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Novel Coronavirus Disease 2019 in a Patient on Durable Left Ventricular Assist Device Support

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In December 2019, an outbreak of a severe respiratory viral illness was first identified in the Hubei province of China. The illness was later discovered to be caused by infection with a novel coronavirus known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

To the best of our knowledge, we present here the first confirmed case of COVID-19 in a patient on left ventricular assist device (LVAD) support. Our case patient is a 66-year-old man with a history of end-stage ischemic cardiomyopathy, on HeartMate II (Abbott Laboratories, Abbott Park, IL) LVAD as destination therapy, hypertension, atrial flutter, and ischemic stroke who presented with a 4-day history of fever, cough, and shortness of breath after recent 2-month travel to Egypt. Initial physical examination revealed a body temperature of 100.9˚F (38.3˚C), normal mean arterial pressure by Doppler, and oxygen saturation of 70% on room air. Arterial blood gas revealed a partial pressure of oxygen in arterial blood of 46 mm Hg despite delivery of 100% fraction of inspired oxygen via nonrebreather ventilation. LVAD parameters were stable on presentation. A chest radiograph showed bilateral pulmonary infiltrates suggestive of multifocal pneumonia (Fig. 1a). The patient seemed to be in moderate respiratory distress, with tachypnea and accessory muscle use. Owing to concern for developing respiratory muscle fatigue and impending respiratory failure, the decision was made to pursue intubation and mechanical ventilation.

Testing for novel coronavirus returned positive for SARS-CoV-2 by polymerase chain reaction. Salient features of the patient's initial clinical laboratory trend include lymphocytopenia, transaminitis, and hyperbilirubinemia, as well as elevated creatinine, lactate dehydrogenase, D-dimer, and ferritin levels. In the following days, the patient developed progressive hypotension requiring initiation of vasopressor agents, acute oliguric renal failure requiring continuous renal replacement therapy, and refractory hypoxemia consistent with acute respiratory distress syndrome (ARDS).

Right ventricular failure was considered as a potential etiology of hypotension in the setting of LVAD and inflammatory surge. The patient was unable to be transferred to the catheterization laboratory for invasive hemodynamic assessment owing to COVID-19. Pulmonary artery catheterization was attempted at the bedside, but placement was unsuccessful. A central line was used to measure central venous pressure (17 mm Hg) and oxygen saturation (central venous oxygen saturation of 75%). Transthoracic echocardiogram revealed baseline moderate right ventricular dysfunction. LVAD parameters otherwise remained stable.

Management of ARDS was further complicated by refractory hypoxemia despite mechanical ventilation. Implementation of prone positioning was considered to assist with oxygenation; however, the presence of LVAD was determined to be a relative contraindication. Although it has previously been shown that prone positioning unloads the right ventricle in ARDS owing to improved pulmonary pressures,[1] the use of this maneuver in the setting of LVAD has not been well-described and may be adversely associated with increase in right ventricular pressures and subsequent right ventricular failure.

The patient's clinical course was later complicated by septic shock in the setting of *Escherichia coli* and *Lactobacillus species* bacteremia, hyperbilirubinemia secondary to acalculous cholecystitis with requirement for percutaneous cholecystostomy, and acute blood loss anemia secondary to gastrointestinal bleed requiring blood transfusion. Owing to prolonged ventilator-dependent respiratory failure, the patient underwent placement of tracheostomy.

The patient was treated with hydroxychloroquine 600 mg twice daily after the initial diagnosis. Oseltamivir 75 mg twice daily was added and, once liquid lopinavir-ritonavir was available at our institution, the patient was switched to lopinavir−ritonavir 400−100 mg twice daily on treatment day 5, but was discontinued in the setting of hyperbilirubinemia on treatment day 9. He was transitioned back to hydroxychloroquine 200 mg twice daily to complete a total 14-day course. The patient was deemed not a candidate for the compassionate use of remdesivir owing to poor creatinine clearance.

