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continued use of colchicine. Genetic studies revealed a likely pathogenic variant in the MYBPC3 gene involving an altered consensus splice site associated with autosomal dominant hypertrophic cardiomyopathy and dilated cardiomyopathy. Symptoms recurred upon tapering of prednisone, requiring a longer course of treatment. The patient also developed symptoms of postural orthostatic tachycardia syndrome. With continued treatment, symptoms significantly improved. Conclusion: Our case suggests that lymphocytic myocarditis could be a rare manifestation of coronavirus disease whose risk factors may include hereditary cardiomyopathies. It has potential to become a chronic condition with a challenging symptomatic management.

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Cardiac And Inflammatory Biomarkers And New Or Worsening Heart Failure In High-risk Patients With Covid-19

PARK, JASON; KAPLAN, MICHAEL; RODRIGUEZ, JESSE; PANE, STUART; BRAD SHAW, JAMES; KHADKA3, MARIA P. PICON4, MARK STUDENY1, CARLOS RUEDA5; 1MARSHALL UNIVERSITY, HUNTINGTON, WV;2DANBURY HOSPITAL, DANBURY, CT;3UNIVERSITY OF UTAH SCHOOL OF MEDICINE, SALT LAKE CITY, UT;4THE UNIVERSITY OF UTAH, SALT LAKE CITY, UT;5NORA ECCLES HARRISON CARDIOVASCULAR RESEARCH & TRAINING INSTITUTE, SALT LAKE CITY, UT

Introduction: Consequences of COVID-19 on the cardiovascular (CV) system are broad and have encompassed conditions including cardiomyopathies and acute coronary syndrome. Inciting triggers are likely multifactorial and associated with mechanisms ranging from a pro-inflammatory cytokine storm to microvascular ischemia. Elevated inflammatory markers including high sensitivity C-reactive protein (hsCRP), d-dimer, and interleukin-6 (IL-6) have been associated with adverse outcomes in COVID-19. We sought to identify the association of cardiac and inflammatory biomarkers with new or worsening HF in patients admitted with COVID-19. Methods: We performed a single-centered prospective cohort study of patients who were hospitalized with COVID-19 from September 2020 through April 2021. Blood serum specimens from enrolled patients were collected upon hospital admission, weekly and upon discharge for quantification of troponin T, B-natriuretic peptide (BNP), and inflammatory markers, including immune cell subtypes, cell activation markers, and plasma cytokines. New or worsening HF was defined as any new left ventricular (LV) systolic dysfunction, decrease in ejection fraction >5% or a decompensated state in those with a prior history of HF. Results: Twelve of the 41 patients who were enrolled presented with new or worsening HF. Of this subgroup, 6 had new or worsening LV systolic function, 7 had concomitant myocardial injury represented by elevated troponin and there were no biopsy-confirmed cases of myocarditis. The average age of this subgroup was 65.8 years (interquartile range [IQR] 55.6-76.0) and 67% were male. All patients had at least 3 CV risk factors upon presentation and 75% had a prior diagnosis of HF (p < 0.01). Clinical profiles were otherwise similar between groups, and there were no significant differences in acute medical intervention (AMI). However, the patients with HF ranged from 50% (suffered acute HF, median BNP was elevated (345, IQR 184-509 vs 95, IQR 38.5-171 pg/mL, p = 0.01), as was median troponin (201, IQR 30-620 vs 16, IQR 0-86.5 ng/L, p = 0.02). Otherwise, there were no significant differences noted in baseline or follow-up biochemic markers of inflammation, including hsCRP, IL-6 and d-dimer, between patients with and without new or worsening HF. Conclusion: Patients admitted with COVID-19 who experience new or worsening HF syndrome have elevated BNP and troponin levels, but similar CV risk factors compared to those who do not. Baseline measures of inflammation are not greater in those who develop HF syndrome in the setting of COVID-19.

