COVID-19 & Cardiac manifestations in adults

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Adverse cardiovascular outcomes in this setting = has not been determined.

COVID-19 & cardiovascular disease:

Impact of pre-existing cardiovascular disease:

- hemodynamic demands in the setting of chronic (preexisting) heart disease, HTN & COVID , IHD & COVID , DCM & COVID
- by an acute exacerbation of chronic disease.

angiotensin-converting enzyme 2 receptor-related signaling pathways have a role in COVID-19-related cardiac injury?

Impact of COVID-19 treatment on cardiovascular disease

commonly manifestations of heart disease:

myocardial injury

- hypoxic injury
- stress (takotsubo) cardiomyopathy
- ischemic injury caused by cardiac microvascular dysfunction,
- small vessel cardiac vasculitis
- endotheliitis
- epicardial coronary artery disease (with plaque rupture or demand ischemia);
- right heart strain (acute cor pulmonale, PTE, ARDS & pneumonia)
- systemic inflammatory response syndrome (cytokine storm)

The causes (mechanisms) of this association =?

- Impaired physiologic reserve (cardiovascular and pulmonary),
- Impaired immune response,
- Augmented inflammatory response,
- Vulnerability to SARS-CoV-2-induced endothelial dysfunction,
- Effects mediated by the angiotensin-converting enzyme 2 receptor

SPECTRUM OF CLINICAL PRESENTATIONS

- No clinical evidence of heart disease,

- Cardiac test abnormalities
- cardiac troponin elevation
- abnormalities on cardiac imaging
- asymptomatic cardiac arrhythmias

- Symptomatic heart disease.

No clinical evidence of heart disease,

- Most patients with COVID-19 with cardiac abnormalitie➔ only have typical symptoms of COVID-19, including cough, fever, myalgia, headache, and dyspnea.

- A minority of patients with COVID-19 present with symptoms that may suggest heart disease (such as palpitations or chest pain)

Asymptomatic heart disease

Most patients with cardiac test abnormalities =lack symptoms of heart disease.

- **Myocardial injury** — troponin elevation (cardiomyocyte death.)
- the causes =? not been fully elucidated / among the causes of injury ?
- myocarditis,
- stress cardiomyopathy,
- myocardial infarction (MI).

CARDIAC TEST FINDINGS, Biomarkers

- **troponin** = 7 to 36 % , **hs-cTnI**= 6.5 %

- **BNP / NT-proBNP** = %12.9

- increased risk of mortality: (hs-cTnI and BNP was an independent predictor of mortality)
- Overall mortality rate = 23.2 percent.

- **Procalcitonin**

- **D-dimer**

- **interleukin 6,**

- **ferritin,**

- **lactate dehydrogenase**

Troponin — myocardial injury ➔ in hospitalized patients + more severe disease + worse outcomes

- ✓ Limited data = troponin elevations in asymptomatic or only mildly symptomatic
- ✓ Troponin elevation = more prevalent = mortality rate during hospitalization was 18.5 percent.
- ✓ even mildly elevated hs-cTnI (0.03 to 0.09 ng/mL) = risk of death (adjusted hazard ratio [HR] 1.75), while greater elevations = adjusted HR 3.03.

A variety of time courses for troponin elevation have been observed:

- **Mild** – (typically <99th percentile URL), = no cardiac symptoms.
- **Moderate time-limited** – (exceed the 99th percentile URL) = clinically suspected myocarditis or stress cardiomyopathy
- **Progressive** –suffer clinical deterioration with respiratory failure

IF progression to cytokine storm ➔ nonsurvivors, with death occurring at a median of 18.5 days after symptom onset

- *Thus, it is not yet possible to determine whether myocardial injury is an independent risk marker in COVID-19 or if the risk associated with it is related to the burden of preexisting cardiovascular disease.*

