Heart Failure in Pregnancy

Uri Elkayam, MD
Professor of Medicine
Professor of Obstetrics and Gynecology
Director Maternal Cardiology
University of Southern California
Los Angeles, California
Pulmonary Edema Early After Delivery

TPGA after arterial switch operation
Importance of Hemodynamic Monitoring

- Hemodynamic optimization prior to delivery.
- Correct hemodynamic instability during delivery.
- Prevent and manage post delivery hemodynamic changes due to increased venous return.

- 1. relief of IVS obstruction
- 3. Transfere fluid from extra cellular space
Impella Assisted Cesarean Delivery in a 27 6/7 Weeks Gestation 30 Year Old Female with Newly Diagnosed Cardiomyopathy & Multifocal Incessant Ventricular Tachycardia

Brendan J Carry, MD, Jason Costa, MD, Alex Reyentovich, MD, Shaline Rao, MD, Stephen Pan, MD, Mara Rosner, MD, Jill Westcott, MD, Francine Hughes, MD, Dan Halpern, MD, Catherine Weinberg, MD

February 24, 1018
PPCM is an idiopathic cardiomyopathy presenting with HF secondary to LV systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of HF is found.
## Mortality in the US

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th># of Patients</th>
<th>Study Type</th>
<th>Mean F/U</th>
<th>% AA</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goland</td>
<td>2009</td>
<td>182</td>
<td>Multicenter Retrospective</td>
<td>19 m</td>
<td>29%</td>
<td>7%</td>
</tr>
<tr>
<td>Modi</td>
<td>2009</td>
<td>44</td>
<td>Single center retrospective</td>
<td>24 m</td>
<td>89%</td>
<td>16%</td>
</tr>
<tr>
<td>Gunderson</td>
<td>2011</td>
<td>110</td>
<td>Population study retrospective</td>
<td>36 m</td>
<td>29%</td>
<td>2%</td>
</tr>
<tr>
<td>Cooper</td>
<td>2012</td>
<td>39</td>
<td>Multicenter Prospective</td>
<td>25 m</td>
<td>39%</td>
<td>0%</td>
</tr>
<tr>
<td>Harper</td>
<td>2012</td>
<td>85</td>
<td>Epidemiologic Retrospective</td>
<td>7 y</td>
<td>59%</td>
<td>16%</td>
</tr>
<tr>
<td>Pillarisetti</td>
<td>2014</td>
<td>100</td>
<td>2 center Retrospective</td>
<td>33 m</td>
<td>55%</td>
<td>11%</td>
</tr>
<tr>
<td>Briasoulis</td>
<td>2015</td>
<td>47</td>
<td>Single center Retrospective</td>
<td>12 m</td>
<td>96%</td>
<td>11%</td>
</tr>
<tr>
<td>McNamara</td>
<td>2015</td>
<td>100</td>
<td>Multicenter Prospective</td>
<td>12 m</td>
<td>30%</td>
<td>4%</td>
</tr>
</tbody>
</table>
Presentation

- 30 YO woman G1 P0
- No previous medical history.
- Running half marathon.
- Presented at 26 weeks gestation with progressive exertional edema and palpitations with increasing frequency.
- No toxic habits.
Case

- Vitals: 96/82, P111, RR 25, 97% RA
- Gen: mild distress
- Neck: Supple, JVD 11-12cm
- Irregularly irregular rhythm, 3/6 systolic murmur at apex radiating to axilla
- Lungs: bilateral crackles at bases
- Abd: Soft, gravid
- Ext: Warm, trace edema
- Fetus: No significant compromise.
CXR

BNP 1680
TROP 0.012X2
Echo
Too Early for PPCM?

- We have a 30 yo F at 26 w gestation with multifocal VT, LVEF 15-20%, severe MR, BNP 1200. Improved on esmolol and lidocaine.

- Possible viral syndrome (mild) at the beginning of pregnancy, No febrile disease throughout.

- HF service recommended steroid treatment.

- Since 26 weeks is kind of early for PPCM I wanted your opinion on making such a diagnosis and where do we stand today with bromocriptine?
Out of 23 women who were diagnosed early, the earliest one was at 17th week and 8 others before the 28th week.
Additional Questions

- Timing of delivery.
Heart Failure Association of the European Society of Cardiology Study Group

Initial evaluation

Assess cardiopulmonary distress
SBP < 90 mmHg; HR > 130/min or < 45/min
RR > 25/min; SpO2 < 90%
Lactate > 2.0 mmol/L; ScvO2 < 60%
Altered mental state; cold skin; oliguria (< 0.5 ml/kg/min)

Confirm diagnosis
ECG
Blood tests incl. natriuretic peptides
Echocardiography, lung ultrasound
Consider additional tests to exclude differential diagnoses

Severe PPCM with cardiopulmonary distress

Optimize preload
Volume vs. diuretics; vasodilators if SBP >110 mmHg

Optimize oxygenation
Consider NIV, invasive ventilation if SpO2 <95%

Add inotropes and/or vaspressors
Consider levosimendan 0.1 mcg/kg/min during 24 h

Urgent delivery (cesarean section)

Consider bromocriptine (2.5 mg bid)

Consider mechanical circulatory support
if refractory cardiopulmonary distress

Recovery?

