Anesthetic Considerations for Class IV Peripartum Cardiomyopathy

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Introduction and Patient History

Peripartum cardiomyopathy (PPCM) is a dilated cardiomyopathy (DCM) presenting between 3rd trimester and 5 months postpartum. It is a diagnosis of exclusion.

Characterized by: <45% ejection fraction (EF) and dilated left ventricle.

Presenting symptoms: fatigue, dyspnea and leg swelling, with increased risk for arrhythmias, thromboembolic events and sudden cardiac death.

Now 43 years old, a G1P1 woman with pregestational HTN was diagnosed with PPCM 3 months after an uncomplicated vaginal delivery (2004). Worsening postpartum dyspnea had prompted a cardiac ECHO showing global DCM with 35% EF. Metoprolol, aspirin and furosemide were started. She developed tricuspid regurgitation (2005), atrial fibrillation (2010), received an automatic implantable cardioverter-defibrillator (AICD) for 15% EF (2012), and had a DVT (2015). Pulmonary hypertension (pHTN; peak systolic 47mmHg) was diagnosed in 2017, and she was placed on heart transplant waiting list and referred for left ventricular assist device (LVAD). A pre-LVAD ECHO showed 6% EF and CI on catheterization was 0.93 L/min/m^2. She was scheduled for LVAD placement and tricuspid annuloplasty under cardiopulmonary bypass (17/10/2017)..

Discussion

Long term follow-up of women with PPCM shows recovery in 50% of cases, with 25% stable on medication and 25% progressing into severe heart failure, as was the case here.

Risk factors: advanced maternal age, multiparity, **HTN**, multifetal pregnancy, and **African decent**. It occurs in 1 of 4000 live births in the US and Canada, 1 of 1000 live births in South Africa and 1 of 300 live births in Haiti.

Presenting symptoms: **fatigue**, **dyspnea** and **leg swelling** with an increased risk for **arrhythmias**, **thromboembolic events** and sudden cardiac death.

These patients benefit from digoxin to increase cardiac contractility, vasodilators to reduce afterload, diuretics to relieve fluid retention (hydralazine during pregnancy and ACE-I after pregnancy), beta blockers for arrhythmia prophylaxis and anticoagulation to prevent DVTs and PEs.

Obstetric cases: patients should be delivered with neuraxial anesthesia. The vasodilation produced by an epidural/CSE sympathectomy is beneficial in left ventricular dysfunction to prevent the increased afterload associated with labor and contractions. Conversely, prevention of severe hypotension is crucial as it could lead to complete cardiovascular compromise.

Anesthestic Management

A pre-induction radial arterial line was inserted and standard ASA monitors and defibrillator pads applied. Milronone was started before induction to minimize pulmonary hypertension. General anesthesia was induced with midazolam and Etomidate to minimize fluctuations in mean arterial pressure and afterload and maintain systemic and coronary blood flows. The endotracheal tube was secured and isofluorane was used to maintain anesthesia. A pulmonary arterial catheter was placed to monitor pulmonary artery pressure (PAP). Inhaled nitric oxide was started at 20ppm to alleviate her elevated PAP.

Cardiopulmonary bypass was started; an LVAD was successfully placed after a tricuspid annuloplasty. The patient was weaned off cardiopulmonary bypass with milrinone, epinephrine, norepinephrine and dobutamine initiated.

Crystalloid administration was minimized due to patient's dilated cardiomyopathy. The patient's chest was kept open to relieve intrathoracic pressure and worsened diastolic dysfunction. Chest closure occurred 36 hours after ICU admission and extubation at 48 hours.

She was discharged on postop day 12 on bumetanide, sildenafil, warfarin and aspirin, and is still awaiting heart transplant.

