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CASE REPORT
Heart failure

Cardiogenic shock due to probable SARS-CoV-2 myocarditis—a case report

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Background Since the start of the COVID-19 pandemic, many case reports have been presented describing different cardiac symptoms due to the SARS-CoV-2 infection. However, severe cardiac failure due to COVID-19 seems to be rare.

Case summary A 30-year-old woman presented with COVID-19 and cardiogenic shock due to a lymphocytic myocarditis. Since she deteriorated under treatment with inotropes, she was referred to our centre, and veno-arterial extracorporeal life support was started. Subsequently, the aortic valve only opened sporadically, and spontaneous contrast appeared in the left ventricle (LV), pointing towards difficulties with unloading LV. Therefore, an Impella for venting the LV was implanted. After 6 days of mechanical circulatory support, her heart function recovered. All support could be weaned, and 2 months later, she had made a full recovery.

Discussion We presented a patient with severe cardiogenic shock due to an acute virus-negative lymphocytic myocarditis associated with a SARS-CoV-2 infection. Since the precise aetiology of SARS-CoV-2-related myocarditis remains to be elucidated and no virus could be detected in the heart, a causal relationship remains speculative.

Keywords COVID-19 • Myocarditis • ECLS • Impella • Case report

ESC Curriculum 7.3 Critically ill cardiac patient • 6.4 Acute heart failure

Learning Points

- COVID-19 myocarditis can lead to severe cardiogenic shock requiring mechanical circulatory support.
- The use of an Impella as an addition to veno-arterial extracorporeal life support may improve left ventricle venting.
- Cardiogenic shock as a complication of COVID-19 myocarditis can resolve within days.

Introduction

COVID-19 infections are well known for their pulmonary involvement. However, other organ systems may suffer from this infection as well. Cardiac symptoms are well described; however, severe cardiac failure seems to be rare.[1] In this report, we describe a patient with severe cardiac failure requiring mechanical support with veno-arterial extracorporeal life support (V-A ECLS), complicated with poor mechanical unloading of the LV (left ventricle).

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Timeline

Time	Events
Day 1. Presentation at regional hospital	COVID-19 with symptoms of fever, chest pain, nausea, and collapse.
	Signs of a cardiac tamponade -> pericardiocentesis.
Day 2	Biventricular heart failure with cardiogenic shock.
Day 3. Admission tertiary ICU	Start VA-ECLS, 2.6 L/min ECLS flow.
Day 4.	Spontaneous contrast left ventricle and sporadic opening aortic valve -> implantation Impella and ECLS flow reduced to 2 L/min.
Day 5.	Biopsy: lymphocytic myocarditis, SARS-CoV-2 RNA negative. Start methylprednisolone 1000 mg daily for 3 days.
Day 6.	Improved cardiac contractility. Reduction of ECLS flow.
Day 8.	Explantation of ECLS and Impella.
Day 10.	Weaned from ventilator and inotropic support
Day 11.	Full recovery of right and left ventricles. Transferred to regional hospital.

Case presentation

A 30-year-old female presented at the emergency department of a regional hospital with COVID-19 and symptoms of fever, chest pain, nausea, and syncope. Remarkably, she had no pulmonary symptoms. She was hypotensive with a blood pressure of 75/40 mmHg. Physical examination revealed signs of hypoperfusion: a cool, mottled skin and lactate of 3.9 mmol/L (normal <1.4 mmol/L) (Society for Cardiovascular Angiography and Interventions classification for cardiogenic shock: SCAI-C). Her medical history was unremarkable, and she did not take any medication. Her electrocardiogram (EKG) showed a sinus tachycardia of 139 b.p.m. without signs of ischaemia. Echocardiography showed signs of a cardiac tamponade for which an urgent pericardiocentesis was performed. She was admitted to the coronary care unit, and dexamethasone and anti-SARS-CoV-2 monoclonal antibody (REGEN-COV, Regeneron, Tarrytown, USA) were administered. After an initial improvement in her symptoms due to the pericardiocentesis and inotropes (SCAI-C), she subsequently deteriorated as a consequence of biventricular heart failure. Troponin T levels increased up to 8041 ng/L (normal <14 ng/L), and lactate levels rose to 6.3 mmol/L. Due to progressive cardiogenic shock, she was intubated and transported to our tertiary intensive care unit for possible mechanical circulatory support 2 days later. At presentation to our hospital, the patient was still in cardiogenic shock (SCAI-D) since there was oliguria, hypotension (77/64 mmHg), and tachycardia (144 b.p.m., *Figure 1*) despite high-dose administration of inotropes (enoximone 9.1 μg/kg/min) and vasopressors (norepinephrine 0.6 μg/kg/min).

