Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

prone to activate the necroptotic pathways. Furthermore, under inflammatory and endotoxemic stress conditions, as in SARS, ET-1–mediated effects are shifted to promote necroptosis through a potent and long-lasting RIP-3 activation,[4,5] thereby enhancing oligomerization of the ORF-3a protein and increasing the catastrophic effects of the proinflammatory necroptotic cell death on SARS-CoV-2 pathogenesis. Blocking of ET receptors with bosentan was able to inhibit the necroptosis pathway in experimental models of microvascular endothelial cells.[5] As ET receptor antagonists counteract the vicious circle of ET-1–mediated RIP-3 activation and propagation of the proinflammatory necroptotic cell death, as it happens in the worst form of SARS, we propose that it seems safe to continue ET receptor antagonists in patients on treatment with this class of drugs.

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Clinical distancing of hospitalized patients with advanced heart failure and cardiac transplantation during COVID-19

Mary Norine Walsh, MD, Ashwin K. Ravichandran, MD, MPH, Erica Seasor, MSN, BS, ASN, and Christopher T. Salerno, MD

From the Heart Failure and Cardiac Transplantation, Ascension St. Vincent Hospital, Indianapolis, Indiana.

Our hospital system includes a central 825-bed, short-term, acute-care teaching facility where heart transplantation is performed under regulatory approval. Located 5.5 miles north of this main campus is a 107-bed cardiac specialty hospital, which operates on a single-bed concept that allows patients to remain in the same bed throughout their hospital stay. All rooms allow for the entire spectrum of care, including hemodynamic monitoring, peri-operative and intensive care, and complete cardiac and device management. At the onset of the expected surge of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), all elective cardiac surgeries and procedures were cancelled on both campuses. Because the central hospital was to be redesigned for the care of patients with COVID-19, we administratively decided to transfer all patients with advanced heart failure and cardiac transplant and services to the peripheral specialty hospital in an effort to protect immunosuppressed and vulnerable patients from exposure to SARS-CoV-2. The specialty hospital did not have a single case of COVID-19 at the time of this writing.

To accomplish this goal, on March 31, 2020, the United Network for Organ Sharing Membership and Professional Standards Committee's subcommittee granted our requested temporary change of geographic location for the cardiac transplantation program to the specialty hospital.

In addition to post-transplant patients, those transferred included 1 patient awaiting transplantation, 1 patient with severe rejection, and several other patients requiring intensive care unit care owing to recent implantation of ventricular assist device (VAD) or device complication of a previously implanted VAD. Cardiac transplant physicians and surgeons continued to care for patients on both campuses, with rotation adjustments to minimize exposure between the campuses. Experienced cardiac transplant critical care nurses accompanied patients on transfer and have continued to provide critical care support at the specialty hospital.

All patients were transferred without complication or worsening illness. Concurrently, we began admitting all post-transplant patients and patients with VAD with non–SARS-CoV-2 medical illness to the specialty hospital. Patients who have symptoms suggestive of SARS-CoV-2 infection and those under investigation continue to be admitted to the central hospital. Similarly, all patients with trauma or need for neurosurgical evaluation continue to be admitted to the central hospital as well. As of now, the 1 hospitalized listed patient underwent successful cardiac transplantation at the specialty hospital.

All employees of the specialty hospital were screened daily for self-reported symptoms through an online reporting system. Employees with symptoms suggestive of viral infection were tested at a testing center remote to the hospital and did not return to work unless symptoms resolved and testing was negative. Routine testing for asymptomatic carrier status has not been performed. Personal protective equipment has been widely available, and appropriate personal protective equipment has been used by all with patient contact (surgical mask for routine care and examination, N95 mask and face shield for invasive procedures such as bronchoscopy). The hospital has imposed a no-visitor policy.

As of now, our experience with this new paradigm has been successful and without significant complications. We propose that other multihospital systems with the option of transitioning advanced heart failure services to another institution consider this approach to preserve patient safety and outcomes. The success of such an initiative will require evaluation of outcomes once the threat of the pandemic settles down.

Myocardial edema in COVID-19 on cardiac MRI

Robert Manka, MD,[a,b,c] Mihaly Karolyi, MD,[a] Malgorzata Polacin, MD,[a] Erik W. Holy, MD, PhD,[b] Johannes Nemeth, MD,[d] Peter Steiger, MD, EMBA,[e] Reto A. Schuepbach, MD,[e] Annelies S. Zinkernagel, MD,[d]

Hatem Alkadhi, MD, MPH,[a]
Mandeep R. Mehra, MD, FRCP,[f] and
Frank Ruschitzka, MD, FRCP[b]

From the [a]Institute of Diagnostic and Interventional Radiology; [b]Department of Cardiology, University Heart Center, University Hospital Zurich, University of Zurich, Zurich, Switzerland; [c]Institute for Biomedical Engineering, University and ETH Zurich, Zurich, Switzerland; [d]Division of Infectious Diseases and; [e]Institute for Intensive Care Medicine, University Hospital Zurich, University of Zurich, Zurich, Switzerland; and the [f]Department of Internal Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts.