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HeartLogic Heart Failure Index As Covid Index: Predicting Severity Of Covid-19 In Patients With Advanced Heart Failure

UTSAV R. PANTA1, KAMANA ACHARAYA2, SHARON D’ANGELO2, SAROJ KHADKA1, MARIA P. PICON2, MARK STUDEY1, CARLOS RUEDA3; 1MARSHALL UNIVERSITY, HUNTINGTON, WV; 2DANBURY HOSPITAL, DANBURY, CT; 3EAST TENNESSEE STATE UNIVERSITY, JOHNSON CITY, TN; 4UNIVERSITY OF COLOMBIA, COLOMBIA; 5MARSHALL UNIVERSITY, PROCTORVILLE, OH

HeartLogic heart failure diagnostic is a personalized, remote heart failure diagnostic and monitoring system using multiple, novel physiologic sensors. It has been proven to have high sensitivity and low-alert burden that combines sensor data into one composite index that provides weeks of advance notice for detecting early signs of worsening heart failure. We present two cases, one with non-ischemic cardiomyopathy, the other with ischemic cardiomyopathy. In both cases, patients had reduced ejection fraction status post cardiac resynchronization therapy defibrillator who were monitored with HeartLogic heart failure diagnostics. In the first case, a rapid increase in sensors alarmed us for impending exacerbation of heart failure and retrospective analysis of differentials led us to diagnosis of COVID-19. The patient developed increase in respiratory rate, mean heart rate and 33 component of heart sound while decrease in thoracic impedance, ultimately with increased index. Patient was admitted, intubated and successfully recovered. Heart Logic Index helped us making the diagnosis in early stage of COVID-19 and manage effectively. We present another case of ischemic cardiomyopathy with reduced ejection fraction awaiting heart transplant due to severe multi vessel coronary artery disease who tested positive for COVID-19 with milder symptoms. He was monitored prospectively to assess changes in Heart Failure Index which remained normal correlating with the milder symptoms from COVID-19 itself. All the indices including S1, S3, respiratory rate and mean heart rate remained normal. Patient recovered without any major residual effects from COVID 19. Hence, Heartlogic index could serve as proactive alert tool like a “COVID index” to suspect, correlate with severity of symptoms and monitor the outcome in patients of heart failure diagnosed with COVID-19.

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Outcomes Following Lvad Implantation In Covid-19 Patients

JACK ZAKRZEWSKI1, LAURA COYLE2, VINH CHAU3, TRACY AICHER2, KRYSTINA CHICKERILLO1, COLLEEN GALLAGHER1, KATELYN KUPER1, CHRIS SCIAMANNA1, ANTONO TATOOLIES1; 1UNIVERSITY OF ILLINOIS, CHICAGO, IL; 2ADVOCATE CHRIST MEDICAL CENTER, OAK LAWN, IL

Purpose: End-stage heart failure patients are at an increased risk of severe disease and complications from coronavirus disease-2019 (COVID-19). Additionally, the disease increases perioperative risks. The purpose of this study was to describe the clinical course following left ventricular assist device (LVAD) implantation in patients with COVID-19. Methods: A single-center, retrospective review between March 2020 and March 2021 identified 6 patients with a history of COVID-19 who subsequently underwent LVAD implantation. Baseline characteristics, clinical course, and outcomes were examined. Results: Patients were male (83%), Black (67%), and implanted with a Heartmate 3 for destination therapy. The time from COVID-19 diagnosis to LVAD surgery ranged from 3 days to 6 months (median 40 days, [IQR 12-114 days]). All patients were supported with an intra-aortic balloon pump (IABP) and high-dose inotropes prior to implant. The median age was 60 years (IQR 57-61 years) and body mass index 30 kg/m² (IQR 24-31 kg/m²). Following implantation, 5 patients (83%) had respiratory failure greater than 7 days on ventilator support, 2 (33%) required tracheostomy, and 2 (33%) were reintubated before successful extubation. Two patients (33%) required temporary right ventricular assist device (RVAD) support, 4 patients (67%) needed continuous renal replacement therapy (CRRT), and 8 patients (13%) had ischemic strokes: two patients on postoperative day 1, and the other on postoperative day 5. The median length of hospital stay following surgery ranged from 16 to 73 days (median 53 days, [IQR 35-67 days]). Five patients (83%) were discharged from the hospital, 2 to acute inpatient rehab, 1 to a subacute rehabilitation facility, and 2 to home. Two patients (33%) were readmitted within 30 days for gastrointestinal bleeding and neuropathic pain. There was one (17%) 30-day hospital mortality due to multisystem organ failure following a stroke and the decision to withdraw care.
Table 1. Characteristics of LVAD implantation in COVID-19 patients

