Alterations in Pre/Post Oxygenator Flows Due to Fibrin Deposition in the CardioHelp System—A Case Report

Tyler Wahl, MHS, CCP,* Angela Stokes, MHS, MBA, CCP,* Caleb Varner, MSP, CCP, FPP,* Burak Zeybek, MD;† Amit Bardia, MD†

*Division of Perfusion Medicine, Yale New Haven Hospital, New Haven, Connecticut; and †Department of Anesthesiology, Yale School of Medicine, New Haven, Connecticut

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Abstract: We present a 62-year-old patient with COVID-19 pneumonia on Veno-venous (VV) Extracorporeal Membrane Oxygenation (ECMO) with unique perturbations to pre and post oxygenator pressures due to fibrin deposition despite being on a Heparin/Bivalirudin infusion and activated Partial Thromboplastin Time (aPTT) within therapeutic range of 60–80 seconds. On Day 8 of ECMO support, it was noticed that flows steadily decreased despite unchanged RPMs. Unlike typical blood flow to circuit pressure relationships, the circuit pressures did not correlate with the observed decreased flow. The Delta Pressure (ΔP) was not elevated. The patient’s vitals were stable. On inspection post change-out, clots were noted in the oxygenator outlets. Oxygenator clots are usually associated with increased ΔP. In this scenario, clots in the oxygenator blocked 1 of the 4 outlets in the oxygenator causing the flow, pressures, and ΔP to drop consecutively. Due to reduced flow, the ΔP was not elevated despite extensive clots. The fibrin clot location in the CardioHelp ECMO circuit may lead to unexpected pressure and flow alterations. Sole reliance on ΔP as a marker for oxygenator clots may be misleading. Careful monitoring and timely diagnosis of coagulation status may lead to changes in anticoagulation goals and meaningfully impact patient outcomes. Keywords: ECMO, fibrin, deposition, oxygenator, pressures.

The CardioHelp System (Maquet Getinge Group, Rastatt, Germany) utilizes integrated sensors in their circuits, allowing for non-invasive circuit pressure monitoring. Standard pressure monitoring consists of measuring inlet pressure (P_{inlet}), pressure between the centrifugal head outlet and the oxygenator membrane (P_{int}), outlet pressure (P_{out}), and the difference between P_{out} and P_{int} (ΔP) (1). Monitoring these pressures are crucial as they provide an indication of a change to the flow dynamics of the ECMO machine or patient’s hemodynamic status. There is a direct correlation between the integrated pressure monitoring and blood flow rate, which yields predictable relationships that are useful when diagnosing complications (2). One key complication is thrombus formation in the oxygenator or the pump inlet/outlets. A commonly described parameter to diagnose this is to trend the ΔP, an increase in which indicates thrombus formation. We describe a unique case where sole reliance on this parameter may be misleading.

DESCRIPTION

A 62-year-old female presented with acute respiratory distress following COVID-19 pneumonia and acute hypoxemic respiratory failure. Over 6 days, the patient failed support via high-flow nasal cannula, and bi-level positive airway pressure (BiPAP) treatment before being subsequently intubated. On day 7 of admission, given the rapid decompensation, VV ECMO was initiated. Cannulation was achieved via 16 Fr FemFlex (Edwards Lifesciences) as the return cannula in the right internal jugular vein and a 25 Fr multistage (Medtronic) drainage cannula in
the right femoral vein using a 7.0 CardioHelp. Extracorporeal support was established and settled at 3,100 RPM, 3.4 LPM, $P_{\text{int}}$ 190 mmHg, $P_{\text{Art}}$ 150 mmHg, $P_{\text{Ven}}$ 50 mmHg, 4 LPM sweep, and 100% FiO2.

Anticoagulation was managed via Heparin, titrated between 850 and 1,600 units/h for a goal aPTT of 60–80 seconds. Increased Heparin demands were required on ECMO day 7 to maintain therapeutic aPTT. Early on day 8, escalating pressor and ventilator support were noted. Heparin was held at 0600 for planned tracheostomy; however, due to increasing support demands, tracheostomy was canceled and anticoagulation was restarted at 1,100 with Bivalirudin due to the development of suspected Heparin resistance. The rate was titrated from .15 to .3 mg/kg/h to maintain the desired aPTT of 60–80 seconds. Changes in ECMO flows and pressures were also seen early on ECMO day 8, summarized in Table 1.

As this event did not cleanly fall into the previously described pressure-flow relationships, possible causes for the changes were investigated. Concerns for pump malfunction or possible decoupling were considered; however, the centrifugal pump speed was manipulated with predictable results. Without clear cause for these changes, RPMs were increased to re-establish full support on ECMO day 9. Of note, the patient did not have any major fluid loss and the hemoglobin levels did not drop during the 24-hour period.

Over the following days, the patient continued to decompensate requiring increased inotrope support for decreased right heart function in addition to continually decreasing oxygenation despite maximum ECMO/Ventilator support. On ECMO day 12, the patient was terminated from ECMO. Upon examination of the CardioHelp disposable, fibrin and clot were noted in the flutes of the centrifugal pump. These formations fall distal the $P_{\text{Ven}}$ transducer but proximal to the $P_{\text{int}}/P_{\text{Art}}$ transducers, see Figure 1. A reduction in flow at this junction would yield the changes in flow and pressure noted in Table 1.