The novel coronavirus[1] has evolved into a global pandemic. Until now, there is limited knowledge about the cardiac involvement in patients with coronavirus disease 2019 (COVID-19). We report a case of novel coronavirus pneumonia with associated acute myocardial injury[2] confirmed by biomarkers and cardiac magnetic resonance imaging (MRI).

A 75-year-old man developed fever, chills, and a productive cough on March 19, 2020. His cardiovascular risk factors included hypertension, obesity, former smoking, and renal failure (Kidney Disease Improving Global Outcomes-1). A severe acute respiratory syndrome coronavirus 2 test was positive, and home quarantine was advised; however, worsening dyspnea required hospitalization. Besides cough, fever (38.7°C), and low oxygen saturation (88%, without oxygen), laboratory results revealed elevated C-reactive protein (82 mg/liter), elevated myoglobin

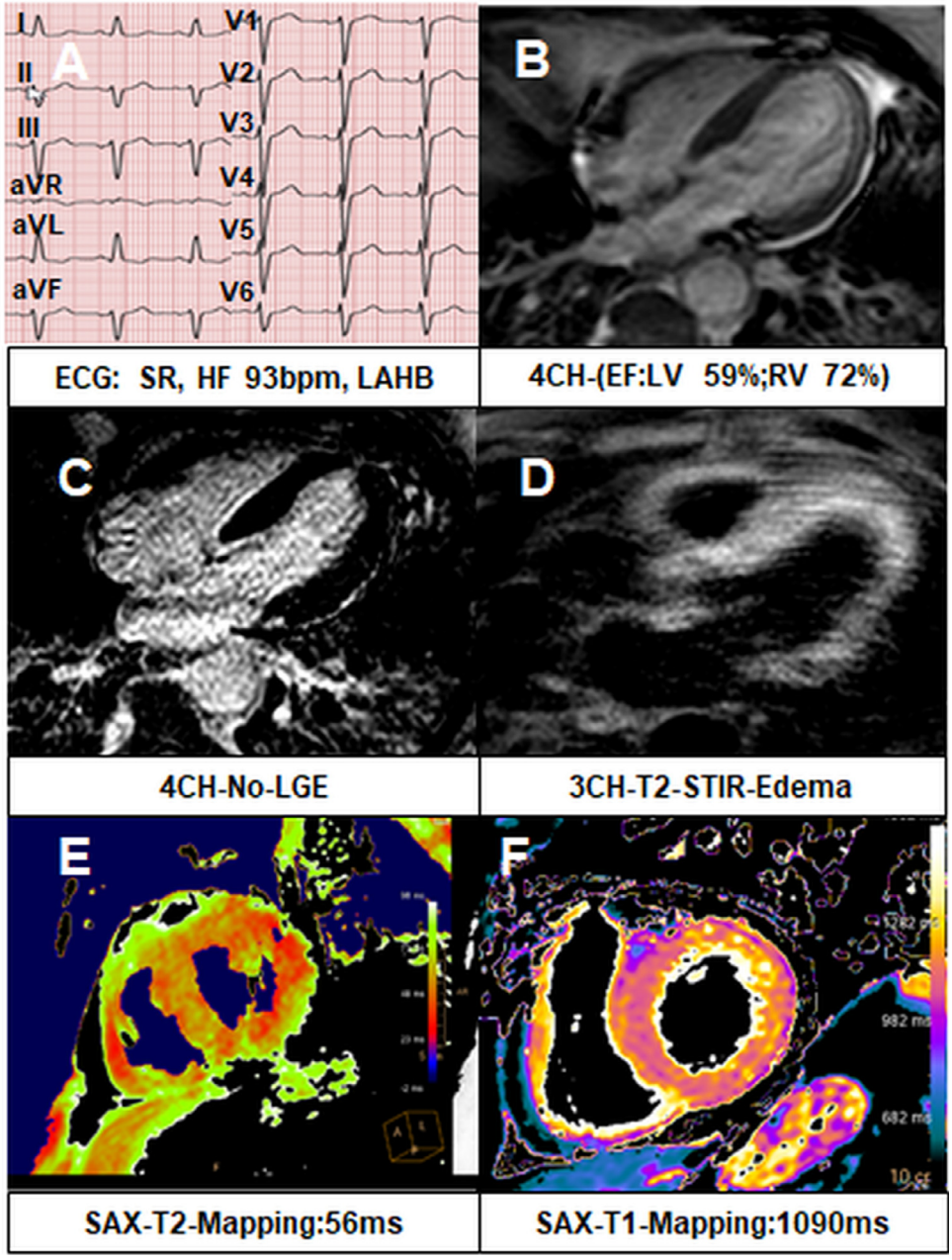

Figure 1 Cardiac MRI in COVID-19 (A) 12-lead electrocardiogram (B) Cine 4 chamber view (C) Late gadolinium enhancement 4 chamber view (D) T2 short tau inversion recovery 3 chamber view (E) T2 Mapping short axis view (F) T1 Mapping short axis view. CH, chamber; EF, ejection fraction; HR, heart rate; ECG, electrocardiogram; LGE, late gadolinium enhancement; LV, left ventricular; RV, right ventricular; SAX, short axis; SR, sinus rhythm; STIR, short tau inversion recovery.

