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CASE REPORT

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Veno-arterial extracorporeal membrane oxygenation for COVID-19-associated acute myocardial injury complicated by refractory cardiogenic shock

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Abstract

Cardiovascular system involvement and its negative prognostic impact have been increasingly identified in coronavirus disease 2019 (COVID-19) patients. Optimal medical treatment allows for safe management of most of these cardiovascular presentations while COVID-19-associated refractory cardiogenic shock could be rescued by veno-arterial extracorporeal membrane oxygenation (VA-ECMO). We present a case of acute myocardial injury related to COVID-19 complicated by refractory cardiogenic shock and treated by VA-ECMO implantation.

KEYWORDS
cardiovascular pathology

1 | INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the etiologic agent of the pandemic coronavirus disease 2019 (also known as COVID-19), whose clinical presentation can vary from mild respiratory symptoms to severe multiorgan failure. The respiratory tract is most frequently affected and acute respiratory distress syndrome (ARDS) is the leading cause of mortality.[1] However, cardiovascular system involvement and its negative prognostic impact have been increasingly identified in COVID-19 patients.[2]

Myocardial injury in COVID-19 has not been completely understood and seems related to various pathophysiologic mechanisms as cytokine storm, viral myocarditis, and stress cardiomyopathy.[2] Optimal medical treatment, including inotropes and vasopressors, allows for safe management of most of these cardiovascular presentations whereas COVID-19-associated refractory cardiogenic shock could be rescued by veno-arterial extracorporeal membrane oxygenation (VA-ECMO).[3,4]

We present a case of acute myocardial injury related to COVID-19 complicated by refractory cardiogenic shock without ARDS and treated by VA-ECMO implantation.

2 | CASE REPORT

A previously healthy 30-year-old woman (170 cm, 67 kg, body mass index 23.1 kg/m^2) was admitted for fever, chest pain, and worsening dyspnea in the last 5 days. At presentation, her blood pressure was 80/50 mm Hg and oxygen saturation 96% on 3 L of oxygen by nasal cannula. The electrocardiogram showed sinus tachycardia at 110 bpm and a diffuse ST-segment elevation (more marked in the lateral and inferior leads; Figure 1). Blood analysis revealed leukocytosis with lymphopenia (white blood cells count 14.9×10^9/L, neutrophils 81.5%, and lymphocytes 10.8%) and increased high-sensitivity troponin I (5891 ng/L). Arterial blood gas analysis demonstrated compensated respiratory alkalosis with hyperlactatemia (pH 7.43, pCO_2 24.8 mm Hg, bicarbonates 16 mmol/L, base excess