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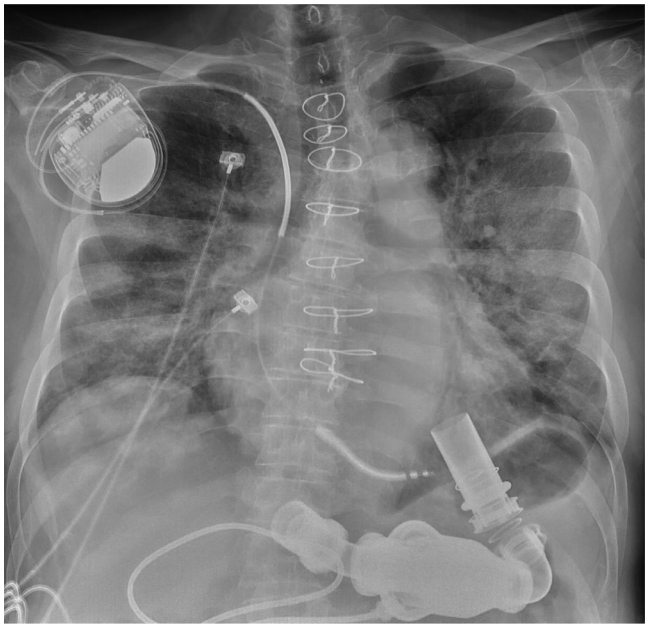
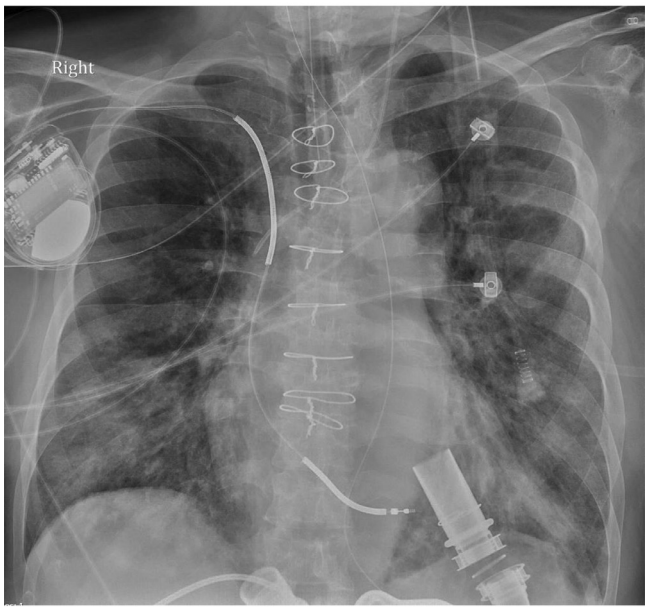


(a)



(b)

Fig. 1. (a) Chest radiograph showing bilateral patchy opacities suspicious for multifocal pneumonia on hospital day 1. (b) Chest radiograph on hospital day 13.

This report describes the first known presentation of illness secondary to SARS-CoV-2 in a patient with long-term LVAD support. At the time of writing, the patient remains critically ill; however, there has been clinical improvement. Recent evidence suggests that patients with cardiovascular comorbidities seem to be at increased risk of morbidity and mortality with COVID-19.[2] Notably, prior studies have shown that cellular immunity is compromised among long-term LVAD recipients.[3,4] This "functionally immunocompromised state" may in part explain the patient's rapid clinical deterioration and prolonged critical illness after infection with SARS-CoV-2. Increased susceptibility to viral infections, particularly COVID-19, in the setting of LVAD has not been reported previously in the literature. Furthermore, this case highlights an important consideration for the management of ARDS, as the safety and efficacy of prone positioning in the presence of LVAD is currently unknown. Finally, we propose the establishment of a COVID-LVAD registry to further understand the impact of COVID-19 on this advanced heart failure population.

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