A Case of Peripartum Decompensated Nonischemic Cardiomyopathy with Real-Time Hemodynamic Demonstration of Postpartum Autotransfusion and Early Right Heart Failure Management

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BACKGROUND

- Pregnancy produces major cardiovascular adaptations including ↓ systemic vascular resistance and ↑ blood volume and cardiac output.
- Labor and delivery result in **autotransfusion of ~300-500 mL of blood** per contraction and additional preload immediately postpartum.
- In patients with **nonischemic cardiomyopathy (NICM)** with reduced ejection fraction, these abrupt preload changes may precipitate **acute heart failure** or **right-sided volume overload**.
- Real time invasive hemodynamic documentation of postpartum autotransfusion in high risk obstetric patients is rarely reported.

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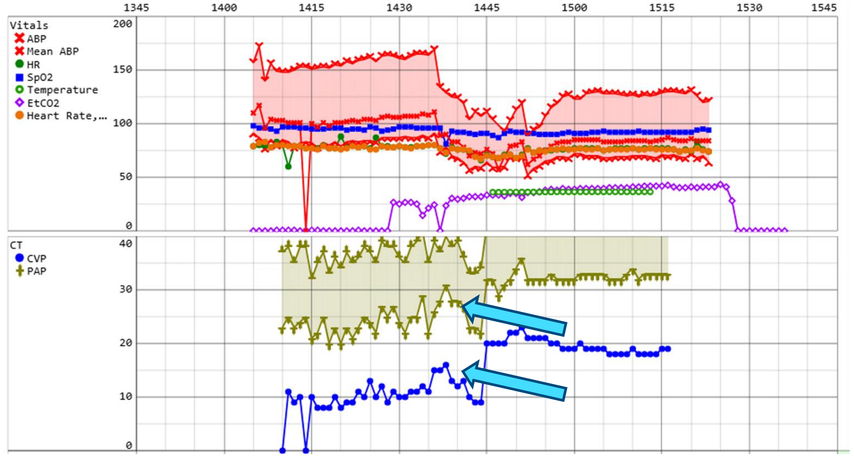
THE PATIENT

27 yo G1P0 at 27w1d with NICM (EF 26%), HTN, SVT, T2DM, and OSA admitted for severe preeclampsia and decompensated heart failure.

- Antepartum:
  - Cardiomyopathy diagnosed pre-pregnancy (EF 40-45%)
  - GDMT partially held
  - Multiple hospital visits for dyspnea and volume overload
  - Repeat echo: EF 25-30%, RSVP 44 mmHg
- Pre-delivery Evaluation:
  - BP 180-200 mmHg, BNP 2572 pg/mL, labs showing pre-e
  - Transferred to CVICU for A-line and Swanz-Ganz cath placement
  - Multidisciplinary decision for urgent C-section
- Anesthetic Plan:
  - ASA IV
  - General anesthesia d/t significant orthopnea and pulmonary edema and c/f hemodynamic collapse
  - Slow controlled induction > RSI
  - Femoral sheaths placed for potential ECMO

CASE

- Intraoperative:
  - After delivery, CVP increased 9 → 20 mmHg, PAP increased, MAP decreased, worsening oxygenation → c/w postpartum autotransfusion
  - Initiated vasopressin, increased epi → hemodynamics stabilized



- Outcome:
  - Transferred to CVICU intubated, extubated POD1, discharged hospital day 11, EF improved to 45-50% 2 months postpartum

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TEACHING POINTS

- Delivery causes abrupt preload shifts
  - Postpartum autotransfusion increases venous return and filling pressures
  - Patients with severe LV dysfunction are vulnerable to acute decompensation
- Invasive hemodynamic monitoring can guide real time management
  - Pulmonary artery catheterization detected rapid rise in CVP and PAP after delivery
  - Allowed **early recognition** of right heart strain and targeted vasoactive support
- **Anticipate** hemodynamic instability in severe cardiomyopathy
  - Prepare for vasopressors, inotropes, and advanced circulatory support
  - Consider ECMO access and multidisciplinary planning in high risk cases
- Anesthetic management must be **individualized**
  - GA with slow controlled induction used due to pulmonary edema and orthopnea
  - Avoid abrupt preload/afterload changes that may precipitate cardiovascular collapse