

## CASE REPORT

# Anaesthesia Management with Deep Hypothermic Circulatory Arrests During Pulmonary Thromboendarterectomy

Roberts Leibuss\*, \*\*, Martins Kalejs\*, Agnese Ozolina\*,\*\*, Andris Skride\*, Peteris Stradins \*,\*\*, Eva Strike\*\*, Romans Lacis \*,\*\*

\* Pauls Stradins Clinical University Hospital, Latvian Cardiology Centre, Riga, Latvia

\*\*Riga Stradins University, Riga, Latvia

## Summary

Chronic thromboembolic pulmonary hypertension (CTEPH) occurs in a minority of patients after acute embolism and belongs to Orphan diseases. There is no specific medical treatment currently approved. Pulmonary thromboendarterectomy (PTEAE) remains as the main and curative treatment for the CTEPH. Case presentation in a patient with acquired CTEPH is a rare condition that can be treated successfully with PTEAE under cardiopulmonary bypass (CPB) with deep hypothermic circulatory arrests.

**Key words:** *General Anaesthesia, Cardiopulmonary bypass, Deep hypothermic circulatory arrest, Pulmonary thromboendarterectomy, Chronic pulmonary hypertension*

## AIM OF THE DEMONSTRATION

CTEPH is associated with significant morbidity and mortality (7). The incidence of CTEPH is estimated 2 – 4 % among patients presenting with acute pulmonary embolism (1, 8). Three-year mortality is reported as high as 90% in patients with pulmonary artery pressure (PAP) > 50 mm Hg (3, 7). Surgical treatment is the main therapy for CTEPH and PTEAE is the surgical procedure of choice (5, 8). During PTEAE proximal fibrous pulmonary artery obstruction is removed in mechanical way (2, 4). The aim is to demonstrate anaesthesia management with deep hypothermic circulatory arrests in the first case of PTEAE in Latvia.

## CASE REPORT

J.S 31 years old male was admitted in the Pauls Stradins Clinical University Hospital, Latvian Cardiology Centre on 7th November, 2010. Because of high PAP heart catheterization was performed where medium pressure in the right atrium was 13 mmHg, right ventricle systolic pressure 107 mmHg and diastolic pressure 18 mmHg. Pulmonary pressures were as following: pulmonary artery systolic pressure 102 mmHg and diastolic pressure 42 mmHg, with mean PAP 64 mmHg. The pulmonary capillary wedge pressure (PCWP) was 10 mmHg. The following conclusion was made - severe pulmonary hypertension (PH) with normal PCWP, high pulmonary vascular resistance (PVR) (11 Wood units), normal cardiac output (5 l/min) and cardiac index (2,67 l/min/m<sup>2</sup>). Vasoreactivity test with adenosine was negative. The patient was discharged on the 14th day in a satisfactory condition with recommendations to continue Warfarin therapy, maintaining International Normalized Ratio (INR) 2-3. For PH treatment Sildenafil 20 mg three times per day was prescribed. The patient was readmitted to the hospital on 23th June, 2013 due to increasing shortness of breath on exertion.

**The main diagnosis:** CTEPH III functional class. Heart failure NYHA III functional class.

**Objective assessment:** Dilated right parts of the heart and cardiac overload, insufficiency of tricuspidal valve (III degree) and mitral valve (I degree) were visualized in transthoracic echocardiography (24th June, 2013). Right ventricle systolic pressure was 120 mmHg. Ejection fraction 60% with appropriate diastolic function. During computer angiography (24th June, 2013) severe PH, dilated right parts of the heart and right ventricle hypertrophy was found. Moreover, partial occlusion with thrombus of the right and left pulmonary arteries in parallel with bilateral embolic pneumonia was discovered.

**Perioperative risk assessment:** American Society of Anesthesiologist physical status classification system before surgery (ASA) class IV, European System for Cardiac Operative Risk Evaluation (EuroSCORE) 9,51 %, Acute Physiology and Chronic Health Evaluation II (APACHE II) score 3,3 %.

