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A Successful Case of Heart Transplantation and Post-Transplant COVID-19 infection in a Patient with Cardiogenic Shock

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ABSTRACT

Cardiogenic shock is one of the main concerns for cardiologists, especially heart failure specialists, and in the majority of cases, the underlying mechanism is an extensive acute myocardial infarction. Despite various interventional and therapeutic advancements in the management of these patients, the mortality and morbidity of these patients remain high and most noninvasive therapies are considered ineffective. Urgent revascularization and application of short-term mechanical circulatory support devices and early referral to heart transplant centers may improve outcomes. Herein we describe a 50-year-old patient presenting with cardiogenic shock, who was successfully treated by VA-ECMO and urgent heart transplant and experienced COVID-19 infection post-transplant.

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Introduction

Cardiogenic shock is one of the major complications of Acute Myocardial Infarction (MI), occurring in about 10% of patients with MI (1). According to the European Society of Cardiology, cardiogenic shock is defined as hypotension accompanied by clinical and laboratory signs of hypoperfusion despite increased filling pressures (2). The occurrence of cardiogenic shock is associated with higher mortality and morbidity in patients with acute MI, estimated to be about 40% (3). Furthermore, the management of such patients can be very challenging. Urgent revascularization and application of short-term mechanical circulatory support (MCS)

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devices and early referral to heart transplant centers may improve outcomes (1, 4). Herein, we describe a 50-year-old patient presenting with cardiogenic shock following extensive acute myocardial infarction, who was successfully treated by VA-ECMO and urgent heart transplantation, but experienced COVID-19 infection after discharge.

Case Presentation

A 50-year-old man complaining of prolonged severe chest pain visited the emergency department. The patient was diagnosed with extensive anterior myocardial infarction, and he received emergent thrombolytic therapy. Due to persistent retrosternal chest pain despite receiving reteplase infusion, he was referred to our tertiary center (Rajaie Cardiovascular, Medical, and Research Center). At the time of admission, his vital signs were HR=118 beats/minute, BP: 110/80 mmHg, Temp: 37.6° centigrade, and RR: 18/minute. Chest wall auscultation revealed rales at the base of the right lung and the remaining physical examination was unremarkable. The obtained electrocardiogram revealed sinus rhythm and ST elevation in V1-V5, I, and avL (Figure 1). Bedside echocardiography indicated a left ventricular ejection fraction of 15%, apical aneurysm, mild mitral regurgitation, akinesia of the anterior wall, normal right ventricular size with moderate systolic dysfunction, with no pericardial effusion or mechanical complications. After a short time, he became hemodynamically unstable due to VF/VT rhythm in the emergency department and received four 150-200 J synchronized cardioversion shocks. The patient underwent emergent angiography revealing a patent left main artery, total thrombotic ostial occlusion of the anterior descending artery (LAD), significant proximal stenosis of right coronary artery, and patent left circumflex artery. Rescue PCI (percutaneous coronary intervention) was done on LAD by XIENCE Alpine™ Stent DES 3.5*28. After the angioplasty, the patient experienced a drop in blood pressure and decreased level of consciousness leading to intubation and insertion of intra-aortic balloon pump (IABP) in the Cath lab. According to the recent guidelines of managing cardiogenic shock (5) and our heart team opinion, he was not a candidate for full revascularization and PCI on RCA. His blood pressure and peripheral perfusion recovered temporarily and he was weaned from IABP after 3 days. However, a short time after weaning from IABP, he experienced repeated cardiogenic shock. At this time the patient's creatinine increased to 7 resulting in considering administration of continuous renal replacement therapy (CRRT) and scheduling for ECMO implantation.

