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CASE REPORT

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Severe re-expansion pulmonary edema after conventional cardiac surgery: Identification and management

Panos N. Vardas MD[1] | Caleb Matthews MD[2] | Carlo Maria Rosati MD[3] | Daniel J. Beckman MD[2]

[1]Division of Cardiac Surgery, Bluhm Cardiovascular Institute, Northwestern University, Chicago, Illinois

[2]Division of Thoracic and Cardiovascular Surgery, Indiana University School of Medicine, Indianapolis, Indiana

[3]Department of Cardiac Surgery, University of Michigan, Ann Arbor, Michigan

Correspondence
Panos N. Vardas, MD, 201 E. Huron St, Galter 11-140, Chicago 60611, IL.
Email: panayotis.vardas@nm.org

Abstract

Re-expansion Pulmonary Edema (REPE) is a recognized but rare complication of lung re-inflation after pathologic collapse or intentional deflation. The presentation of REPE may be highly variable, ranging from a clinically asymptomatic, incidental radiologic finding to acute respiratory failure accompanied by severe, life-threatening hypoxemia. With the current report, we present a patient with severe aortic insufficiency, severe mitral regurgitation, coronary artery disease, pulmonary hypertension, who underwent aortic valve replacement, mitral valvuloplasty, coronary artery bypass grafting, and developed at the immediate post- operative period severe respiratory failure due to REPE, requiring venous-venous Extracorporeal Membrane Oxygenation (VV-ECMO).

1 | PATIENT PROFILE

A 62-year-old male with a history of congestive heart failure (New York Heart Association class IV) presented with shortness of breath and exercise intolerance due to severe aortic and mitral insufficiency, coronary artery disease, and pulmonary hypertension. He had a 6-month history of recurrent bilateral pleural effusions. Additionally, his past medical history was significant for hyperlipidemia, hypertension, gastroesophageal reflux disease, transient ischemic attack and reported a significant smoking history having quit 2 years before presentation. Preoperative transesophageal echocardiography (TEE) indicated right coronary territory wall motion abnormalities, severe aortic insufficiency with eccentric jet, mild tricuspid regurgitation, severe mitral regurgitation with central jet and poor coaptation due to annular dilation and normal leaflets (Carpentier type I mitral valve dysfunction). Pulmonary hypertension and depressed ejection fraction (50%) were also noted. A left heart catheterization showed a mid-right coronary lesion at 70% stenosis. A right heart catheterization revealed elevated pulmonary artery pressures (PAP) (64/25 mm Hg) and elevated pulmonary capillary wedge mean pressure of 27 mm Hg. The patient deteriorated on guideline-directed medical therapy. Although high risk, double valve and coronary artery bypass surgery were offered to the patient and he consented.

An intraoperative TEE confirmed the preoperative findings. A diastolic arrest was achieved with augmented retrograde blood cardioplegia given at 10-minute intervals. The right coronary artery was bypassed with reversed saphenous vein graft. Exposure of the mitral valve through the interatrial grove permitted mitral valve repair with a 31 mm Duran AnCore annuloplasty ring (Medtronic, St Paul, MN). Aortic valve replacement used a 23 mm Inspiris® pericardial tissue valve (Edwards Life Science, Irvine, CA). Before separating from cardiopulmonary bypass, 3 L of serous pleural effusion was drained from the pleural spaces. Cessation of cardiopulmonary bypass was achieved with adequate oxygenation and ventilation on inotropic and vasopressor support with milrinone (0.375 mcg/kg/minutes), norepinephrine (6 mcg/min), and vasopressin (0.04 units/minutes).

