Covid-19 infection in Left Ventricular Assist Device patients

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Abstract

We describe two cases of favorable and unexpected recovery in positive patients with COVID-19, suffering from multiorgan comorbidity and already assisted with the left ventricular assist device. We have observed that, although in the presence of more comorbidities, when the maintenance of a valid support of the cardiovascular function is guaranteed, the possibility of successfully overcoming the SARS-CoV-2 infection is still alive.

Introduction

Coronavirus pandemic is a serious public-health issue (1,2). Many patients present mild symptoms like fever, cough, pharyngodynia and fatigue, the minority of them (14%) develop more severe symptoms (1-3).

Even though it has been suggested that SARS-CoV-2 can cause cardiac involvement, particularly in patients with underlying risk factors and diseases, data is still scarce and controversial (1-3). As far as left ventricular assist device (LVAD) is concerned, no reports have been published.

We describe two positive COVID-19 patients, affected by multi-organ comorbidities and assisted with LVAD, who favorably resolved.

Case report

Patient 1

On March 2020 a 61 years old patient was admitted to our Department because of acute right heart failure, 2-month after LVAD implantation for primary dilated cardiomyopathy. He was also affected by obesity with body mass index of 36 kg/mq, type 2 diabetes, Chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD), atrial flutter, moderate to severe mitral regurgitation. A Cardiac Resynchronization Therapy Defibrillator (CRT-D) had been implanted six years before.

Clinical examination at admission revealed peripheral edema, severe dyspnea and increased weight

(>10 kg compared to previous discharge). He was apyretic, hemodynamically stable with an oxygen saturation of 98%. Patient's chest X-ray is shown figure 1, with evidence of left pleural effusion. Transthoracic echocardiogram concluded for right ventricular dysfunction and pulmonary hypertension. The LVAD was functioning well. Infusion of dobutamine, levosimendan and furosemide was immediately started.

The day after the admission, a nasopharyngeal swab was positive for SARS-CoV-2. Thus he was transferred to a dedicated COVID-19 ward.

During the following days his hemodynamic condition gradually improved, with a weight loss of 10 kg. Dobutamine was progressively reduced and discontinued. We report a unique episode of fever during the

second day after admission, which was treated with paracetamol. The patient did not develop pneumoniae and, after 8 days, since clinical conditions, blood and radiological tests improved, he was discharged.

At clinical follow up two and half months later he was hemodynamically stable, without signs of right heart failure. At follow-up the swab test for SARS-CoV-2 was negative.

Patient 2

A 72 years old man, affected by post-ischemic dilated cardiomyopathy and supported with Jarvik 2000 LVAD since 2016, was admitted to our hospital for exit-site infection of the LVAD cable. He presented also several comorbidities such as type 2 diabetes, CKD, atrial fibrillation, dyslipidemia, and a history of endocarditis and two cerebral ischemic strokes. Ten days before the admission he reported an episode of fever, treated at home with paracetamol. During the hospitalization, his roommate suddenly developed an acute respiratory syndrome. He was tested for COVID 19, found positive and died after two days. For this reason, our patient underwent a nasopharyngeal swab resulting positive for SARS-CoV-2.

The exit-site infection was successfully treated with antibiotics (levofloxacin) and no other problems related to the device were found. He did not develop any pulmonary symptoms or CT-scan signs of pneumonia. Patient's chest X-ray is shown in figure 2. He is still in hospital under monitoring.

Discussion:

Coronavirus 2019 (COVID-19) has rapidly spread from China all over the world due to its highly contagious nature. Symptoms mostly involve mild respiratory problems in the vast majority of COVID-19 patients, with complete recovery within a few weeks. However, about 14% of cases are severe and 5% are critical, with an estimated mortality ranging from 2.3% to 3.83% (1-3)

Few data is currently available regarding the incidence of late complications, viral persistence, or the prognoses in different categories of patients (4). There are few and conflicting data focusing on cardiac involvement in COVID 19 patients (5).

It is established that patients at highest risk of mortality are older and with additional comorbidities. (6,7).

For instance, as reported by Yang et al (8), patients with cardiovascular diseases are vulnerable to complications of COVID-19, that can lead to death. These data have been confirmed by two reports published by Shi (6) and Guo (7) who highlighted that patients with diabetes and cardiovascular comorbidities tend to have more severe acute COVID 19 illness and higher short-term mortality rate.

On the other hand, the virus itself might cause cardiac injury, with different mechanisms. Firstly, it has been suggested that the inflammatory response in COVID 19 patients could provoke plaque erosion or rupture in patients with coronary atherosclerosis. Secondly, the respiratory disease, causing hypoxemia, might be a trigger for atrial fibrillation and other arrhythmias (8).

To summarize, the virus is able to cause death through several mechanisms, among which the exacerbation of underlying cardiac disease could cause rapid worsening of patients' clinical conditions. (6,7)

According to the first hypothesis, it can be assumed that, in presence of low cardiac output, the virus pathogenicity is favoured. In our two cases, no adverse events occurred, the hospital stay was uneventful (without multi-organ involvement) with hospital discharge in few days. Thus, although in the presence of more comorbidities, the maintenance of a valid support of the cardiovascular function seems to play a role on overcoming the SARS-CoV-2 infection.

Fried et al. (9) described the case of a COVID 19 patient with acute respiratory distress syndrome, who was initially supported with veno-venous extracorporeal membrane oxygenation (ECMO), then upgraded to veno-arterial. The authors concluded that "the addition of an arterial conduit might provide the necessary circulatory support without inducing left ventricular (LV) distension".

This concept is even more empathized in LVAD patients where LV unloading is more granted than with ECMO.

The second hypothesis deals with the protective role of anticoagulation. Preliminary data suggest that SARS-CoV-2 infection could be a precipitant factor for acute venous thrombo-embolism (10). In this scenario, the compulsory anticoagulation needed in patients with LVAD can be a protective factor as well.

In this case report, we would like to highlight the unpredictable behaviour of SARS-CoV-2 infection. Up to now, the virus has been more aggressive in fragile individuals and our patients were at extremely high risk due to their multiple comorbidities.

We report that the presence of LVAD per se and the oral anticoagulation that VAD-required, might played a role in mitigating the cardiovascular complications of COVID 19.

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Figure legends

Figure 1:

Title: Posteroanterior chest radiographies of patient 1 with HeartMate 3

Legends: Chest X-ray of Patient 1 at the admission(A) and discharge (B);

Figure 2:

Title: Posteroanterior chest radiographies of Patient 2 with Jarvik 2000

Legends: Chest X-ray of Patient 2 at the admission (A) and discharge (B).