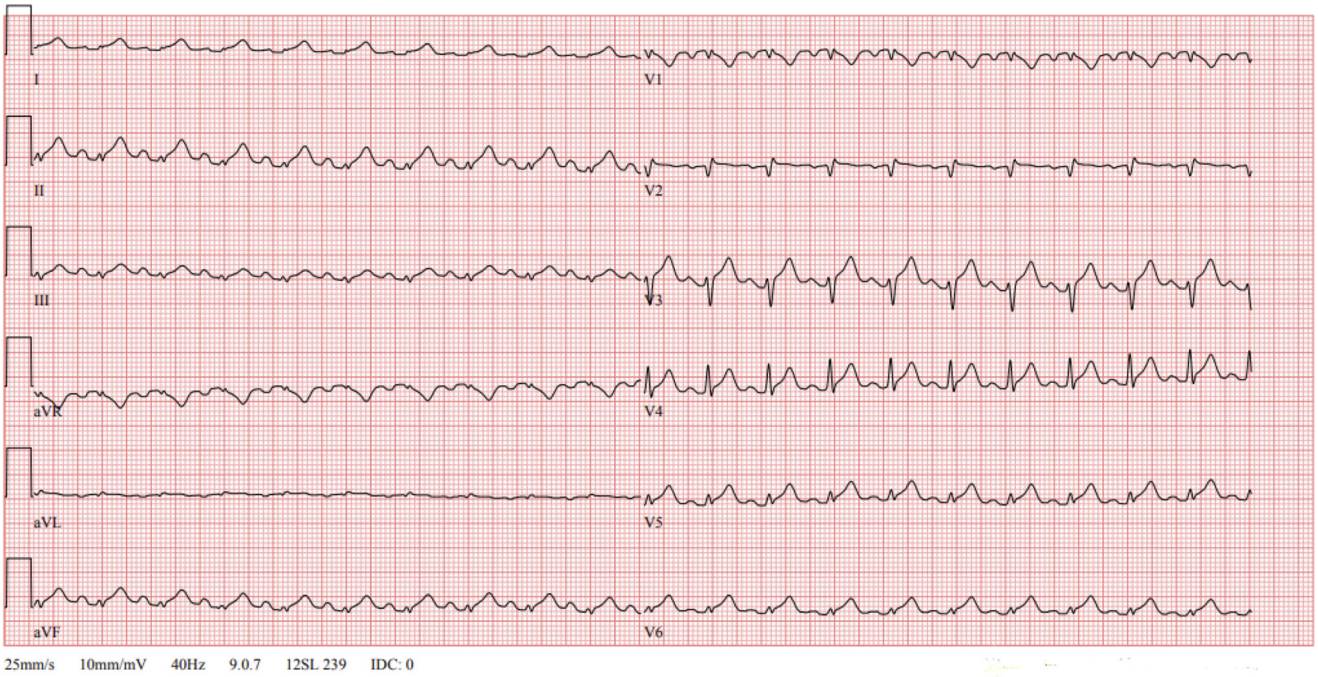

FIGURE 1 Baseline electrocardiogram performed at hospital admission showing sinus tachycardia and ST-segment elevation more marked on lateral and inferior leads

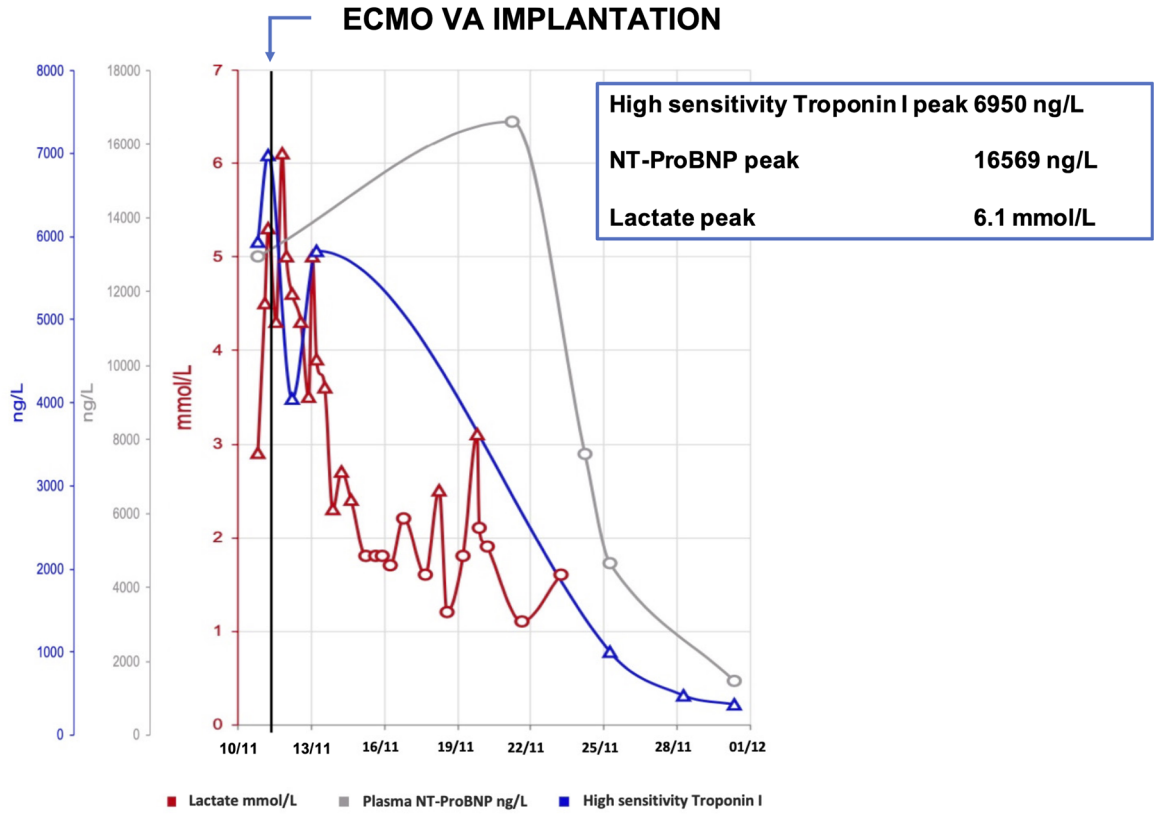

FIGURE 2 Temporal trend of blood analysis. NT-ProBNP, N-terminal pro-brain natriuretic peptide; VA-ECMO, veno-arterial extracorporeal membrane oxygenation

−7.5 mmol/L, and lactate 2.9 mmol/L). The nasopharyngeal swab was positive for SARS-CoV-2 on polymerase chain reaction test.