| Characteristics | PT 1 | PT 2 | PT 3 | PT 4 | PT 5 | PT 6 |
|----------------|------|------|------|------|------|------|
| Age, years     | 56   | 64   | 59   | 47   | 61   | 61   |
| Sex            | M    | M    | M    | F    | M    | M    |
| BMI            | 37   | 32   | 30   | 31   | 19   | 22   |
| Race/Ethnicity | Black| White| Black| Black| Hispanic| Black|
| Hypertension   | Y    | Y    | Y    | Y    | Y    | Y    |
| Diabetes       | Y    | Y    | N    | N    | N    | N    |
| Lung Disease   | Y    | N    | Y    | N    | Y    | N    |
| Chronic Kidney Disease | N    | Y    | Y    | Y    | Y    | N    |
| Prior Stroke   | Y    | N    | N    | N    | N    | N    |
| INTERMACS Profile | 3    | 3    | 3    | 3    | 3    | 3    |
| Time from +SARS-COV-2 PCR to LVAD support, days | 194 | 33 | 172 | 243 | 45 | 140 |
| Days from implant to discharge | 50 | 30 | 71 | 55 | 16 | 73 |
| Days from implant to extubation | 7 | 30 | 71 | 55 | 16 | 73 |
| Tracheostomy   | N    | Y    | Y    | Y    | N    | N    |
| RVAD Support   | Y    | N    | Y    | N    | Y    | N    |
| CRT            | Y    | Y    | Y    | Y    | N    | N    |
| Stroke         | N    | Y    | N    | Y    | N    | Y    |
| Alive at end of follow-up | Y | N | Y | Y | Y | Y |

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Use Of A Disposable At-Home System To Support Contactless Sleep Apnea Testing & Therapy Initiation In A HF Clinic During The Covid-19 Pandemic

DANIEL R. BENSIMHON, LISA M. CURRAN, HEATHER SCHUB, TRACI R. TURNER; CONE HEALTH, GREENSBORO, NC

Background: The COVID pandemic presented many challenges to providing care to our HF patients. One of our challenges was the closure of our sleep lab for sleep apnea testing and CPAP titration. We also encountered patient reluctance to perform sleep testing with re-usable home equipment. In an attempt to address these issues, we employed the WatchPAT™ ONE device (Iammare Medical) - a disposable Bluetooth-enabled technology that uses peripheral arterial tonometry - to assess patients for sleep-disordered breathing (SDB) without an in-person visit or a facemask. At-home CPAP initiation and auto-titration was performed in appropriate patients to create a fully contactless testing and treatment program. Methods: From March 2020 to March 2021 patients with suspected SDB during a virtual HF Clinic visit were referred for home sleep testing using the WatchPAT™ ONE device. Susception for SDB breathing was based on meeting at least 1 of 5 screening criteria used in our HF Clinic and a STOP-BANG score > 3. After insurance approval, a device was mailed to the patient for over-night testing. Results were uploaded into the CloudPAT platform and read by our Center's sleep cardiologist. Patients without complex SDB, underwent remote device fitting via an online DME company followed by CPAP auto-titration. Time to therapy and compliance with CPAP was assessed using the cloud-based system.