Quick-look transthoracic echocardiography (TTE) displayed poor left and right systolic ventricular function with only basal contractions. Veno-arterial extracorporeal life support was initiated (Cardiohelp, Getinge, Goteborg, Sweden). An arterial 19 French cannula was introduced in the right femoral artery as well as a 6 French antegrade cannula in the superficial femoral artery to preserve leg perfusion, and a venous 25 French cannula was introduced in the right jugular vein. Extracorporeal life support flow was maintained at 2.6 L/min resulting in normalization of lactate levels and an increase in diuresis.

The following morning, transoesophageal echocardiography revealed spontaneous contrast in the apex of the heart, a LV ejection fraction of ~5–10% and only sporadic opening of the aortic valve. To further unload the LV and prevent intracardiac thrombus formation, an Impella CP (Impella CP Smart Assist, Abiomed, Aachen, Germany) was implanted via the left femoral artery, functioning as left ventricular vent. At the P3 setting of the Impella, we attained a calculated flow of 1.8 L/min. Subsequently, V-A ECMO flow was decreased to 2 L/min. This resulted in improved contractility of the LV, improved opening of the aortic valve and decreased spontaneous left ventricular contrast, whereas the right-sided radial artery PaO2 remained normal. The latter is used as a marker for the oxygenation of the blood in the LV. Depending on the ratio cardiac output/ECLS flow, it may be a marker for the oxygenation of blood to the coronary arteries and brain arteries.

A right ventricle biopsy of the myocardium showed a lymphocytic infiltrate, but no SARS-CoV-2 RNA could be detected. There were no findings compatible with eosinophilic or giant cell myocarditis. We concluded that there was a lymphocytic myocarditis (most probably due to COVID-19) and high-dose methylprednisolone was administered (1000 mg intravenously during 3 days. Viral molecular detection and bacterial cultures of pericardial effusion remained sterile. Real-time polymerase chain reaction (PCR) detected SARS-Cov-2 in nasopharyngeal swabs and sputum samples (cycle threshold 21).

Daily transthoracic and transoesophageal echocardiography showed improvement of the cardiac contractility and function. Four days after V-A ECLS implementation, the flow of the V-A ECLS could be reduced. On the 6th day of admission to our centre, both Impella and V-A ECLS were discontinued and explanted. The patient was subsequently weaned from mechanical ventilation and inotropic support on day 8 after referral and was transferred to the referring hospital the day after. Echocardiography demonstrated a full recovery of both right and left ventricular function. Troponin T levels had decreased to 643 ng/L. On follow-up 2 months later, she had made a full recovery and was taken off medication by her own cardiologist.

Discussion

Since the start of the COVID-19 pandemic, many case reports have been presented describing different cardiac symptoms due to the SARS-CoV-2 infection, such as arrhythmias, acute coronary syndrome, and myocarditis.[1]

The aetiology of COVID-19 related myocarditis is still unclear. It remains to be elucidated whether the virus itself (in which case a sample error may be the explanation for the virus-negative biopsy) or its systemic effects cause myocardial injury. For example, whereas Sala et al. also presented a patient with acute myocarditis with a virus-negative lymphocytic myocarditis,[2] others did find virus particles in the myocardium.[3]

Since there was little pulmonary involvement and a low C-reactive protein of 52 mg/L, no interleukin-6 inhibitor was administrated for the SARS-CoV-2 infection in our patient.

In general, treatment options for viral myocarditis are limited to heart failure therapy and supportive care. We did start methylprednisolone although the use of corticosteroids in viral myocarditis is controversial.[4] Since cardiac recovery occurred within days, it was thought to be unlikely due to methylprednisolone. Therefore, methylprednisolone was discontinued.