**Table 1.** Temporal changes in the Pressure Flow dynamics of the VV ECMO: A decrease in flow can be seen with constant rpms and no increase in $P_{\text{int}}$. This was probably due to outlet flow obstruction by thrombus formation as described in the case scenario.

<table>
<thead>
<tr>
<th>Time (POD)</th>
<th>RPM</th>
<th>Flow</th>
<th>$P_{\text{ven}}$</th>
<th>$P_{\text{int}}$</th>
<th>$P_{\text{Art}}$</th>
<th>$\Delta P$</th>
</tr>
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<tbody>
<tr>
<td>0000 (POD 8)</td>
<td>3,100</td>
<td>3.25</td>
<td>-52</td>
<td>191</td>
<td>171</td>
<td>20</td>
</tr>
<tr>
<td>0400 (POD 8)</td>
<td>3,100</td>
<td>3.08</td>
<td>-48</td>
<td>175</td>
<td>154</td>
<td>21</td>
</tr>
<tr>
<td>0800 (POD 8)</td>
<td>3,100</td>
<td>3.09</td>
<td>-48</td>
<td>180</td>
<td>159</td>
<td>21</td>
</tr>
<tr>
<td>1200 (POD 8)</td>
<td>3,100</td>
<td>2.4</td>
<td>-32</td>
<td>173</td>
<td>110</td>
<td>15</td>
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<tr>
<td>0000 (POD 9)</td>
<td>3,500</td>
<td>3.34</td>
<td>-41</td>
<td>218</td>
<td>194</td>
<td>24</td>
</tr>
</tbody>
</table>

POD, postoperative day; RPM, rotations per minute; $P_{\text{ven}}$, pressure in the venous arm; $P_{\text{int}}$, pressure at the outlet; $P_{\text{Art}}$, $P_{\text{int}}$ is measured between the centrifugal head outlet and the oxygenator membrane; $\Delta P$, pressure difference between $P_{\text{Art}}$ and $P_{\text{int}}$.

**Figure 1.** CardioHelp Centrifugal Head Clot formation.

**COMMENT**

The CardioHelp consists of a centrifugal head, oxygenator, and heat exchanger integrated into a single disposable unit. $P_{\text{int}}$ is measured between the centrifugal head outlet and the oxygenator membrane. While $P_{\text{Art}}$ is measured at the outlet of the oxygenator before it is returned to the patient. The difference between these two is represented as $\Delta P$, or pressure drop across the membrane. This calculation is the first indication that the membrane may be developing an obstruction such as a clot. This presents as an increase in $P_{\text{int}}$ and decrease in $P_{\text{Art}}$ because the blockage in the membrane prevents blood flow. If the $\Delta P$ stays the same but $P_{\text{int}}$ and $P_{\text{Art}}$ increase or decrease this indicates a change between the membrane and the patient or with the patient’s hemodynamics. A clot in the arterial cannula, arterial line, or connector may cause an increase in both pressures with a result in lower blood flow. Low flow may also correlate with an increase in the MAP as the circuit flow has increased afterload to push against. The consistent RPM setting allows the blood flow to fluctuate up or down with the decreasing or increasing afterload respectfully (3).

This blood flow to circuit pressure relationship typically serves as a reliable representation of overall circuit and patient status. However, the perturbations in pressure and flow described in this case serves as a highly clinically relevant “teaching point.” The increase in blood flow and $P_{\text{int}}/P_{\text{Art}}$ pressures, combined with decreasing $P_{\text{ven}}$ pressure, is usually seen with a decrease in the RPMs. However, as the RPMs were not manipulated, we suspected decoupling of the centrifugal head or malfunction in the console itself. This would be unlikely because no alarms were present alerting pump failure which typically results in cessation of blood flow.
Fibrin and clot burden on ECMO circuits and oxygenators are unavoidable. With other ECMO consoles such as the Centrimag (Thoratec Corp, Pleasanton, CA) console, the oxygenator and centrifugal head are separate components. This allows thrombus build-up to be seen pre and post oxygenator as well as on the centrifugal head. The CardioHelp console combines the oxygenator and centrifugal head for a compact design. The limitation of this design is the inability to visualize clot formation pre-membrane or on the centrifugal head without detaching the unit from the console. Additionally, the design of the centrifugal head is different than other commonly available devices. Typical centrifugal heads have one large inlet and one large outlet. The CardioHelp’s centrifugal head has one large inlet and four small outlets which has the benefit of evenly distributing the blood flow across the membrane; however, the disadvantage of this design is the fibrin buildup on the smaller diameter outlets (Figure 2).

Clinical scenarios such as planned procedures and bleeding often necessitate holding anticoagulation in patients on ECMO support (4). Surveillance of oxygenators during ongoing ECMO therapy to predict clot formation to avoid critical situations such as circuit failure and thrombus embolization is thus pertinent (5,6). Although monitoring circuit pressures may serve as a guide to ongoing hemodynamic changes, they cannot be solely relied upon since they may be misleading as seen in this case scenario. A judicious multidisciplinary approach incorporating all the elements of patient’s clinical status, circuit pressures and visual examination of the circuit is recommended for optimal patient outcomes.

REFERENCES