Extracorporeal Membrane Oxygenation Used in a Massive Lung Bleeding Following Pulmonary Endarterectomy

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Abstract: The authors present a case of massive lung bleeding following pulmonary endarterectomy (PEA) that was treated with peripheral veno-venous extracorporeal membrane oxygenation (VV ECMO). The patient repeatedly underwent bronchoscopy for airway blood clot obstruction and finally was successfully weaned off the support. The authors discuss the indications for ECMO in treatment of the most serious complications following PEA, and emphasize the importance of echocardiographic evaluation of the right ventricular function in relation to the indicated type of extracorporeal support. Anticoagulation strategy for patients shortly after the major surgery connected to ECMO is also discussed.

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Introduction
Pulmonary endarterectomy (PEA), a method of choice for treatment of selected patients with chronic thromboembolic pulmonary hypertension (CTEPH), is a surgical procedure with high frequency of postoperative complications. Although morbidity reaches up to 49%, mortality in many centres is less than 5% (Mayer et al., 2011). Massive lung haemorrhage following PEA belongs to infrequent but life threatening complications. Apart from several conservative methods (Manecke et al., 2004), the use of central veno-arterial ECMO (VA ECMO) in a patient with massive lung bleeding and the right ventricle (RV) failure was also reported (Pretorius et al., 2009). We hereby report the use of less invasive VV ECMO in a patient with preserved RV function.

Case report
50-years-old severely dyspnoic lady with CTEPH following repeated pulmonary embolism was indicated to PEA. Her medical history was remarkable for thyroidectomy followed by pulmonary embolism and splenectomy for hereditary haemoglobinopathy. Preoperative echocardiography showed D-shaped left ventricle (LV) with good systolic function and dilated RV with preserved contractility. Her preoperative pulmonary artery pressure (PAP) was 90/38/53 mm Hg. She was chronically cyanotic with SpO₂ less than 90% on room air. After receiving O₂ mask a standard invasive monitoring was secured (femoral artery, central venous and Swan-Ganz catheters into the right internal jugular vein). Measurements post induction to general anaesthesia showed PAP of 65/54/40 mm Hg, cardiac index (CI) of 1.6 l/min/m² and pulmonary vascular resistance (PVR) 557 dyn/s/cm⁵.

The PEA was performed by dissecting in the medial layer of the main pulmonary arterial tree to the subsegmental level. The procedure required two periods of a deep hypothermic circulatory arrest lasting for 22 and 18 minutes, respectively. When mechanical ventilation and pulmonary blood flow were established, large amounts of fresh blood appeared in the endotracheal tube. Total amount of suctioned blood was 950 ml. The endotracheal tube was replaced with left-sided double lumen endobronchial tube aiming for selective lung ventilation. Fibreoptic bronchoscopy revealed blood coming mainly from the right lower lobe.

Cardiopulmonary bypass was quickly disconnected and blood products (fresh frozen plasma, platelets, plasma factor concentrate – Prothromplex Total, Baxter AG, Wien, Austria) together with protamin, in dose 21,000 IU, were given. Measured activated clotting time (ACT) was 118. The bleeding diminished, however, arterial hyposaturation SpO₂ 84–86%, on FiO₂ 1.0 and hypercarbia PaCO₂ 51.6 mm Hg occurred, thought the patient was hemodynamically stable with significant decrease of PAP (26/16/14 mm Hg), compared with preoperative values, measured by pulmonary catheter. Transoesophageal echocardiography (TEE) showed dilated RV with function still preserved and good LV function. Therefore, decision was made to start peripheral VV ECMO. At the end of surgery the patient was

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hemodynamically stable with minimal blood loses from the chest tube. However, stepwise deepening of hypoxemia $\text{SpO}_2$ 79% on $\text{FiO}_2$ 1.0 and hypercarbia $\text{PaCO}_2$ 53.1 mm Hg caused gradual increase of $\text{PAP}$ and hemodynamic parameters before leaving the operating theatre was as follows: $\text{PAP}$ 56/40/32 mm Hg, $\text{CI}$ 2.6 l/min/m$^2$, $\text{PVR}$ 167 dyn/s/cm$^5$.