The echocardiography showed a 20-mm pericardial effusion compressing the right heart chambers and left ventricular systolic dysfunction (left ventricular ejection fraction [LVEF] 35%) with global hypokinesis. We performed a percutaneous subxiphoid pericardiocentesis (evacuation of 200 ml of citrine yellow effusion) and introduced an inotropic support with dobutamine (5 μg/kg/min) leading to a slight improvement in the hemodynamics. The subsequent course was characterized by the rapid development of cardiogenic shock in less than 12 h with signs of systemic hypoperfusion (systemic arterial pressure <60 mm Hg, sweating, vomiting, oliguria, and lactate 4.5 mmol/L) and further degradation of the left ventricular systolic function (LVEF 10%, left ventricular outflow tract time-velocity integral 6 cm) despite an escalation of the inotropic (dobutamine 10 μg/kg/min) and vasopressor (noradrenaline 0.17 μg/kg/min) support. There was no evidence of right ventricular dysfunction. The patient was sedated, intubated and mechanical ventilation was instituted. After a multidisciplinary discussion, we opted for the implantation of peripheral VA-ECMO as a bridge to recovery. As per institutional protocol,[5] the implantation of the VA-ECMO was performed in a surgical manner in our operatory room. Anticoagulation with unfractionated heparin was titrated to achieve an anti-Xa activity between 0.30 and 0.70 IU/ml during ECLS support. The progressive hemodynamic stabilization led to complete myocardial recovery allowing for VA-ECMO removal five days later. No complications related to VA-ECMO were noted. The temporal trend of high-sensitivity troponin I, N-terminal pro-brain natriuretic peptide, and lactate is depicted in Figure 2. The patient did not require adjunctive therapies (inhaled nitric oxide, prone positioning, and conversion to a veno-arterial-venous or veno-venous ECMO) owing to the absence of ARDS. The patient experienced ventilator-associated pneumonia (*Streptococcus pneumoniae* and *Haemophilus influenzae*) and was weaned from mechanical ventilation two days after VA-ECMO removal. No arrhythmic events were enregistered at any time-point during the hospitalization. Serial echocardiographic controls showed a complete recovery of the left ventricular systolic function (LVEF 60%, left ventricular outflow tract time-velocity integral 22 cm) and she was transferred to a rehabilitation center.

From an etiologic standpoint, coronary angiography performed during VA-ECMO support found no evidence of significant lesions. Routine blood assays (cytomegalovirus, Ebstein-Barr virus, and parvovirus B19) were negative. The pathologic study of the endomyocardial biopsy samples performed during VA-ECMO support showed slight interstitial fibrosis without inflammation. Immunohistochemical staining (CD3, CD20, and CD68) confirmed the absence of interstitial inflammation. Cardiac magnetic resonance imaging showed an area of myocardial late enhancement compatible with the diagnosis of myocarditis (Figure 3). At 3-month follow-up, echocardiography confirmed the complete myocardial recovery (LVEF 65%) and cardiac magnetic resonance imaging showed no signs of myocardial residual fibrosis.