Cardiac troponin elevation

Myocardial ischemia

coronary ischemia

Non-coronary ischemia

Myocardial injury with no ischemia

Comorbidities

Specific identifiable precipitants

Myocardial ischemia
Acute coronary syndrome
STEMI
NSTEMI
Other coronary ischemia
Arrhythmia: tachy- or brady-
Cocaine/methamphetamine use
Coronary intervention (PCI or cardiothoracic surgery)
Coronary artery spasm (variant angina)
Stable coronary atherosclerotic disease in setting of increased O_2 demand (eg, tachycardia)
Severe hypertension
Coronary embolus
Aortic dissection
Coronary artery vasculitis (SLE, Kawasaki)
Noncoronary ischemia
Shock (hypotension)
Hypoxia
Hypoperfusion
Pulmonary embolism
Global ischemia
Cardiothoracic surgery
Myocardial injury with no ischemia
Comorbidities
Renal failure
Sepsis
Infiltrative diseases
Acute respiratory failure
Stroke
Subarachnoid hemorrhage
Specific identifiable precipitants
Extreme exertion
Cardiac contusion
Burns >30% BSA
Cardiotoxic meds: anthracyclines, herceptin
Electrical shock
Carbon monoxide exposure
Other
Stress (takotsubo) cardiomyopathy
Myocarditis
Myopericarditis
Rhabdomyolysis involving cardiac muscle
Hypertrophic cardiomyopathy
Peripartum cardiomyopathy
Heart failure, malignancy, stress cardiomyopathy

Cardiac complications

- myocardial injury:
- heart failure (HF):
- cardiogenic shock:
- cardiac arrhythmias: (including sudden cardiac arrest.)

Heart failure

- **Myocarditis**

caused by SARS-CoV-2 has not been definitively confirmed

- **Stress cardiomyopathy** — Stress (takotsubo) cardiomyopathy + marked recovery of left ventricular (LV) systolic function within days
- **hypertensive crisis.**
- **Myocardial infarction** —
- **Heart failure**

➢ Pre-existing known or undiagnosed heart disease

➢ acute hemodynamic stress (eg, acute cor-pulmonale),

➢ acute myocardial injury (eg, acute MI, stress cardiomyopathy, cytokine storm, and other possible etiologies described above)

Limited data on the incidence of HF in COVID-19.

2 study in in Wuhan =

- 1st ➔49 percent of patients who died and in 3 percent of patients who recovered, (less than 1 percent baseline prevalence of chronic HF)
- 2end ➔ in 52 percent of patients who died and in 12 percent of patients who recovered
- **Right heart failure** — Acute cor pulmonale : PTE / ARDS

- Venous thromboembolism

- **Cardiogenic shock** — Case reports ➔ inotrope and VA-ECMO ➔ Rapid recovery?= possible stress cardiomyopathy

- Endomyocardial biopsy =did not show findings of myocarditis

- **Multisystem inflammatory syndrome in adults (MIS-A)**

➢ In children (MIS-C) ➔ as a Kawasaki-like illness associated with fever, gastrointestinal symptoms, shock, LV systolic dysfunction, and elevated inflammatory markers.

➢ In adults (MIS-A) ➔ presenting with fever, gastrointestinal symptoms, and shock with vasoplegia, LV systolic dysfunction, and elevated inflammatory ➔ highly responsive to parenteral steroids.

Cardiac arrhythmias

- sinus tachycardia.
- AF (5-6 %), atrial flutter.
- PAC = in 7.7 percent , PVC = 3.4 percent.
- monomorphic or polymorphic VT.
- Bradyarrhythmias, including sinus pauses or high-grade heart block with slow escape rhythms.
- RBBB = 7.8 percent, , LBBB = 1.5 percent, nonspecific IVCD = 2.5 percent.
- LQTS ➔ medication or other clinical scenario (ie, hypokalemia or hypomagnesemia) .
- Repolarization abnormalities (ST elevation= 0.7 percent, localized T-wave inversion = 10.5 percent, nonspecific repolarization = 29 percent.)

