiology will determine the appropriateness of performing invasive evaluations such as cardiac catheterization, endomyocardial biopsy, or TEE.

VII. Guidelines for Cardiology Consultation

The following are indications for cardiology consultation:

- Rising Troponin levels.
- ACS is suspected.
- New HF or concern for cardiogenic shock.
- Concern for acute myocarditis.
- VT, Ventricular fibrillation, or polymorphic VT.
- Supraventricular tachycardias that are difficult to control or associated with hemodynamic compromise.
- New ventricular dysfunction.
- Congenital long QT syndrome with plans to initiate Hydroxychloroquine.

VIII. Cardiovascular Complications of COVID-19 Pharmacologic Therapy

1. **Hydroxychloroquine** (HCQ): known to block Kv11.1 (HERG) and can cause drug-induced long QT. Syncope and torsade de pointes is generally limited to chronic use, use of multiple QT prolonging medications (e.g. azithromycin), metabolic derangements, or renal failure. Widely tolerated, especially during relatively short courses for COVID-19. Specific precautions should be considered for select patients:
    - Congenital Long QT Syndrome*
    - Severe renal insufficiency
    - Concomitant QT prolonging drugs (including azithromycin) with QTc or QTc corrected ≥ 500 msec
    - Electrolyte imbalance (hypocalcemia, hypokalemia, hypomagnesemia) need to be corrected prior to use and monitored regularly.
    - Class III antiarrhythmic medications (amiodarone, sotalol)*

    * Consider cardiology consultation

2. **Remdesivir:** At this time, safety and efficacy have not been established in adult patients. No specific drug-drug interaction data with cardiac drugs are available.

3. **Tocilizumab:** increased serum cholesterol (20%), hypertension (1-6%).

4. **Azithromycin:** There is an FDA communication (from 2013) that azithromycin increases risk of potentially fatal heart rhythms. Azithromycin can increase the QT interval and increases the risk of TdP, and these complications are more commonly seen with co-administered medications that prolong QT.

When co-administered with chloroquine or HCQ, QT increases in a dose-dependent fashion. Other risk factors for QT prolongation include bradycardia, age ≥ 68 years, female sex, loop diuretic use, sepsis, heart failure, hypokalemia and hypomagnesemia, and certain anti-arrhythmic medications.

5. **Monitoring QTc on HCQ ± Azithromycin**
    - In patients with wide QRS due to bundle branch blocks or pacing, QTc should be corrected: QTc= QTc – (QRS – 100) msec
    - If baseline QTc or corrected QTc ≥ 500 msec, assess on-therapy QTc within 4-hours of the first dose of HCQ and again at 48-hours. Consider HCQ alone rather than in combination with azithromycin
    - If the change in QTc ≥ 60 msec or on-therapy QTc (or corrected QTc) is ≥ 500 msec, consider pausing HCQ therapy, discontinue QT prolonging medications including azithromycin, and repeat electrolytes

Key points

- *QTc prolongation is a potential complication of hydroxychloroquine and azithromycin*
- *Novel therapies may have other unknown CV adverse side effects*

IX. Patients Post-Cardiac Transplant or with a Ventricular Assist Device

- The HF team should be called if a cardiac transplant patient or a patient with a ventricular assist device tests positive for COVID-19.

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