What Just Happened??!! Fulminant Pulmonary Edema Requiring ECMO Initiation in a 3 year-old Patient following Cardiopulmonary Bypass.

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Objectives:

1. **Discuss the implications of left-sided obstruction in the setting of partial AV canal defect.**
2. **Understand the differential diagnosis for fulminant pulmonary edema following cardiopulmonary bypass cessation in congenital cardiac surgery.**
3. **Demonstrate an understanding of the workup of suspected blood transfusion reactions and current American Association of Blood Banks risk mitigation strategies.**
4. **Understand the consequences of emergent VA ECMO cannulation for respiratory failure.**

Case history:
A 3 year old, 15kg male had undergone surgery for repair of a partial AV canal and subaortic stenosis approximately 2 years prior. Following this, the patient developed recurrent subaortic stenosis and severe mitral insufficiency and underwent a redo-sternotomy six months following his first sternotomy. The patient then subsequently developed severe mitral stenosis and left ventricular outflow tract obstruction. It was felt the patient would benefit from repeat surgery to relieve the obstruction.

Questions:
What is the nature of lvot obstruction following partial AVC repair? What other concurrent defects are associated with partial AVC repair? What are the implications of left sided obstructions in partial av canal repair?

Case history (cont):
A preoperative 2-D echocardiogram was performed in preparation for surgery; it revealed a severely dilated left atrium with abnormal left ventricular diastolic filling, and left ventricular outflow tract obstruction (peak gradient of 78mmHg). There was accessory AV valve tissue in the region of the LV outflow tract (lvot) and moderate LVH. The patient demonstrated severe mitral stenosis with restricted motion of the anterior leaflet. Moderate mitral regurgitation was appreciated, increased from previous study.

There was also an estimated RV systolic pressure of 68mmHg, approximately 2/3 systemic pressures.

Questions:
What are the pathophysiologic implications of left ventricular outflow tract obstruction? What are the anesthetic implications for pediatric patients with severe mitral stenosis and left ventricular outflow tract obstruction?

Intraoperative Care:
Induction and maintenance of anesthetic was uneventful with excision of excess chordal tissue at the mitral valve and pericardial patch augmentation. Intraoperative medications included fentanyl, isoflurane, and rocuronium. Cefuroxime and decadron had been given prior to bypass initiation per institution protocol. Amicar was utilized for antifibrinolysis, and one unit of ffp and one unit of packed red blood cells had been utilized during the pump run. Prior to the cessation of bypass, ventilation was commenced and intraoperative TEE was performed. There was no residual LVOT obstruction, with a minimal Doppler gradient. There was mild residual mitral valve stenosis with a Doppler gradient peak of 14mmHg. RV pressure measured by TR jet was less than half the systemic pressure. Hemodynamics appeared adequate with a continuous infusion of low-dose dopamine (5mcg/kg/min). Modified ultrafiltration (MUF) was performed. The decision was made to administer protamine, and a total of 45mg was given as a bolus. One pooled unit of platelet products, along with three donor units of cryoprecipitate was administered. Additional doses of fentanyl, rocuronium, and cefuroxime were administered. Bleeding was controlled, 150cc of salvaged blood was begun, and chest closure was commenced.
As the chest was being closed, there was noted to be a sudden decrease in ventilation, and ventilation by hand was difficult. Suctioning from the ETT produced a copius amount of pink frothy fluid, eventually totaling 400cc. SpO2 decreased to 83%.

Questions:
What is your differential diagnosis for the sudden change in ventilation and pulmonary edema? What are your immediate next steps in management of this patient?

Intraoperative Care (cont):
Repeat evaluation of TEE does not show any additional obstruction, with normal function of the aortic valve and continued good function of the mitral valve. A bubble study was performed that showed no evidence of new right-to-left shunt. ABG reveals a prominent respiratory acidosis with paCO2 of 90mmHg and paO2 as low as 46mmHg. At this time oxygen saturations are dipping as low as 65% and bilateral frontal nirs monitors are more then 30% below baseline. The patient's heart rate has increased from 120bpm to 150bpm; the systolic blood pressure has decreased from around 80 to 65mmHg, but appears responsive to fluid boluses. CVP measurement trends with the blood pressure, decreasing to a low of 6mmHg and increasing with fluid bolus.

Questions:
What is the likelihood of anaphylaxis in this patient? What are the signs and symptoms of a protamine reaction? What are the types of protamine reaction? What testing can be done to determine if a true protamine reaction has occurred? What is the management for protamine reaction?

What is TRALI? What are the clinical signs and symptoms of TRALI? When do the symptoms of TRALI appear? What is the treatment for TRALI? What blood components are implicated in TRALI? What is the incidence of TRALI? How does one test for suspected TRALI?

Intraoperative Care (cont):
The chest has been closed and a stat CXR is obtained showing bilateral infiltrates suggestive of pulmonary edema, right side worse than left. Bronchoscopy was considered, however the patient would not tolerate any further diminished ventilation. Ventilation continued mostly by hand. A phenylephrine drip was begun to provide further support of hemodynamics. It was felt that the patient would not tolerate transfer to the ICU; the decision was made to institute VA ECMO through the right neck.

Questions:
How has the use of ECMO for respiratory failure and subsequent prognosis changed over the years? What are the most common complications of ECMO?

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