We present the first case of left ventricular (LV) rupture after myocardial infarction treated with only extracorporeal membrane oxygenation (ECMO). Magnetic resonance imaging (MRI) scans after treatment revealed good ventricular function and a healed rupture. We show for the first time the use of ECMO as a means of supporting a patient with LV rupture without surgery. Furthermore, it has the potential of stabilizing the patient preoperatively so that surgery can be performed under optimal circumstances.

Left ventricular rupture is a feared complication following myocardial infarction (MI). It occurs in approximately 1% to 4% of the patients with MI and accounts for 12% to 21% of their mortality. Diagnosis is usually clinical (hemodynamically unstable patient) and supported by transthoracic echocardiography (TTE), indicating cardiac tamponade. It is recommended that, for patients with substantial bleeding, suturing with a patch is the technique of choice. For slow leakage, the sutureless technique is preferable. Here, we report what we believe to be the first case of ventricular rupture and circulatory shock successfully supported with ECMO without performing cardiac surgery. Even after the acute phase, surgery was no longer deemed necessary.

Case
A 63-year-old male nonsmoker, with a history of hypertension and a prior posterior MI, was referred to the emergency room (ER) because of central chest pains with a duration of 4 days. Since he was stable, a subacute coronary angiography was planned. The morning after admission, his condition worsened, with progressive cardiogenic shock, syncope, inferior ST-segment elevation, and pericardial exudate (PEX) of 20 mm. The patient was then transported to Karolinska University Hospital, where an acute angiography showed an occluded circumflex coronary artery and a posterolateral branch of the right coronary artery (culprit) (Figure, panel 1). Owing to the age of the infarction and probable myocardial rupture, revascularization was not undertaken. The patient deteriorated after 1 hour and became hemodynamically unstable. TTE showed progression of the PEX measuring up to 30 mm to 40 mm, and the patient showed all the clinical signs of cardiogenic shock (Movie 1).

Figure. Coronary angiograms and magnetic resonance imaging (MRI) of the heart. Top (1) depicts A, coronary angiogram of the right coronary artery, demonstrating an occluded posterior-lateral branch; and B, left coronary artery, demonstrating occlusion of the circumflex coronary artery at the initial arrival at the hospital. White arrows denote occluded vessels. Middle (2) demonstrates A, midventricular, short-axis view of the frozen cine image through the area of myocardial rupture (thin white arrow), with myocardial thinning due to infarction (left); B, the same section, with T2-weighted images identifying edema/increased water content; and C, the same section as T1-weighted anatomic image. Images acquired 14 days following initial presentation, demonstrating the site of rupture (thin white arrow) and large amounts of blood and fluid in the pericardial sac (thick white arrow) and the rupture site in the inferolateral wall covered by an adhesive layer of fibrin clot. Bottom (3) demonstrates A, long-axis, 2-chamber, T2-weighted image, identifying the site of rupture (arrow); B, same area and rupture site (arrow) in short-axis view; and C, the infarcted/scared area (white myocardium/arrow) and rupture site in long-axis view. Images acquired following full clinical recovery, with no signs reminiscent of rupture.
To avoid the high risks of emergency surgery, it was decided to stabilize the patient by placing him on venoarterial ECMO (CentriMag Levitronix LLC) by cannulating the left femoral vessels. The vein was cannulated with a Carmeda-coated 23 French vein cannula (Medtronic Bio-Medicus) and the artery with an 18 French Fem-flex arterial cannula (Edwards Lifescience LLC). Owing to the high probability of hemodynamic collapse associated with general anesthesia, the procedure was performed under local anesthesia. The patient also received a 6 French cannula in the femoral artery to perfuse the left leg distal to the 18 French cannula.

After establishing cardiopulmonary support, lactate levels dropped, central venous pressure (CVP) decreased from 42 mm Hg to 13 mm Hg, and central venous oxygen saturation (ScvO₂) increased from 40% directly to 70% (Table 1). The patient was weaned off ECMO under general anesthesia after 8 days of cardiopulmonary support and extubated the next day without complications.