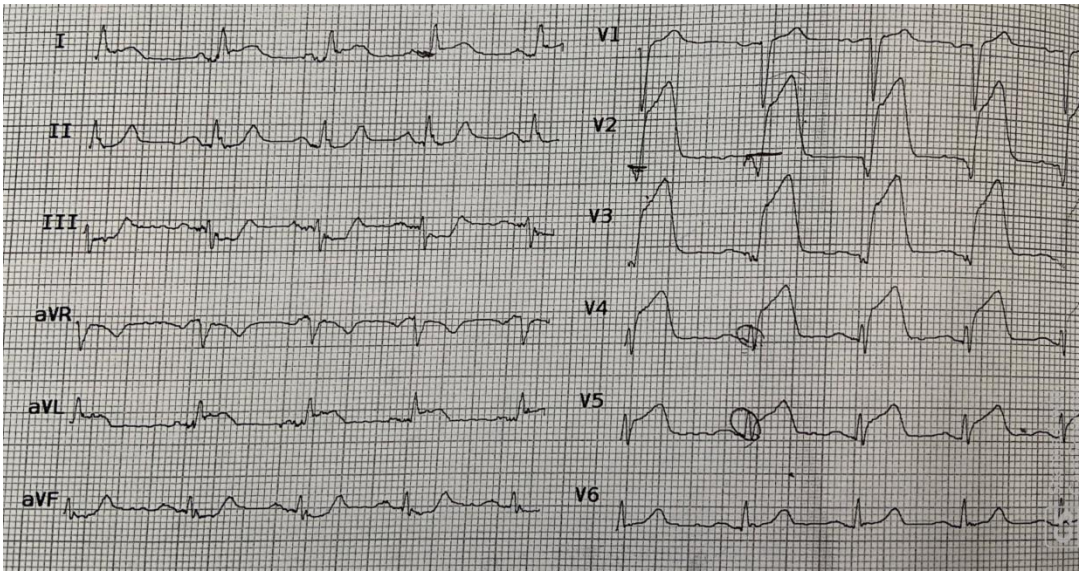

Figure 1. The initial ECG revealed sinus rhythm, ST elevation in V1-V4, I, avL.

Inotrope and vasopressor support was initiated for the patient, as well as close monitoring of organ perfusion and serial laboratory examination. Table 1 demonstrates baseline laboratory data and its course during hospitalization. On day 22, the patient was transferred to the operating room for ECMO implantation due to cardiogenic shock INTERMACS 2 status. The VA-ECMO was implanted through percutaneous cannulation of the right femoral vein and left femoral artery. After 2 weeks he was still inotrope and ECMO dependent and because of no recovery and the absence of ventricular assist devices in our center, an urgent heart transplant was planned for the patient. Fortunately, a size match donor heart was found in a short time period and heart transplant and ECMO removal were performed simultaneously. Transplantation was done using the Shumway biatrial technique with a pump time of 131 minutes and aortic clamp time of 83 min. The first right heart catheterization (RHC) and endomyocardial biopsy (EMB) were performed 12 days after heart transplant. The ISHLT rejection grade was zero and the patient did not experience any rejection symptoms or complications during the indexed hospitalization. He was discharged with normal biventricular function and no significant valvular abnormalities on follow up echocardiography exam. One month later, the patient visited the emergency department with complaints of fever and myalgia, and due to the COVID-19 epidemic, we performed COVID-PCR and lung HRCT which were both positive for COVID-19 pneumonia (Figure 2). The patient received standard dose of remdesivir while continuing his previous immunosuppressive regimen, and was discharged a few days later in a good general condition. Six months later in surveillance RHC hemodynamic parameters seemed to be acceptable (table-2), however, the biopsy revealed acute rejection ISHLT grade 2R. The patient received high-dose intravenous corticosteroid and maintenance immunosuppressive therapy was adjusted, resulting in dissolving signs of any rejection on subsequent EMB. After one-year follow-up, he remained asymptomatic, and on the first coronary angiography patent coronary arteries were demonstrated.

Table-1. Laboratory data during hospitalization

	baseline	Before ECMO implantation	Before transplantation
Troponin	50 ng/dl	36 ng/dl	3.49 ng/dl
FBS	146 mg/dl	171 mg/dl	188 mg/dl
BUN	24mg/dl	81 mg/dl	49 mg/dl
Creatinine	1.1 mg/dl	6 mg/dl	0.8 mg/dl
K	4.3 mEq/lit	4.6 mEq/lit	4.2 mEq/lit
Na	139 mEq/lit	143 mEq/lit	136 mEq/lit
Mg	2.2 mg/dl	2.7 mg/dl	2.1 mg/dl
LDL	160 mg/dl	-	-
TG	142 mg/dl	-	-
SGOT	520 IU/L	396 IU/L	25 IU/L
SGPT	92 IU/L	427 IU/L	93 IU/L
ALK-ph	228 IU/L	362 IU/L	101IU/L
Total bilirubin	2.1 mg/dl	1.5 mg/dl	0.9 mg/dl
Hemoglobin	13.5 g/dl	12.5 g/dl	8 g/dl
WBC	13500 cells/mm3	9100 cells/mm3	12100 Cells/mm3
Platelet	234000 10^3mm3	203000 10^3 mm3	221000 10^3 mm3
PH	7.34	7.41	7.45
HCO3	25	21	24
PCO2	45	33	36

Table 2. Surveillance RHC results

	First	After 6 months	After 24 months
Cardiac output (Fick method)L/min	5	4.3	5.4
Cardiac index L/min/m2	2.7	2.2	2.8
Systemic arterial pressure (mmHg)	130/80	130/90	110/70
Mean CVP (mmHg)	4	6	2
PA pressure(mmHg)	24/4	22/8	24/12
PCWP(mmHg)	6	8	6
Systemic vascular resistance(WU)	18	22.03	15
Pulmonary vascular resistance(WU)	1	1.08	1.88

Abbreviation: CVP: central venous pressure, **PCWP:** pulmonary wedge pressure, **PA:** pulmonary artery

Echocardiography at the same time showed normal left ventricular size with an ejection fraction of 50%, normal right ventricular size, and function, mild mitral and tricuspid regurgitation without signs of pulmonary hypertension or pericardial effusion and fortunately, after 3 years, his heart is doing well on repeated echocardiography exams and biopsies.