The use of V-A ECLS will improve the oxygen delivery to the tissues but will also increase LV afterload. Poor mechanical unloading of the LV can lead to pulmonary oedema or even thrombus formation within the LV.[5] Adding or increasing inotropes and/or lowering ECLS flow might resolve this; however, this may cause oxygen delivery to decrease too much. In this case, unloading the LV might be necessary. Unloading the LV can be done by using an Impella (*Figure 2*) or intra-aortic balloon pump (IABP) or

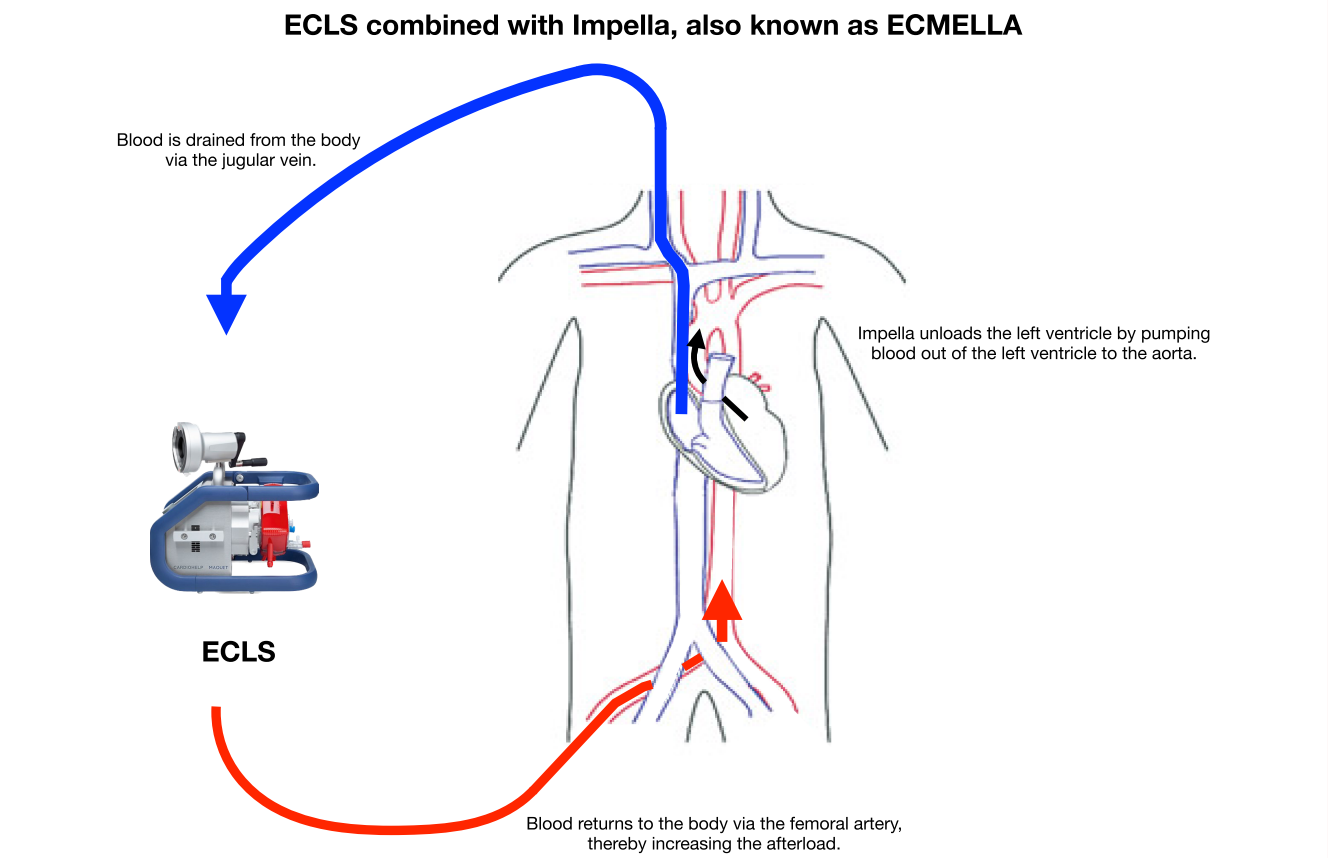

Figure 1 Electrocardiogram at presentation to our hospital. Sinustachycardia, 150 b.p.m., intermediate heart axis, normal conduction times, Q-waves in V1-2, diffuse ST elevation in all leads besides aVR (ST depression), no reciprocal ST depression, low voltage. ECLS, extracorporeal life support.