Magnetic resonance imaging (MRI) scan of the heart at day 13 after ECMO implantation showed 50% ejection fraction (EF) and a normal right ventricular function. A PEX measuring 25 mm behind the LV without significant hemodynamic effect was detected. A transmural infarction localized within the area of the circumflex artery, with a small ventricular rupture in the apical portion of the infarction, was seen (Figure, panel 2 and Movie 2). Since the pericardial fluid regressed without any operative approach, and healing of the LV lateral wall was observed, definitive surgical repair was deemed not necessary (Movie 3–5). An MRI scan 4 months after admission showed thinning of the myocardium at the site of the rupture. Compared with the previous MRI scan, there was a regression of PEX and thrombosis, as well as an improved LV function (EF 55%): all signs of healing (Figure, panel 3 and Movie 6).

**Discussion**

This case demonstrates the successful use of ECMO as a treatment of a life-threatening ventricular rupture after MI: a diagnosis that previously was perceived only to be managed by emergency surgery; however, repair 2 to 3 weeks after perforation is considered to be safer and with superior results, compared with emergency procedures, since the edges of the defect are more firm and fibrotic. Only 10% to 15% of the patients can be treated conservatively with medication and/or intra-aortic balloon pump (IABP) for a period of 2 to 4 weeks preoperatively. It clearly stands out that there is a clinical need for a procedure that stabilizes patients during the acute phase of myocardial rupture as a bridge to safer surgery or recovery.

Here we demonstrated that ECMO may be one possible stabilizing procedure. This was also shown by Anastasiadis et al. These cases demonstrate that it can be beneficial for the patient to be put on ECMO as an optimizing step bridging to surgery when the patient is more stable.

Bleeding and increased risk for thromboembolic events are the down sides of ECMO. Duration of support is therefore limited to 7 to 10 days after which the risks for the above complications increase. Because the outcome after an MI-associated ventricular rupture is very dismal, we argue that the potential benefits of ECMO as a bridge to decision or full recovery outweigh the high risks in these cases.

**Disclosures**

None.

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**Table 1. Laboratory and Clinical Parameters Before Implantation and Explantation of ECMO**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>At Admission</th>
<th>Last Value Before ECMO</th>
<th>24 Hours on ECMO</th>
<th>48 Hours on ECMO</th>
<th>Last Value on ECMO</th>
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<tbody>
<tr>
<td>ALT (U/L)</td>
<td>32.4</td>
<td>1792.8</td>
<td>9533.8</td>
<td>7955.8</td>
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<td>AST (U/L)</td>
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<td>1393.8</td>
<td>11891.8</td>
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<td>107.4</td>
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<tr>
<td>BE (mmol/L)</td>
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<td>(—)</td>
<td>−5.4</td>
<td>−2.5</td>
<td>−2.9</td>
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<tr>
<td>Creatinine (mg/dL)</td>
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<td>2.57</td>
<td>2.66</td>
<td>2.21</td>
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<tr>
<td>CRP (mg/dL)</td>
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<td>5.4</td>
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<td>14.9</td>
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<td>CVP (mmHg)</td>
<td>(—)</td>
<td>42</td>
<td>10</td>
<td>9</td>
<td>4</td>
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<tr>
<td>Hb (g/dL)</td>
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<td>10.0</td>
<td>10.2</td>
<td>9.2</td>
<td>9.8</td>
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<td>Lactate (mmol/L)</td>
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<td>1.3</td>
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<td>MAP (mmHg)</td>
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<td>ScvO₂ (%)</td>
<td>(—)</td>
<td>40</td>
<td>78</td>
<td>83</td>
<td>60</td>
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</tbody>
</table>

ECMO indicates extracorporeal membrane oxygenation; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BE, base excess; Blank spaces, data not available; CRP, C-reactive protein; CVP, central venous pressure; Hb, hemoglobin; MAP, mean arterial blood pressure; SAP, systolic arterial pressure; ScvO₂, central venous oxygen saturation.
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Disclosures
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