Discussion

We presented a case of extensive myocardial infarction complicated by cardiogenic shock, which was persistent despite the insertion of short-term mechanical circulatory supports (i.e. IABP and VA-ECMO). The patient became a candidate for heart transplantation according to the last guidelines in the management of heart failure (4) and experienced a lucky end in spite of several factors worsening his prognosis such as ventricular arrhythmia, repeated cardiogenic shock, and acute cellular rejection after COVID infection.

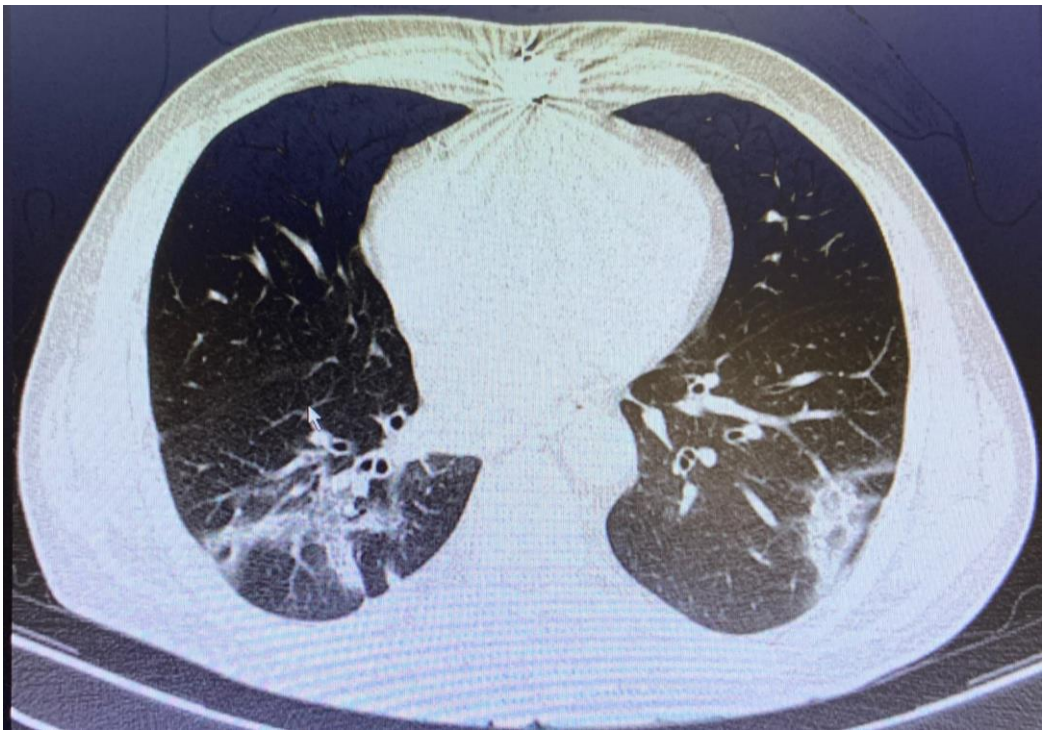

Figure 2. The HRCT revealed a multiple ground glass pulmonary opacities in both lungs due to COVID-19.

It should be noted that at the time of the case presentation, LVAD was not available in our center and a size-match donor heart was available in a short time period. Although routine use of VA-ECMO in cardiogenic shock is not superior to initially conservative therapy (6), our patient was unresponsive to full medical treatments due to extensive myocardial damage and insufficient blood supply to other vital organs. Thus, the administration of VA-ECMO would preserve organ function in order to prepare the patient for heart transplant as a destination therapy. There are few reports of successful heart transplant in myocardial infarction complicated by ventricular septal rupture, addressing its high mortality rate (7). Herein we propose that a successful urgent heart transplant could improve the patient's outcome in persistent cardiogenic shock irrespective of underlying mechanism but with other poor prognostic factors, and may serve an initial option in case of failing conservative and revascularization managements, with acceptable long-term survival.

Additionally, there are limited reports of COVID-19 infection in heart transplant recipients responding well to the administration of remdesivir (8-10). Our patient was infected with COVID-19 one month after heart transplantation. Despite respiratory involvement, he responded well to the administration of standard dose of remdesivir without the need for reducing the dose of immunosuppression drugs, based on close monitoring of mentioned drugs' serum level. There are also a few case reports of acute cellular rejection after COVID-19 infection among heart transplantation patients (11, 12). Hanson and colleagues proposed that the pathological characteristics of post-transplantation tissue rejection in COVID-19 patients are similar to COVID-19 related cardiac injury in non-transplant cases (11). Our patients' long term outcome was not affected by acute rejection probably related to COVID-19 infection; However, further studies are required to shed light on the consequences and management of COVID-19 infection in heart-transplanted patients.

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