(636 μg/liter), troponin T (high sensitive, 80 ng/liter), and N-terminal proB-type natriuretic peptide (833 ng/liter). Electrocardiogram revealed left anterior fascicular block and T-wave inversion in lead aVL (Figure 1a, 12-lead electrocardiogram). Screening tests for other viruses (Adenovirus; Coronaviridae 229E, HKU1, NL63, OC43; Human Bocavirus; Metapneumovirus; Rhinovirus/Enterovirus; Parainfluenza 1−4) were negative. Severe acute respiratory syndrome coronavirus 2 infection was reconfirmed in the laboratory. Because troponin was rising (191 ng/liter), coronary angiography was performed on March 23, 2020, and epicardial stenosis was excluded, but left ventricular (LV) end-diastolic filling pressure was elevated at 14 mm Hg. Echocardiogram performed on the same day revealed normal LV and right ventricular function with signs of concentric LV remodeling and no regional wall motion abnormality. Cardiac MRI was performed on March 24, 2020 for evaluation of possible inflammatory myocardial injury. Because of dyspnea, the cardiac MRI (Philips 1.5 Tesla) examination was performed during free breathing using mostly single-shot sequences. Normal LV and right ventricular function and no regional wall motion abnormalities were noted (Figure 1b, Video 1−Cine single-shot 3-dimensional balanced turbo-field-echo sequence—4 chamber view). No focal fibrosis was detected in late gadolinium enhancement sequences (Figure 1c, single-shot inversion recovery sequence—4 chamber view). Global edema in T2 weighted images was visible (Figure 1d, T2 [short tau inversion recovery] sequence—3 chamber view) as well as globally elevated T2 (56 ms, referent 48 $\pm$ 3 ms) (Figure 1e, T2 mapping—short-axis view) and T1 (1,090 ms, referent 989 $\pm$ 28 ms) (Figure 1f, T1 mapping—short-axis view) mapping times, suggesting acute myocardial injury. On March 26, 2020, hypoxic respiratory failure (saturation of 80%) required mechanical ventilation. The patient improved and was extubated, and the level of cardiac biomarkers declined (N-terminal proB-type natriuretic peptide 631 ng/liter, troponin 61 ng/liter) in due course.

Cardiac MRI with its unique accuracy in defining cardiac morphology and function and its ability to provide tissue characterization makes it well suited to study cardiac involvement in COVID-19. Recently, Inciardi et al[3] proved severe biventricular myocardial injury with edema and late gadolinium enhancement. In the absence of epicardial coronary artery stenosis, sub-clinical myocardial dysfunction in COVID-19 may be a consequence of an impairment of microcirculatory endothelial function observed during the early stages of the systemic inflammatory response to the infection, which portends a poor prognosis in patients with established cardiovascular disease and impaired microcirculatory endothelial function.[4] In addition, direct COVID-19–mediated infection of endothelial cells might contribute to cardiac injury.[5]

In summary, we show that elevated biomarkers of cardiac injury were associated with generalized myocardial edema without late gadolinium enhancement in cardiac MRI despite a normal echocardiogram during COVID-19.

Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.healun.2020.04.025.

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COVID-19 in a pediatric heart transplant recipient: Emergence of donor-specific antibodies

Matthew R. Russell, MD,[a] Nancy J. Halnon, MD,[a] Juan C. Alejos, MD,[a] Morris M. Salem, MD,[a,b] and Leigh C. Reardon, MD[a,c]

From the [a]Division of Pediatric Cardiology, Department of Pediatrics, University of California, Los Angeles, UCLA Mattel Children's Hospital, Los Angeles, California; [b]Division of Pediatric Cardiology, Department of Pediatrics, Kaiser Permanente, Los Angeles, California; and the [c]Ahmanson/University of California Los Angeles Adult Congenital Heart Disease Center, Los Angeles, California.

Early reports have suggested that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) associated coronavirus disease 2019 (COVID-19) generally causes mild disease in children.[1] Pediatric solid organ transplant recipients are generally more susceptible to viral respiratory infections and have increased morbidity and mortality.[2] There have been limited reports of COVID-19 disease in heart transplant recipients.[3]

A 3-year-old female child underwent orthotopic heart transplantation at 11 months of age for congenital dilated cardiomyopathy in late 2017. Her post-transplant course had been unremarkable, except for persistent Ebstein Barr virus (EBV) viremia, for which the intensity of immunosuppression had been reduced to tacrolimus monotherapy. In the first week of March 2020, the patient developed a productive cough with rhinorrhea and nasal congestion. She was afebrile with no symptoms of shortness of breath. One month before this illness, she was treated for febrile bronchiolitis as an outpatient. The potential for COVID-19 was considered, but community incidence was low at the time. The patient had no Centers for Disease Control risk factors for infection, and testing