The patient was connected to VV ECMO 1 hour after the admission to ICU whenever continuous worsening of the gas exchange was obvious ($\text{SpO}_2$ 70%, $\text{FiO}_2$ 1.0). Drainage cannula 24 F inserted via Seldinger technique to the femoral vein as well as return cannula 20 F (both Duraflo Edwards Lifesciences, Irviene, CA, USA) in the right internal jugular vein were connected with oxygenator Maquet PLS. The closed circuit was driven by centrifugal pump Maquet Rotaflow RF 32 (Maquet Cardiopulmonary AG, Hirrlingen, Germany). ECMO blood flow was set to 5 l/min, $\text{FiO}_2$ 0.9 and heparin infusion was commenced after 12 hours to achieve $\text{ACT}$ between 160–180 s and $\text{aPTT}$ of around 50–60 s. Blood loses from the chest wound drains were 250 ml during the first 24 postoperative hours.

Beginning of the ECMO therapy led to immediate improvement of oxygenation parameters. Thus the aggressive ventilator management was tapered to a “soft” BiLEVEL mode (PIP/PEEP 18/8 cm H$_2$O with $\text{Vt}$ less than 6 ml/kg of body weight, $\text{FiO}_2$ 0.4). Biluminal endotracheal tube was changed for single lumen 4 hours after admission to the ICU. Before commencing heparin infusion, the patient underwent bronchoscopy to remove blood clots totally obstructing the right upper lobe. The remaining clots with mildly oozing fresh blood were left in right lower lobe. Antifibrinolytics and diluted epinephrine were used locally to stop the bleeding. The left sided bronchial tree contained significant spill-over of clots which were removed with careful suctioning and lavage. The patient underwent another bronchoscopy for final removal of blood clots, particularly obstructing lower and middle lobes of the right lung and left lower lobe later. After the procedure no signs of bleeding were observed in the airways.

The patient was finally weaned off the VV ECMO on the 4$^{th}$ postoperative day when the support flow was 5 l/min and $\text{FiO}_2$ 0.21. By that time, the patient was set on BiLEVEL mode with PIP/PEEP 26/14 cm H$_2$O, $\text{PSV}$ 12 cm H$_2$O, $\text{FiO}_2$ 0.5. $\text{SpO}_2$ was 98–100% and $\text{PAP}$ 38/19/26 mm Hg. The patient was extubated on the 6$^{th}$ postoperative day and spent totally 12 days in ICU. Finally she was discharged form hospital after 25 days.

**Discussion**

It seems that extracorporeal support may be helpful in treatment of the most serious complications following PEA. Both VA ECMO in the RV failure, residual pulmonary hypertension, pulmonary oedema or massive lung bleeding, and VV ECMO in late reperfusion oedema were successfully used in these life threatening situations (Ogino et al., 2006; Thistlethwaite et al., 2006; Berman et al., 2008).

Extracorporeal Support in Peroperative Lung Bleeding
PEA patients, in terms of the RV function, are an inhomogeneous group. Some of them undergo surgery with severely depressed RV function; however the others have it still well preserved. This variety emphasizes the importance of perioperative TEE in situations when any inotropic or mechanical support is considered and massive lung bleeding is one of such situation. Pulmonary haemorrhage can result either from reperfusion injury in which capillary permeability is increased or from surgical trauma to the pulmonary artery adventitia (Pretorius et al., 2009). Whenever the control of haemorrhage by selective ventilation and bronchial blockade results in deterioration of the gas exchange, one should consider the indication to ECMO. In case of failing RV the central VA ECMO through open chest seems to be very reasonable option and should be introduced without delay (Pretorius et al., 2009). Thought it increases risk of bleeding complications (Berman et al., 2008). However, in the patients with good RV function the decision on ECMO modality is a matter of hours rather than minutes. In these patients, who are already used to tolerate hypoxemia, mildly delayed institution of peripheral VV ECMO may enable control of wound bleeding and closing of the chest without necessity for its later reopening.

Even though heparin infusion targeted at ACT of 160–180 s and aPTT of 50–60 s is recommended in all ECMO settings by the manufacturers from the very beginning of the support, the ECMO running without heparin for limited period of time may be acceptable in patients following surgery in order to decrease risk of wound bleeding (Berman et al., 2008). Additionally, it was even more desirable in our patient with lung bleeding.

References