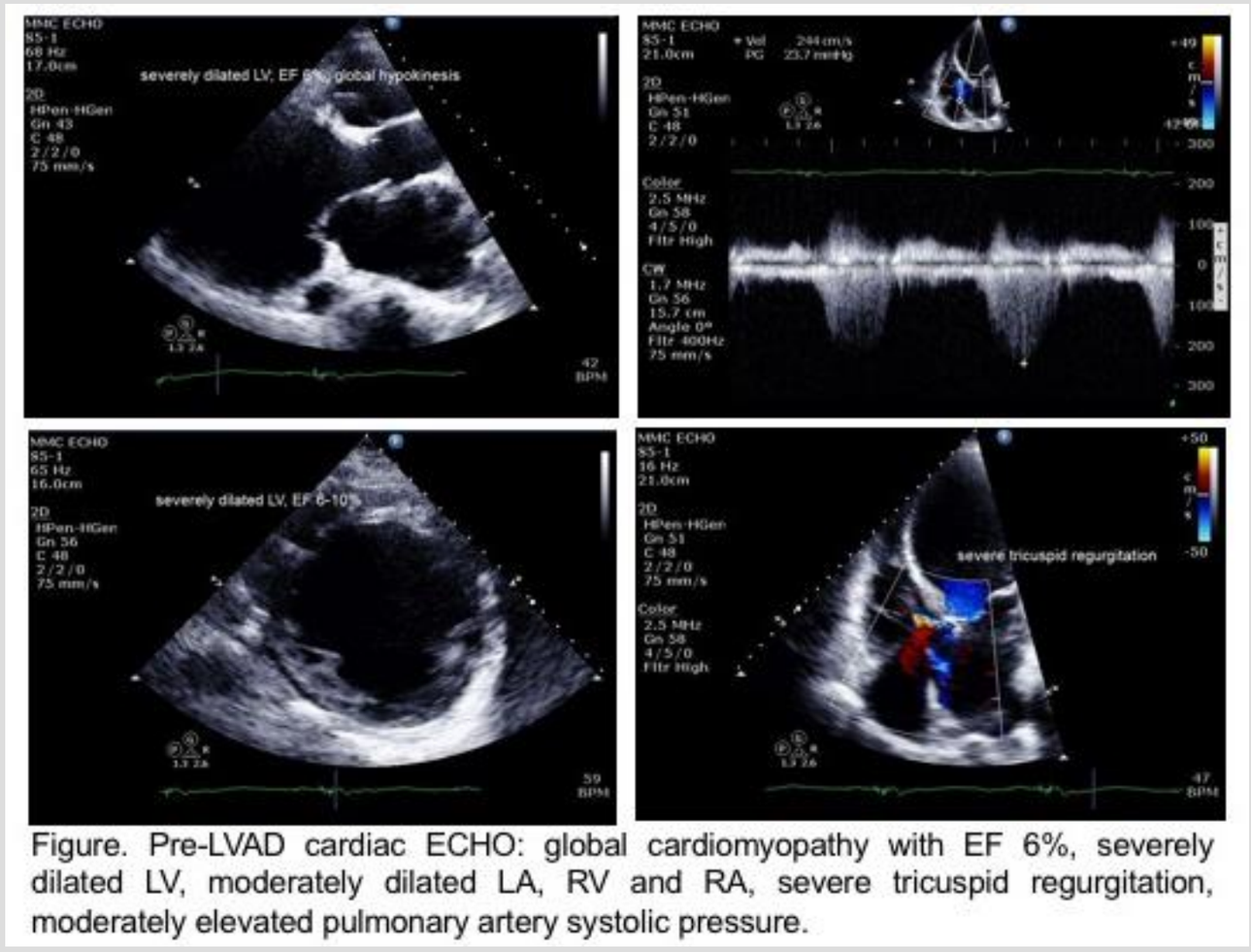

Figure. Pre-LVAD cardiac ECHO: global cardiomyopathy with EF 6%, severely dilated LV, moderately dilated LA, RV and RA, severe tricuspid regurgitation, moderately elevated pulmonary artery systolic pressure.

Non-obstetric cases: Cases requiring general anesthesia should have slow, controlled opiate inductions. A large reduction in preload will cause a fatal reduction in cardiac output and an excessive reduction in afterload will compromise coronary perfusion. Fluid management should be stringent and managed by following the patient's CVP. A pulmonary artery catheter should be placed; milronone can be used to vasodilate the pulmonary vasculature and reduce afterload on the right ventricle. Inhaled nitric oxide can be utilized to augment the same effect. Finally, use of intraop TEE is encouraged as a sensitive indicator of cardiovascular changes.

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References:
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