The patient received 1000 mL of crystalloids, 500 mL of colloids, and 350 mL of cell-saver blood. Excellent urine output ensued, and no blood products were administered. Postoperative PAP was 35/16 mm Hg. While preparing to transport the patient to the critical care unit, oxygen desaturation occurred associated with frothy fluid in the endotracheal tubing. Management included adjustments in ventilatory

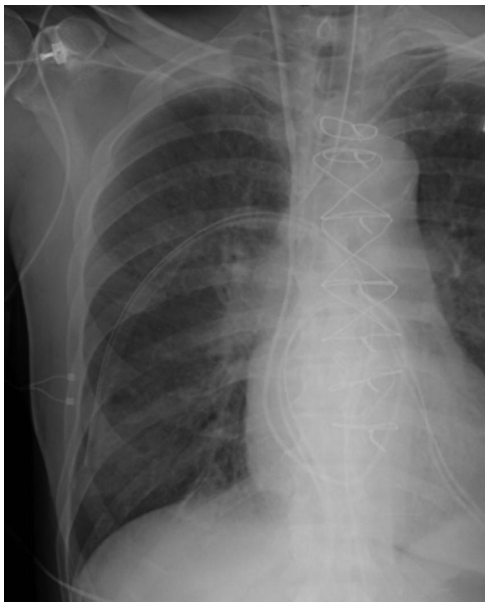

FIGURE 1 Shows the initial intraoperative chest X-ray after encountering difficulty with oxygenation and following

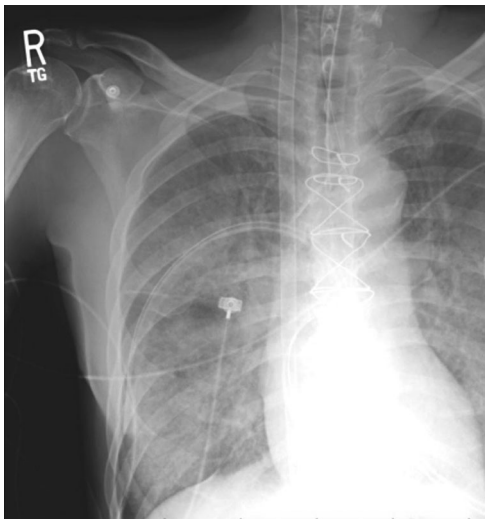

FIGURE 2 shows the degree of pulmonary edema accrued after only 2 to 4 hours following the initial postoperative chest-XR. Note the Avalon Elite Bi-Caval Catheter extending to the right atrium

support, diuretics, and a bronchoscopy was performed revealing normal airways but a significant amount of serous fluid. Intraoperative TEE revealed normal aortic prosthesis function, no residual mitral valve regurgitation, no tricuspid regurgitation and confirmed no wall motion abnormalities and the pre-operative ejection fraction (~50%). An intraoperative chest radiograph showed only expected post-operative findings (see Figure 1). His condition precipitously deteriorated on transfer to the intensive care unit. Severe pulmonary edema developed with voluminous clear, frothy fluid present in the endotracheal tubing. Pulmonary compliance worsened (peak airway pressures exceeded 70 mm Hg), with severe hypoxemia (paO_2 was 45 mm Hg on 100% FiO_2 and on positive end-expiratory pressure (PEEP) of 12 cm H_2O). Initiation of inhaled nitric oxide and intravenous diuretics produced no improvement in oxygenation. Repeat TEE confirmed stable left and right ventricular function, normal prosthetic aortic valve performance, no mitral regurgitation, no systolic anterior motion or left ventricular outflow obstruction and a normal mitral valve gradient. Non-cardiogenic pulmonary edema of unknown etiology could not be conclusively eliminated as a possible diagnosis.

Because maximal efforts at resuscitation with conventional ventilatory support (including volume controlled and pressure controlled mechanical ventilation, increased PEEP and FiO_2) did not result in improvement, emergent Veno-Venous extracorporeal membrane oxygenation (V-V ECMO) was instituted at the bedside. An Avalon Elite Bi-Caval Catheter (Getinge, Wayne, NJ) was used placed through the right internal jugular vein, under fluoroscopic and TEE guidance (see Figure 2). V-V ECMO achieved adequate flows and oxygenation improved shortly afterwards. Metabolic derangements were corrected, and throughout the next 36 hours, inotropic agents and vasopressors were weaned. V-V ECMO was removed on postoperative day 3. On postoperative day 4 positive pressure ventilation was discontinued. On postoperative day 10, the patient was discharged from the hospital in satisfactory condition. The patient currently remains asymptomatic after 3 years of follow-up with no pulmonary sequela.