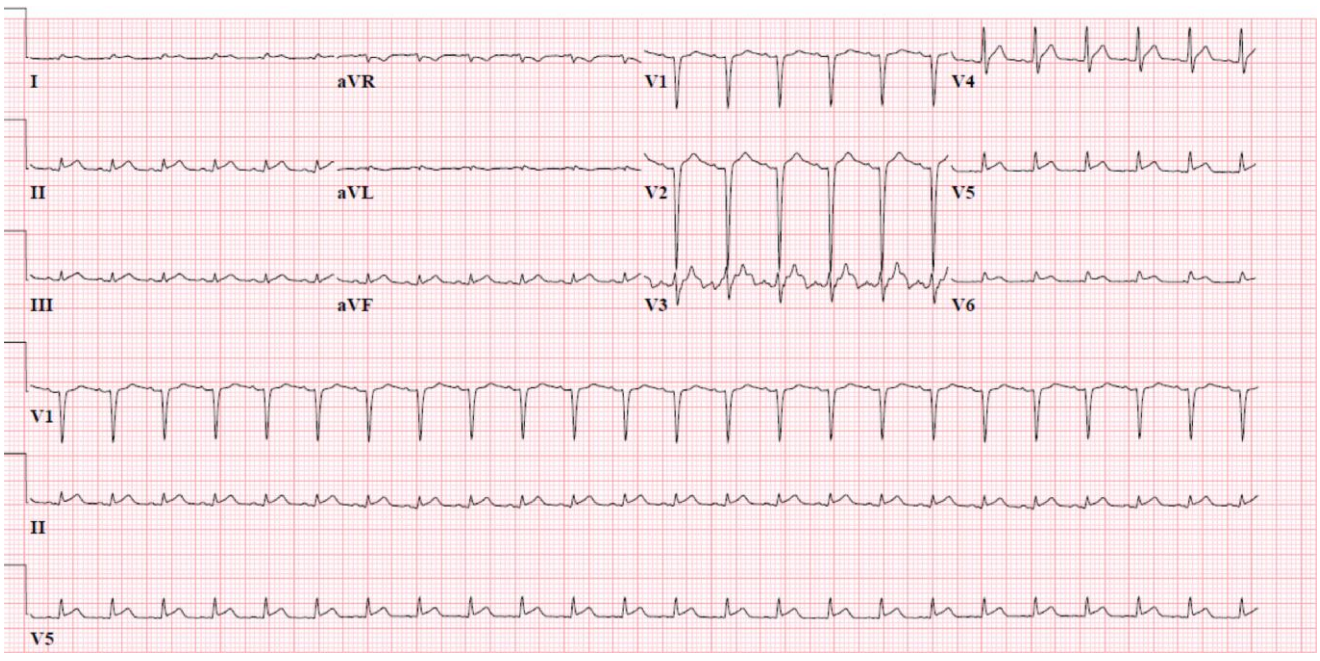

Figure 2 Extracorporeal life support and Impella.

a left atrium/ventricle vent (either directly or percutaneous via an atrioseptostomy) or by using a temporarily right ventricle and left ventricle assist device. It is not clear which is the better option, IABP or Impella. We normally use the Impella. Impella use in cardiogenic shock patients treated with V-A ECLS has been associated with lower mortality; however, patient selection and timing are still a matter of debate.[6]

Since the recovery of the heart was spectacular and signs of haemolysis (most likely due to Impella use) were increasing, we decided to explant the ECLS and the Impella at the same time. Usually, we would choose to wean and explant the ECLS as soon as possible, whereas the Impella use would be maintained to support the LV by lowering its afterload.

Until now, only a few patients have been described with severe cardiac failure probably due to SARS-CoV-2 infection requiring implementation of V-A ECLS combined with a LV unloading device like the Impella.[7] As in our patient, recovery of cardiac function within a few days is not uncommon.[3,7,8]

In conclusion: we presented a patient with severe cardiogenic shock due to an acute virus-negative lymphocytic myocarditis associated with a SARS-CoV-2 infection. After 6 days of mechanical circulatory support, the patient made a full recovery. The precise aetiology of SARS-CoV-2-related myocarditis remains to be elucidated.

Lead author biography

Dr. Joep M. Droogh is an intensivist at the University Medical Centre Groningen, the Netherlands, where he primarily treats patients after cardiothoracic surgery. His interests are ventilation, ECMO, (other) mechanical circulatory support and the interhospital transport of critical ill patients.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as *Supplementary data*.

Consent: Consent for publication was given by the patient in accordance with COPE guidelines.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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