**Results:**

| Patients tested | 111 |
|-----------------|-----|
| Positive for SDB (AHI ≥ 5) | 81 (73%) |
| Patients with significant central SA (cAHI ≥ 5) | 20% |
| Patients with successful remote CPAP initiation | 24 (30%) |
| Mean time from virtual visit to therapy | 530 |

**Conclusions:** Sleep apnea is a common comorbidity in HF patients • The WatchPAT™ ONE system was an effective way to continue to test our HF patients for SDB in a virtual setting with a disease prevalence similar to standard in-lab testing and had the ability to discern between obstructive and central sleep apnea • In our initial virtual sleep apnea program, nearly 1/3 of patients with SDB went onto CPAP treatment via a remote DME turnkey service with a favorable time to treatment and a completely contactless workflow.

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Covid 19 And Pericarditis: Should We Be Worried About Tamponade?

SAMUULLAH ARSHAD1, MUHAMMAD HAMDAN GUL1, MAYA GUGLIN2; 1UNIVERSITY OF KENTUCKY, LEXINGTON, KY; 2INDIANA UNIVERSITY SCHOOL OF MEDICINE - DEPARTMENT OF MEDICINE RESIDENCY PROGRAM, INDIANAPOLIS, IN

Introduction: Little is known about cardiac manifestations of COVID-19 infection, yet cases of pericarditis, pericardial effusion and with tamponade due to COVID-19 have been reported. Because of the life-threatening nature of this complication, we wanted to investigate the features of pericardial effusion and the rate of occurrence of tamponade in patients with COVID-19. Methods: This systematic review was conducted by searching for studies in Pubmed/Medline and Google Scholar for the search terms ‘COVID-19’, ‘SARS-COV-2’, ‘Pericarditis’, ‘Pericardial Effusion’ and ‘Cardiac Tamponade’, performed on December 7, 2020. Results: A total of 47 patients with COVID-19 with pericarditis were included in the review from 39 published cases. There were 29 (62%) males and 18 (38%) females and mean age of patients was 53 years. Pulmonary infiltrates were seen in 30 (64%) patients, while 17 (36%) patients did not have pulmonary manifestations. Concomitant myocarditis was present in 16 (34%) patients. 43 (91%) had pericardial effusion: 4 (9%) had small, 10 (21%) had moderate, 3 (6%) had a large pericardial effusion and 26 (55%) patients had cardiac tamponade. 7 (15%) patients with tamponade died. Pericardiocentesis was done in 27 (57%) patients and pericardial window was created in 5 (11%) patients. Of these 13 (27%) patients had an exudative effusion while 1 (2%) patient had a transudative effusion. Conclusion: We found that pericarditis in patients with COVID-19 infection can be present in patients with pulmonary infiltrates and without them, as well as with myocarditis or as an isolated feature of cardiac involvement. The effusion is predominantly exudative. More than half of the patients with pericardial involvement present with tamponade, and mortality in this subset is high. The pattern of patients presenting solely with pericarditis and effusion without pulmonary infiltrates warrants further investigation.

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Maskophobia As A Cause Of Non-exertional Dyspnea In The Era Of Covid-19

JASMINE TRAN1, SIU-HIN WAN2; 1NORTHWESTERN UNIVERSITY, EVANSTON, IL; 2UT SOUTHWESTERN MEDICAL CENTER, DALLAS, TX

Introduction: COVID-19 can cause dyspnea through many mechanisms, and uncovering the underlying etiology greatly affects management. Diagnostic examinations can help uncover pulmonary, cardiac, or hematologic conditions that can contribute to post-viral COVID dyspnea. Hypothesis: Non-exertional dyspnea could be caused by maskophobia and not cardiopulmonary conditions. Methods: A 59-year-old woman with no history of heart failure and a history of mild exercise-induced asthma, hyperlipidemia, anxiety, depression, and migraines was hospitalized for resting dyspnea. Three months prior, she tested positive for SARS-CoV-2, and a repeat test was positive a week prior to presentation. Upon exercise, oxygen...