- **Patients receiving QT-prolonging treatments** — hydroxychloroquine or chloroquine

Monitoring for QT prolongation —

- In general, patients with the following QTc intervals are at low risk for significant QT prolongation and polymorphic VT:

- QTc <460 milliseconds in prepubertal males/females

- QTc <470 milliseconds in postpubertal males

- QTc <480 milliseconds in postpubertal females

- **Arrhythmia-related procedures**
- **Perioperative cardiac implantable electrical device management**
- **Cardiac implantable electrical device interrogations**
- **Patients requiring cardiopulmonary resuscitation (CPR)**

Other cardiac test abnormalities

ECG — various ECG findings

Cardiac imaging

- **Echocardiogram** —
  - RV: dilation and dysfunction (39 percent), Femoral DVT= 5 of 12 patients with RV failure.
  - LV: diastolic dysfunction (16 percent), and LV systolic dysfunction (10 percent).

- **Cardiovascular MRI :**
  - elevated myocardial native T1 (73 percent),
  - elevated myocardial native T2 (60 percent),
  - myocardial late gadolinium enhancement (LGE; 32 percent),
  - pericardial LGE (22 percent).

- **Endomyocardial biopsy**

lymphocytic infiltration; necrosis was not described and no viral genome was detected.

diagnoses

- acute MI = 3 %
- Myocarditis = 3 %
- stress cardiomyopathy = 3 %
- Severe ventricular (left, right, or biventricular) dysfunction =14 %.
- Cardiac tamponade = 1 %.

Myocardial histology and viral genome analysis

confirmed myocarditis BUT viral myocarditis

caused by SARS-CoV-2 has not been

definitively confirmed by histologic and viral

genome analysis.

COVID-19: Evaluation and management of cardiac disease in adults

Indications

- new-onset HF (including left HF and acute cor pulmonale),
- unexplained cardiac arrhythmias,
- ECG changes (particularly ST elevation).

The approach to cardiac evaluation in COVID-19 ➔ may differ from the standard approach to evaluation ➔IF change management and guide prognosis. such as acute MI.

General approach

- **Supportive cardiac care**
- Patients with COVID-19 and HF or asymptomatic HF➔ should receive standard therapy for these conditions including pharmacologic therapy, careful management of fluid balance, and advanced therapies as needed.
- **ACE inhibitors and ARBs**:

because ACE2 is a receptor for this virus➔ there is no evidence that treatment with these drugs worsens the clinical course of SARS-CoV-2 infection.

- **thromboprophylaxis**

Hypercoagulability and the role of thromboprophylaxis in patients with COVID-19 are discussed separately.

Investigational agents

- **cytokine release syndrome :**

No treatment has been identified for this syndrome.

interleukin-6 inhibitors tocilizumab and sarilumab are ongoing.

- **Mechanical circulatory support** :

ISHLT➔ guidance, VAD limited to INTERMACS status 1 to 3 patients.

ECMO: in patients with COVID-19.

- **Cardiac transplantation** :

waiting at least 14 days after initial COVID-19 diagnosis
+ **and** two negative PCR-based tests at least 48 hours apart.

Management of cardiac transplantation recipients + COVID-19

- **Mild COVID-19:** (no shortness of breath or hypoxia)

➔ at home for two weeks + continuation of baseline maintenance immunosuppression with frequent follow-up to monitor for worsening symptoms.

- **moderate disease** (shortness of breath or hypoxia requiring supplemental oxygen via nasal cannula) or **severe disease** (respiratory failure requiring intensive care unit admission and/or ventilator support, acute respiratory distress syndrome, circulatory collapse, acute kidney failure, cardiomyopathy, and/or clinical syndrome compatible with cytokine storm)

➔ in-hospital supportive care.

➔Many heart transplantation centers reduce (or hold) antiproliferative agents (eg, mycophenolate mofetil or azathioprine) in heart transplantation recipients hospitalized with COVID-19 with close monitoring for rejection

generally, calcineurin inhibitors and prednisone doses have been maintained in this setting.