2 | DISCUSSION

Re-expansion pulmonary edema (REPE) is a rare but well-known entity. However, to our knowledge, it has not been described following conventional (sternotomy) cardiac surgery without associated blood transfusions. REPE was first reported in the mid-1800s and is most commonly seen following large volume thoracentesis.[1–3] Suggested risk factors for the development of REPE include; age between 20 and 40 years old, diabetes, duration of lung collapse, high negative pressures following drainage (more negative than - 20 cmH2O), and draining greater than 1.5 L of fluid.[4,5] Feller-Kopman et al[2] found in their retrospective review of 185 patients that REPE did not correlate with the amount of fluid withdrawn from the chest. Postoperative unilateral pulmonary edema, often subclinical, occurs in up to 25% of patients undergoing minimally invasive cardiac surgery for which one-lung ventilation is used for prolonged periods.[6] Risk factors include Chronic Obstructive Pulmonary Disease (COPD), pulmonary hypertension, right ventricular dysfunction, and increased CPB time. A report of REPE following conventional (sternotomy) coronary bypass surgery described an association with transfusion of fresh frozen plasma, which was not a factor in our case.[7] Draining pleural effusions is a common practice during cardiac surgery. Our patient's effusions were large (3 liters) and chronic (more than 4 months). In addition, the CPB time was prolonged. Systemic Inflammatory Response Syndrome (SIRS) occurring after cardiac surgery under cardiopulmonary bypass is due to surgical trauma, contact of blood with foreign materials, abnormal sear stress, ischemia-reperfusion, hypothermia, and lack of homeostasis. Severe inflammatory response to CPB was included in our differential diagnosis which in association with

ischemia-reperfusion injury (from lung collapse during CPB) may produce a similar clinical manifestation with REPE.

The pathophysiologic mechanism of REPE has been extensively studied and is likely multifactorial. Ischemia-reperfusion injury and increased vascular permeability from activation of the proinflammatory cascade (mediated by IL-8 and leukotriene B4), subsequent neutrophilic and macrophage invasion, surfactant dysfunction, and hyaline membrane formation all play roles in its pathogenesis. Sohara suggests that chronic lung collapse leads to abnormally hardened pulmonary microvessels. The mechanical stress of re-expansion and alveolar recruitment then produces REPE. Lung injury caused by ischemic-reperfusion results in free radical damage and leukocyte sequestration.[8] Compared to two-lung ventilation controls, the duration of one-lung ventilation for greater than 120 to 180 minutes shows a significant increase in the production of reactive oxidative species.[8] Lung injury in this setting is accentuated by poor perfusion secondary to shunted blood flow to the contralateral lung. It is postulated, in our case, that the combination of chronic congestive heart failure, large bilateral chronic pleural effusions leading to parenchymal collapse, followed by prolonged CPB initiated REPE. Rapid recognition of this life-threatening refractory respiratory insufficiency led to the institution of V-V ECMO, which might have intensified the severity of SIRS. Our patient's oxygenation improved on V-V ECMO and he recovered from what would have been a fatal postoperative outcome. Based on our experience, preoperative drainage of chronic large pleural effusions as a part of medical optimization for complex cardiac surgery, as well as, intraoperative periodic lung inflation/deflation during CPB should be a consideration.

CONFLICT OF INTERESTS

This case was presented at American College of Cardiology (ACC) IN Chapter Meeting in 2017 and won the first prize in case reports session.

ORCID

Panos N. Vardas http://orcid.org/0000-0002-1872-5347

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