## Anaesthesia management

General anaesthesia with deep hypothermic circulatory arrests under CPB for a safe surgical approach was provided. Perioperative monitoring included: invasive arterial and central venous pressures, measurements of continuous cardiac output (CCO), pulmonary and systemic vascular resistances (PVR, SVR) and mixed venous oxygen saturation (SvO<sub>2</sub>). Arterial blood gas analysis was performed every 10 - 30 minutes to estimate glucose, lactate, sodium and potassium levels as well as to provide mild hypocapnic ventilation. Bilateral constant measurements of regional cerebral oxygen saturation (SrO<sub>2</sub>) was provided. Urine output in ml per hour and the difference of core body temperature (Celsius, °C) was measured in the bladder and in the oesophagus. Transesophageal echocardiography was performed to evaluate anatomical structures of the heart and blood flow in aorta.

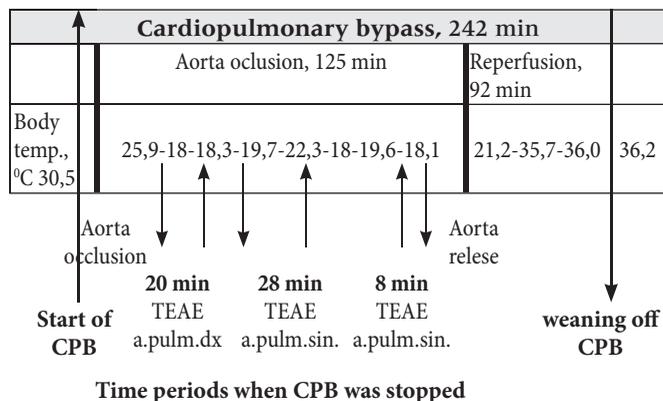
Anaesthesia was induced with ketamine 50 mg, fentanyl 0.2 mg and etomidate 20 mg intravenously (i.v.). Cisatracurium 16 mg was used for muscle relaxation. Mechanical lung ventilation was provided in pressure regulated volume control mode. Anaesthesia was maintained with sevoflurane administered at MAC 0.8-1.0. During CPB, anaesthesia was maintained with fentanyl 0.05 µg/kg/min, propofol 75 µg/kg/min and cisatracurium 1 µg/kg/min i.v. Standard pulsatile CPB with an extracorporeal circuit consisting of a polypropylene membrane oxygenator with deep hypothermia (rectal temp. 18 °C) was used. Before the start of CPB, heparin was administered in a dose of 25 000 units to achieve and maintain activated coagulation time above 480 seconds during CPB. After cannulation, CPB was started and during induced ventricular fibrillation, cross clamping of aorta was performed. During the surgery normovolemic haemodilution was maintained. Myocardial protection was achieved by antegrade cardioplegia filling coronary arteries with 600 ml, repeatedly 400 ml of Custodiol 8 °C solution. Total CPB time was 242 minutes, aorta occlusion - 125 minutes and reperfusion - 92 minutes. Circulatory arrests in deep hypothermia were performed temporarily for three times as depicted Figure 1.



**Fig. 1. Cardiopulmonary bypass with deep hypothermic circulatory arrests performed for three times**

Much attention was applied to provide cerebral protection during the surgery. Therefore deep hypothermia, local ice applications on the head, trendelenburg position, mild hypocapnia (arterial blood pCO<sub>2</sub> 29-32 mmHg) and haemoglobin around 9 g/L was maintained. To reduce cerebral blood flow, consequently cerebral oxygen consumption Thiopental 500 mg x 3 i.v. was administered each time before circulatory arrests. After aorta cross clamping and before reperfusion Solu-Medrol 250 mg i.v. was given to decrease systemic inflammatory response which can aggravate ischemia-reperfusion injury. Additionally, for reducing cerebral edema Mannitol solution 15% - 250 ml was added in CPB system during the reperfusion. Cerebral protection

was monitored with cerebral oximeter INVOS monitoring SrO<sub>2</sub>, which has to be equal on the left and right sides. The lowest acceptable limit for SrO<sub>2</sub> is 40% (Figure 2). Before starting CPB SrO<sub>2</sub> was equal on both sides, right - 69% and left side 70%, respectively. The lowest values of SrO<sub>2</sub> during the first circulatory arrest were 38% and 44%, while in the second circulatory arrest - 30% and 28%. At those moments circulation shortly was restored until SrO<sub>2</sub> returned to baseline. In the third circulatory arrest SrO<sub>2</sub> values were in the normal ranges - 70% and 68%. After all circulatory arrests were performed temporarily for three times as depicted in Figure 2. When weaning of CPB, values of SrO<sub>2</sub> were in normal range 74% and 71% on the right and left side, respectively.