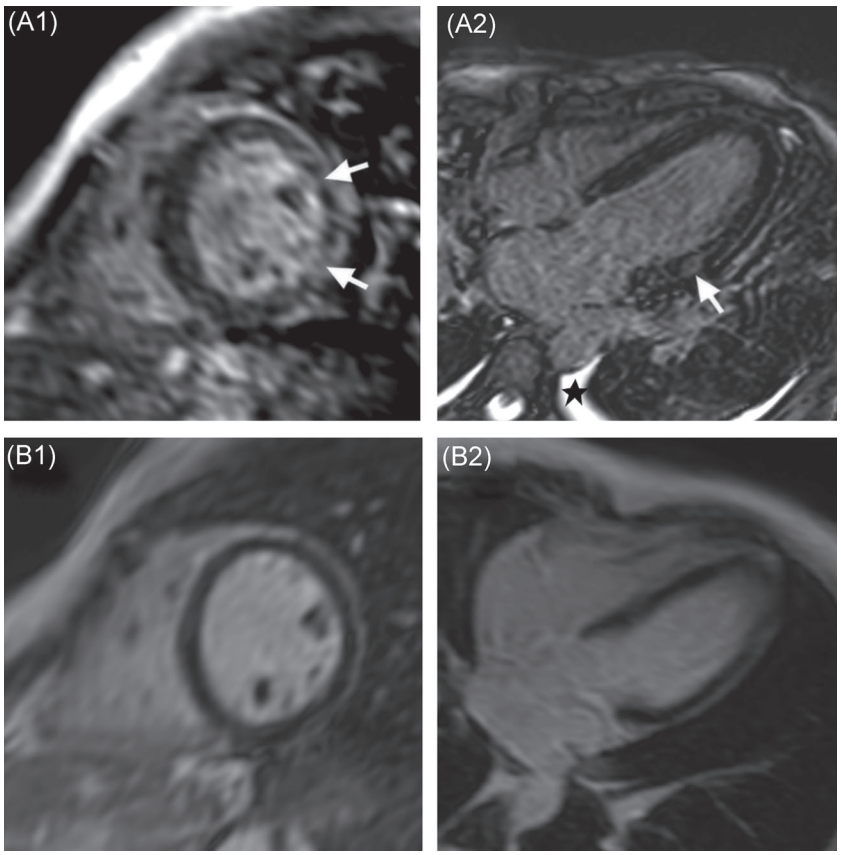

FIGURE 3 Cardiac MRI performed 4 days after VA-ECMO removal: patchy epicardial area of late enhancement (arrows) of the mid-ventricular lateral wall of the left ventricle visible on a short axis (A1) and 4-chamber (A2) view. A2 shows also a thin bilateral pleural effusion (asterisk). Cardiac MRI performed at 3-month follow-up: the myocardial lesions are no more visible in either view (B1: short axis; B2: 4-chamber view). MRI, magnetic resonance imaging; VA-ECMO, veno-arterial extracorporeal membrane oxygenation

3 | DISCUSSION

Acute myocardial injury is reported in more than 20% of COVID-19 patients and yields increased mortality.[2] The role of VA-ECMO in the management of patients with COVID-19 acute myocardial injury is still unclear and the existing literature is limited to isolated case reports.[6–8]

Although cardiac injury usually appears within the context of the overall respiratory infection rather than the first manifestation of disease,[6] our patient rapidly evolved towards a refractory cardiogenic shock successfully rescued with VA-ECMO. Interestingly, Sampaio et al.[8] published a case of COVID-19 myopericarditis complicated by tamponade and cardiac arrest and eventually managed with VA-ECMO, thus highlighting the wide and potentially life-threatening phenotypic presentation of isolated COVID-19 acute myocardial injury. Accordingly, ARDS did not complicate the clinical course of our patient neither in the initial phase nor during VA-ECMO support, as previously described.[7,8] Conversely, Tavazzi et al.[6] published the first case of biopsy-proven myocardial localization of SARS-CoV-2 viral particles presenting with cardiogenic shock rescued with VA-ECMO and then switched to veno-arterial-venous ECMO as a consequence of severe persistent hypoxemia.

In the specific setting of COVID-19-associated cardiovascular manifestations, viral myocarditis is a rare cause of acute myocardial injury and the term is used more often on the basis of increased blood troponin levels or signs of myocarditis on cardiac magnetic resonance imaging rather than on a pathological tissue diagnosis.[9] Despite a complex pathophysiology, the high potential of myocardial recovery is a key feature in COVID-19-associated acute myocardial injury and should help defining the role of VA-ECMO in the management of this subgroup of COVID-19 patients.

Acute myocardial injury may negatively affect the clinical course of COVID-19 patients. VA-ECMO support should be considered in rapidly evolving acute myocardial injury with refractory cardiogenic shock despite optimal medical treatment. Further studies are strongly recommended to evaluate the effectiveness and safety of VA-ECMO in the management of COVID-19-associated acute myocardial injury complicated by cardiogenic shock.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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