Unilateral Pulmonary Edema after Atrial Septal Defect Repair

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Unilateral pulmonary edema is a rare complication of cardiothoracic surgery and usually is related to direct injury to the lung parenchyma,1 rapid evacuation of a pneumothorax,2 or an alteration in pulmonary blood flow.3 We report a case of acute fulminant unilateral pulmonary edema (UPE) with an unusual cause that occurred during an atrial septal defect repair.

CASE REPORT

The patient was a 20-year-old man who presented with symptoms of increasing dyspnea on exertion. He had no history of cyanosis, paroxysmal nocturnal dyspnea, orthopnea, asthma or other pulmonary disease, was taking no medications, and had no allergies.

Physical examination revealed a well-developed, well-nourished man in no acute distress. His pulse was 75 beats per min, blood pressure 110/60 mmHg, respiratory rate 14 breaths per min, and weight 60 kg. Airway examination was within normal limits; the lungs were clear bilaterally to auscultation; heart rhythm was regular; and S1 and S2 were normal with a III/VI systolic murmur heard at the apex. His electrolytes were: sodium 137, potassium 4.0, chloride 108, and carbon dioxide 21 mEq/L, and glucose 100, blood urea nitrogen 18, and creatinine 0.8 mg/dL. The hematocrit was 40%, and coagulation studies were normal. The chest x-ray showed no evidence of acute or chronic pulmonary disease and showed a normal cardiac silhouette. The electrocardiogram showed normal sinus rhythm with a right-bundle-branch pattern. Cardiac catheterization revealed a large secundum type atrial septal defect, with normal right- and left-sided pressures.

The patient received morphine sulfate 6 mg and scopolamine 0.3 mg intramuscularly 1 h prior to surgery. Monitoring included seven-lead electrocardiogram, right radial arterial catheter, right internal jugular central venous pressure catheter, pulse oximetry, end-tidal carbon dioxide, breath sounds by auscultation, Foley catheter, and rectal temperature.

Anesthesia was induced with thiopental 175 mg and sufentanil 100 µg, and tracheal intubation was facilitated with vecuronium 10 mg. Ventilation was controlled with 100% oxygen using a Siemens-Servo 900D anesthesia machine. Anesthesia was maintained with an infusion of sufentanil 2 mg·kg⁻¹·h, midazolam 20 mg·kg⁻¹·h⁻¹, and vecuronium 60 mg·kg⁻¹·h⁻¹. Arterial blood gas results after intubation were: pH 7.48, carbon dioxide tension (Paco2) 32 mmHg, and oxygen tension (PaO2) 504 mmHg. The patient received heparin 3 mg/kg.

and after bicalvial cannulation, full cardiopulmonary bypass (CPB) was instituted.

A Dacron patch closure of the atrial septal defect was performed via the right atrium under induced ventricular fibrillation. After completion of the repair, the right atrium was closed, and appropriate maneuvers to remove air from the left side of the circulation were performed. The heart was defibrillated once into normal sinus rhythm, and the patient was successfully separated from cardiopulmonary bypass. Anticoagulation was reversed with protamine sulfate. Within 5 min the hemoglobin oxygen saturation (Sao2) had decreased from 100 to 87% and arterial blood gas values were pH 7.34, Paco2 45 mmHg, and PaO2 62 mmHg.

A suction catheter passed down the endotracheal tube obtained 500 ml of pink frothy serosanguineous fluid. Visual examination of the lungs and opening of the right pleura revealed a severely congested and hyperemic right lung with a normal-appearing left lung and markedly distended right pulmonary veins. A diagnosis of obstruction of the right pulmonary veins by the septal patch was made. After anticoagulation with heparin, CPB was reinitiated, and the surgical repair of the atrial septal defect was taken down and corrected.

The patient was again successfully separated from CPB, at which time arterial blood gas values were pH 7.40, Paco2 39.9, and PaO2 100 mmHg. There was no longer any serosanguineous drainage from the endotracheal tube. Five centimeters of positive end-expiratory pressure (PEEP) was added, resulting in improvement of the patient's oxygenation.

Initial blood gas values in the intensive care unit were pH 7.41, Paco2 39 mmHg, and PaO2 365 mmHg. Fractional inspired oxygen concentration (FiO2) was 100%, tidal volume 600 ml, respiratory rate 10 breaths per min, and PEEP 5 cmH2O. The patient's trachea was extubated on the 1st postoperative day, and he was discharged home on the 8th postoperative day.

DISCUSSION

Acute pulmonary edema in the operating room has many possible etiologies. These include, among others, cardiogenic pulmonary edema of ischemic or valvar origin,4 pulmonary embolism,5 laryngeal obstruction,6 bronchial aspiration,7 naloxone use,8 acute respiratory distress syndrome,9 and post-cardiopulmonary bypass.10

UPE is a more uncommon entity. It has been shown to be a rare indicator of acute left ventricular dysfunction. Keren et al.11 reported UPE subsequent to acute myocardial infarction, and Van Meerhaeghe12 reported its association with a failing prosthetic aortic valve and aortic dissection. Kramer and Melzer13 describe its occurrence after an endobronchial intubation, and Morisaki et al.14 describe its association with acute subglottic edema after removal of an endotracheal tube.

The patient's position on the operating table also can be a contributing factor, and UPE has been reported in patients undergoing both thoracic15 and nonthoracic16

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surgery. The lateral decubitus can compromise lung mechanics and worsen a ventilation–perfusion mismatch. Although endobronchial intubation and pulmonary aspiration must be ruled out, the increased perfusion to the dependent lung with the patient in the lateral decubitus, especially in situations when volume overload occurs during prolonged procedures, can result in UPE.\(^7\)

Reexpansion unilateral pulmonary edema can occur after the rapid evacuation of a large quantity of air or fluid in the treatment of a pneumothorax or pleural effusion.\(^2\)

Systemic-to-pulmonary artery shunts have become critical interim procedures in patients with complex cyanotic heart disease. Unfortunately, UPE has been reported after most surgically created left-to-right shunts, including the Blalock-Taussig, Waterston, and Potts shunt procedures.\(^5,18\) This newly increased blood flow to this segment of the lung acutely increases the capillary hydrostatic pressure, resulting in transudation of fluid.

In the current case, the surgically created obstruction to the right pulmonary veins acutely increased the hydrostatic pressure in the entire right lung, leading to acute unilateral pulmonary edema and thereby necessitating immediate surgical correction.

In summary, our report demonstrates an unusual case of UPE caused by an acute increase in unilateral pulmonary venous pressure as a result of obstruction by the atrial patch of the pulmonary veins. Although this is certainly a rare complication of atrial septal defect repair, it describes the dynamic appearance of acute pulmonary edema and illustrates the need to correlate sudden changes in the patient’s condition with events in the surgical field, prior to the institution of therapy.

REFERENCES