**Time periods when CPB was stopped**

**Fig. 2. INVOS cerebral oximetry monitoring during deep hypothermic circulatory arrests**

Measurements of lactate, glucose, potassium and sodium levels are essential to evaluate tissue perfusion and acidosis. In the present case the maximum serum lactate level was 5,0 mmol/L during the reperfusion period. In parallel, acidosis is accelerated in the presence of hyperglycemia which compounds ischemic cerebral injury, therefore glucose level was maintained below 10 mmol/L. Weaning off CPB after the surgical procedure was performed after rewarming the patient to a rectal temperature of at least 36 °C. After separation from CPB, protamine in a dose of 1 mg per 100 units of heparin was administered. Total time of surgery was 480 minutes.

**Surgical treatment**

PTEAE was performed in standard surgical fashion via median sternotomy. Dissecting was done within the superficial media of the vessel wall, freeing the inner part of the vessel containing thrombi and scar tissue. The right pulmonary artery was exposed between aorta and the vena cava superior, an incision was made starting medially and extending beneath the mobilized vena cava superior, but not extending outside the pericardium. During the first episode of circulatory arrest for 20 minutes TEAE from the right pulmonary artery and its branches was performed. Then, for a short period circulation was resumed and right pulmonary

arteriotomy closed with a running suture. During second episode of circulatory arrest of 28 minutes plus 8 minutes TEAE for the left pulmonary artery was performed. Thrombus removed from right and left pulmonary arteries are presented in Figure 3. After surgery patient was admitted in the Intensive Care Unit of Cardiac Surgery for 3 days with successful recovery.



**Fig. 3. Thrombus removed from left and right pulmonary arteries**

#### DISCUSSION

The greatest challenge of general anaesthesia during CTEAE is to provide deep hypothermic circulatory arrest management, which can lead to tissue hypoxia (7, 9).. During circulatory arrest in deep hypothermia the cerebral metabolic rate is related exponentially to brain temperature, significantly decreases by about 50% for each 6°C drop in brain temperature, as latter helps to reduce anaerobic glycolysis and accompanying acidosis. Therefore deep hypothermia is essential during PTEAE as it prolongs brain tolerance to ischemia up to 45 minutes (9). In the presented case pharmacological and non-pharmacological methods for the brain protection were used. Additionally, low-flow CPB in time periods between circulatory arrests was applied to augment brain protection. Pharmacological protection was achieved by using barbiturates, propofol, steroids and Mannitol. Although, steroids might lead to an alteration in glucose metabolism, their role to decrease proinflammatory cytokines, which are thought to play a role in brain ischemic injury, is pivotal. Disadvantages of deep hypothermic circulatory arrests were prolonged CPB time, that increase the risk of coagulopathy and reperfusion syndrome with edema formation. Therefore, relative hypovolemia was maintained. The worse complication is neurological injury, with incidence of 3-12%, which significantly affects patient outcome (6, 10). In our case cognitive disorders and neurological deficiency was not observed postoperatively suggesting of good cerebral protection during anaesthesia. The cooling was done gradually, long enough to achieve homogenous cooling of all organs. Ice packing on the head enhances cerebral hypothermia via conduction across the skull. Moreover, it also helped to prevent

an undesirable rewarming of the brain during surgery. Rewarming phase was longer than the cooling to avoid brain hyperthermia. Rewarming was stopped when core body temperature reached 36 °C.

#### CONCLUSIONS

Our first experience providing general anaesthesia for PTEAE with deep hypothermic circulatory arrests was relatively safe without cognitive disorders and hypoxemia related complications in perioperative period. Moreover, it provided good conditions for full extent surgical treatment.

**Conflict of interest:** None

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**Address:**

Roberts Leibuss  
Department of Anaesthesiology and Cardiac surgery,  
Pauls Stradins Clinical University Hospital,  
Pilsonu street 13, Riga, Latvia  
LV-1002  
E-mail: roberts.leibuss@gmail.com