Transplantation
Weaning

PPCM without cardiopulmonary distress

Antepartum
HF therapy
Hydralazine
Nitrates
BB (metoprolol)
Consider diuretics

Consider delivery
(vaginal delivery with PDA)

Postpartum
HF therapy
ACEi (or ARB)
BB
Spironolactone
Diuretics
Consider icabradine

Consider WCD therapy
if LVEF ≤ 35%

Continue HF therapy
for ≥ 12 months after recovery of LV-function
## Table 2. Crude Outcomes by Gestational Age at Birth. *

<table>
<thead>
<tr>
<th>Outcome</th>
<th>All Infants</th>
<th></th>
<th>Infants Who Received Active Treatment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Hospital Rate‡</td>
<td>Overall Rate†</td>
<td>Hospital Rate‡</td>
</tr>
<tr>
<td></td>
<td></td>
<td>median (interquartile range)</td>
<td>mean (95% CI)</td>
<td>median (interquartile range)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25 Wk of gestation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Survival</td>
<td>72.0 (69.4–74.5)</td>
<td>71.2 (65.7–79.5)</td>
<td>72.3 (69.7–74.8)</td>
<td>71.7 (65.7–79.5)</td>
</tr>
<tr>
<td>Survival without severe impairment</td>
<td>61.1 (58.3–63.8)</td>
<td>59.3 (54.7–64.3)</td>
<td>61.4 (58.5–64.1)</td>
<td>59.9 (56.2–64.5)</td>
</tr>
<tr>
<td>Survival without moderate or severe impairment</td>
<td>44.3 (41.5–47.2)</td>
<td>46.0 (34.9–51.7)</td>
<td>44.5 (41.7–47.4)</td>
<td>46.5 (35.0–51.7)</td>
</tr>
<tr>
<td>26 Wk of gestation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Survival</td>
<td>81.4 (79.2–83.6)</td>
<td>81.0 (78.2–84.0)</td>
<td>81.6 (79.3–83.7)</td>
<td>81.3 (78.9–85.7)</td>
</tr>
<tr>
<td>Survival without severe impairment</td>
<td>75.6 (73.2–78.0)</td>
<td>75.7 (69.5–80.0)</td>
<td>75.7 (73.3–78.1)</td>
<td>76.4 (70.8–80.3)</td>
</tr>
<tr>
<td>Survival without moderate or severe impairment</td>
<td>58.5 (55.8–61.3)</td>
<td>58.9 (51.6–65.4)</td>
<td>58.6 (55.9–61.4)</td>
<td>59.8 (53.6–67.0)</td>
</tr>
</tbody>
</table>
28 patients with cardiogenic shock.
- 8 due to PPCM.
- All treated with Levosimendan.
- CI increased 1.2 +/- 0.6 L/Min/cm² (P<0.001).
- PCW decreased 11 +/- 4 mmHg (P<0.001).
- RA decreased 6 +/- 5 mmHg (P<0.001).
- Mean LVEF increased from 27% to 38% (P<0.001).
24 patients randomized to standard care (SC) or SC plus levosimendan.

Mean follow up 21 months.

Mortality 6 patients (25%), 3 in each group.

Persistent LV dysfunction 7 patients (27%), 4 in the levosimendan group.
Is Dobutamine Unsafe in PPCM?

- Data from the German PPCM registry showed that dobutamine treatment in patients with severe PPCM was associated with an adverse outcome (heart transplantation or LVAD implantation) while almost all patients not receiving dobutamine recovered.

Haghikia A. *Basic Res Cardiol* 2013;108:366
Dobutamine is Unsafe in PPCM?

- $\beta_1$-AR stimulation with isoproterenol in STAT3 knockout mice induced severe cardiac dysfunction and high mortality. In STAT3-deficient cardiomyocytes $\beta_1$-AR stimulation impaired glucose uptake and subsequently induced energy depletion, oxidative stress, dysfunction, and death.

