Heart failure exacerbation as only presenting sign of COVID-19

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With the increasing number of confirmed cases and accumulating clinical data, our understanding of COVID-19 continues to evolve. Here we describe the case of a patient who was initially admitted for decompensated heart failure with reduced ejection fraction (HFrEF). Only later in his course did he develop fever that led to testing for severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Although we are aware of the common respiratory failure induced by SARS-CoV-2, we have scant information that describes cardiac manifestations caused by this novel virus.

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Introduction

Coronavirus disease 2019 (COVID-19), the syndrome caused by the novel severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has emerged as a pandemic with high morbidity. The statistics to date are persistently increasing thus highlighting the need for prompt testing and isolation of individuals who display signs and symptoms of infection to prevent further spread. At onset of illness, the typical symptoms include fever, fatigue, dry cough, myalgia, and dyspnea [1]. Cardiac manifestations have been described infrequently in medical literature [1,2]. The following is a case report of COVID-19 presenting with heart failure exacerbation.

Case report

A 68-year-old African-American man presented to his electrophysiologist for a scheduled catheter ablation of atrial flutter. He had underlying obesity (BMI 47 kg/m²), hypertension, non-ischemic cardiomyopathy with dual-chamber pacemaker, heart failure with reduced ejection fraction (HFrEF) of 30–35%, stage 3 chronic kidney disease, and type 2 diabetes mellitus. On day of procedure, the patient had no complaints, but on review of systems endorsed 20-lb weight gain and new lower extremity swelling. These findings were confirmed on physical exam along with elevated jugular venous distention at 14 cm and bibasilar crackles. The patient did not experience any unusual changes to his lifestyle and was adherent to fluid restriction, diet, and medications. Social history revealed no alcohol, tobacco, or illicit substance use. He had no known sick contacts and just returned from visiting family out of state three days prior. The ablation procedure was deferred and he was admitted for medical management of acute kidney injury with decompensated HFrEF with possible etiology being new-onset atrial fibrillation found on pacemaker interrogation.

On the night of day 5 of admission, the patient spiked fever of 39.3 °C but was otherwise asymptomatic – no fatigue, cough, myalgia, dyspnea, chest pain, abdominal pain, or diarrhea. Vital signs showed blood pressure 144/95 mmHg, heart rate 81 beats per minute, and respiratory rate 18 per minute, with peripheral capillary oxygen saturation at 92% on room air. Portable chest x-ray demonstrated mild hazy interstitial and alveolar densities in bilateral central lung fields which was confirmed on CT chest revealing multifocal patchy ground glass opacities concerning for developing superimposed pneumonia. Given the clinical scenario, COVID-19 was suspected and later confirmed via detection of SARS-CoV-2 nasopharyngeal nucleic-acid amplification testing. Contact tracing revealed that a family member the patient had recently come into contact was now also having symptoms and subsequently diagnosed with COVID-19 after detection of SARS-CoV-2.

The patient was placed on airborne precautions and given supportive medical care but renal dysfunction continued to worsen. On day 11, he developed sudden respiratory distress and a rapid requirement for escalating supplemental oxygen, and subsequent endotracheal intubation in the medical intensive care unit. Inflammatory markers revealed elevated CRP 6.4 mg/dL, ferritin 699 ng/mL, LDH 854 U/L, D-dimer >20 ug/mL, and interleukin-6 was 7305 pg/mL spurring concern for “cytokine storm”.

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storm”. The patient received compassionate treatment with hydroxychloroquine, azithromycin, and tocilizumab due to his critical illness. On day 32, he was eventually extubated.

Discussion

The common clinical presenting signs and symptoms of COVID-19 include fever, fatigue, dry cough, and dyspnea, but we also recognized atypical manifestations as the pandemic has evolved. Our patient developed decompensated HFrEF and exacerbation of atrial dysrhythmias three days after exposure to another family member with COVID-19, suggesting a possibility that the cardiac symptoms were likely due to the COVID-19 infection. However, we cannot state with certainty that there was a cause and effect relationship between the COVID-19 infection and the development of decompensated HFrEF. Our case was interesting as only later in his clinical course did he develop typical fever and respiratory disease that triggered testing for SARS-CoV-2. Cardiac complications are being described among those with COVID-19. One study of 138 hospitalized patients from Wuhan, China reported 16.7% had arrhythmias, with 7.2% developing acute myocardial injury (1). Similarly, among 187 patients with COVID-19 from the same province, 27.8% had myocardial injury which resulted with further cardiac dysfunction and developing arrhythmias (2). Although the HFrEF exacerbation could have likely been caused by new-onset atrial fibrillation rather than the COVID-19 infection, its temporal relationship with exposure to COVID-19 without any changes in lifestyle, diet, or changes in medication could suggest a possible etiology. In conclusion, this case highlights the need for clinicians who care for those with underlying cardiac disease to be attentive to the subtle cardiovascular signs and symptoms that may represent the early manifestations of COVID-19.

Author statement

All authors have seen and approved the content of the submitted revised manuscript. The paper presents original work not previously published in similar form and not currently under consideration by another journal.

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Author contribution

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Declaration of Competing Interest

The authors have no conflict of interest to declare.

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