SARS-CoV-2 infection in a patient with destination left ventricular assist device

Abstract

Recipients of LVAD for destination therapy may represent a challenge in the treatment of COVID-19. We present a case of a 58-year-old male with LVAD support complicated with SARS-CoV-2 who declines for hospital admission despite interstitial pneumonia and lower O₂ saturation. The patient received ambulatory support and treatment with anticoagulation, supplementary O₂, steroids, antibiotics, ivermectin with successful evolution and recovery.

Keywords: COVID-19, SARS-CoV-2, LVAD, heart failure

Introduction

Despite the importance of control measures to prevent severe acute respiratory distress syndrome due to coronavirus 2 infection (SARS-CoV-2), pandemic; it has been widely demonstrated that hypertension, diabetes, and cardiovascular disease have a higher risk of adverse outcomes in patients with coronavirus disease-2019 (COVID-19).³⁴

As health systems worldwide are focused in the COVID-19 pandemic, we must consider that patients with durable left ventricular assist devices (LVAD) support represent another group population at risk for the disease.³⁴ We present a case of a patient with destination LVAD therapy, who developed SARS-CoV-2 infection.

Case presentation

An overweight 58-year-old male, diagnosed with dilated cardiomyopathy (CMD) in 2015 and NYHA functional class IV. On June 20, 2018 he was implanted with the LVAD Heart Mate III ® (Abbott Laboratories, Abbott Park, IL). Its evolution remained in NYHA II functional class, with basic medication: oral anticoagulation, aspirin, sildenafli, amiodarone, levetiracetam and furosemide.

In November 2020, he developed productive cough, deterioration of his NYHA functional class to III. Given the COVID-19 pandemic era, SARS-CoV-2 infection was initially suspected, but its SARS-CoV-2 PCR test was negative and had normal chest X-ray film. He received oral moxifloxacin for 7 days. The cough decreased considerably. Three weeks later, again increases productive cough, diaphoresis, fever of 37.5°C and greater dyspnea, evolution to IV NYHA functional class, hypotension of 80/50 mmHg, tachycardia of 98 beats per minute, O₂ saturation drops to 82%. A new X-ray film showed peripheral in upper and lower regions of both lungs, ground glass opacities areas and right basal condensation (Figure 1). SARS-CoV-2 PCR testing in nasal mucosa scraping was positive. The patient refused hospitalization and self-quarantine at home and medication was indicated: Ivermectin 200 mcg/kg every 24 h for 4 days, azithromycin 500 mg every 12 h, levofloxacin 750 mg every 24 h, pravastatin 20 mg every 24 h, famotidine 20 mg every 12 h, ibuprofen 600 mg every 12 h, colchicine 1 mg every 24 h and intramuscular dexamethasone 8 mg/day at least 7 days. He required 3 l/min supplementary medicinal oxygen. On the fourth day of treatment the O₂ saturation (SpO₂), decreases to 84% despite additional increase of oxygen support to 5 l/min. It was indicated in-hospital management but patient strongly refuses it, so we decided continue home treatment. After the 7th day treatment his clinical condition and ability to tolerate exercise improves, cough and dyspnea decreases, constant saturation at 88-90% with extra oxygen at 3 l/min. At 15 days treatment SpO₂ improves to 93%, oxygen requirements decrease to 1-2 l/min and sometimes without extra oxygen supply. In chest X-ray film 3 weeks after treatment ground glass opacities disappear (Fig 2). At 4 weeks panel antibodies for SARS-CoV-2 IgG was positive. Two months later NYHA functional class II improved, with adequate physical activity.

Figure 1 Chest X-Ray film with difusse ground glass opacities.
Conclusion

It was concluded that LVAD recipients exposed to SARS-CoV-2 virus infection requires a cautious evaluation and strict follow-up for treatment and prevention of complications.

Acknowledgments

None.

Conflicts of interest

The Authors declares that there is no conflict of interest.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

References

1. Guan WJ, Ni ZY, Hu Y, et al. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med. 2020;382:1708–1720.
2. Hanaei S, Rezaei N. COVID–19: developing from an outbreak to a pandemic. Arch Med Res. 2020;51(6):582–584.
3. Drake D, Morrow CD, Kiniaw K, et al. Cardiothoracic surgeons in pandemics: ethical considerations. Ann Thorac Surg. 2020;110(2);355–358.
4. Chau VQ, Oliveros E, Mahmood K, et al. The imperfect cytokine storm: Severe COVID–19 with ARDS in a patient on durable LVAD support. JACC Case Rep. 2020;2(9):1315–1320.
5. Piperata A, Bottio T, Gerosa G. COVID-19 infection in left ventricular assist device patients. J Card Surg. 2020;1–4.
6. Bösch F, Börner N, Kemmner S, et al. Attenuated early inflammatory response in solid organ recipients with COVID–19. Clin Transplant. 2020 Oct;34(10):e14027.
7. Caly L, Druce JD, Catton MG, et al. The FDA–approved drug ivermectin inhibits the replication of SARS–CoV–2 in vitro. Antiviral Res. 2020 Jun;178:104787.
8. Kaur H, Shekhar N, Sharma S, et al. Ivermectin as a potential drug for treatment of COVID–19: an in–sync review with clinical and computational attributes. Pharmacol Rep. 2021;73(3):1–14.
9. Mahmood K, Rashed ER, Oliveros W, et al. Predisposition or protection?: COVID–19 in a patient on LVAD support with HIV/AIDS. J Am Coll Cardiol Case Rep. 2020;2(9):1337–1341.
10. Frick WH, Mallory RD, Guglin M, et al. Unusual Case of Pump Thrombosis in LVAD Patient with COVID–19. The VAD Journal. 2020;6(2): e2020622.

Discussion

Patients with cardiovascular diseases are vulnerable to complications of COVID-19. The description of cardiovascular manifestations of COVID-19 are rapidly emerging and it has been recognized as a death cause, due to associated viral cardiac injury, systemic pro-inflammatory cytokine storm with local inflammatory response and associated plaque damage in atherosclerotic coronary vessels or development of arrhythmias probably due to hypoxia and microvascular insult with myocardial damage as deleterious effect.1,5,6

No proven, effective therapies currently exist to treat SARS-CoV-2 infection, including ivermectin use.1 However with some clinical information,7,8 and local experience we have found clinical improvement in selected patients with this medication.

Piperata et al.,5 suggests that the presence of LVAD and the oral anticoagulation associated with this devices, probably decreases the cardiovascular complications of COVID-19, and also it has been demonstrated that LVAD support may decrease the inflammatory cytokines levels when compared to pre-implant samples.1,9 On the other side the inflammatory response to an infection is an event that may originate a systemic hypercoagulable state, increasing the incidence of LVAD pump thrombosis,9 and in COVID-19, has been demonstrated a high rates of arterial and venous thrombosis.10 In our patient, there was no hemodynamic adverse events or evidence of thrombosis only the transient acute respiratory failure, successfully treated.

Figure 2 Chest X-Ray film three weeks after medical treatment without ground glass opacities.