Low STAT3 expression sensitizes to toxic effects of $\beta$-adrenergic receptor stimulation in peripartum cardiomyopathy

Staple B et al Eur Heart J 2017;38:349-361
Current management of patients with severe acute PPCM

HF Association of the ESC

- Optimize oxygenation – Consider NIV, invasive ventilation if SpO2 < 95%.
- Add inotropes and/or vasopressors – Consider levosimendan.
- Urgent delivery (Cesarean section).
- Consider bromocriptine (2.5 mg bid).
- Consider mechanical circulatory support.

Bauersachs J et al Eur J HF 2016;18;1096-1105
18 YO G1 developed tachycardia and hypertension with severe hypoxemia (PO₂ 30%) and acidosis during C section which was not corrected with Inotropic support and 100% oxygen. LVEF 18%. ECMO was used successfully for 24 h, extubated on day 4 and was D/C home on day 12. 1 month after D/C LVEDD of 42 mm and LVEF 56%.

Severe hypoxemia and acidosis during C section, LVEDD 52 mm, EF 18%

Fig 1. Severe pulmonary edema was shown in the chest x-ray taken at admission.

1 month after D/C LVEDD 42 mm, EF 56%

Fig 2. Chest x-ray taken during an outpatient visit (1 month after discharge).
Mice with homozygous or heterozygous cardiomyocyte-specific knockout of STAT3 (transcriptional activator) develop PPCM

↑Oxidative stress ↑superoxide production (↓MN SOD) → Cardiac Cathepsin D expression

Generates cleaved form of PROLACTIN (16 kDa)

Anti-angiogenic Pro-apoptotic

Endothelial cell apoptosis, Vasoconstriction, ↓capillary density

Impaired microcirculation and myocardial dysfunction

A Cathepsin D-Cleaved 16 kDa Prolactin Mediates PPCM
Hilfiker-kliner d et al Cell 2007;128:589
Bromocriptine in Management of PPCM
A Randomized Study on 96 Women in Burkina Faso
Yameogo NV et al J Cardiol Clin Res 2017

<table>
<thead>
<tr>
<th>Parameters</th>
<th>STHF+BR N=48</th>
<th>STHF N=48</th>
<th>P value</th>
</tr>
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<tbody>
<tr>
<td>BL LVEDD (mm)</td>
<td>59±3</td>
<td>58±4</td>
<td>0.6</td>
</tr>
<tr>
<td>BL LVEF</td>
<td>37±7%</td>
<td>37±5%</td>
<td>0.12</td>
</tr>
<tr>
<td>6 months LVEDD (mm)</td>
<td>53±2</td>
<td>55±2</td>
<td>0.002</td>
</tr>
<tr>
<td>6 months LVEF</td>
<td>50±2%</td>
<td>41±6%</td>
<td>0.001</td>
</tr>
<tr>
<td>12 months LVEDD (mm)</td>
<td>52±2</td>
<td>54±3</td>
<td>0.001</td>
</tr>
<tr>
<td>12 months LVEF</td>
<td>54±4%</td>
<td>46±6%</td>
<td>0.001</td>
</tr>
<tr>
<td>6 months mortality</td>
<td>17%</td>
<td>29%</td>
<td>0.0001</td>
</tr>
</tbody>
</table>
German Bromocriptine Study
Hilfiker-Kleiner D et al Eur Heart J 2017

**Figure 2** Analyses of global left ventricular ejection fraction (LVEF) change from baseline to 6 months follow-up determined by CMR. (A) Individual courses of LVEF change from baseline to 6-months follow-up in the 1W group (n = 23) and 8W group (n = 28) with a between-groups difference at 6-months follow-up of 2.0% in favour of the 8W groups (P = 0.38). (B) Individual courses of LVEF change from baseline to 6-months follow-up for the subgroup of patients with LVEF <30% at study entry in the 1W group (n = 14) and the 8W group (n = 18) with between-groups differences at 6-months follow-up of 4.3% and for LVEF change of 4.7% in favour of the 8W groups (P = 0.22).

**Figure 3** Outcome of patients at 6-months follow-up. (A) Left ventricular ejection fraction (LVEF) at 6-months follow-up according to predefined categories in all patients of the present study (treated 1W, n = 32 or 8W, n = 31 with bromocriptine, baseline LVEF <30%). Red columns illustrate the percentage of patients with no recovery (event or final LVEF <35%, prematurely terminated the trial or had missing LVEF data), yellow columns illustrate the percentage of patients with partial recovery (final LVEF 35% to <50%) and green columns depict percentage of women with complete recovery (final LVEF >50%). (B) Step-wise change in LVEF measured by echocardiography during follow-up period in the 1W (n = 21) and the 8W group (n = 24). The number 1–5 marks time course of the five patients who did not recover LVEF >35% after 6 months. However, after >12 months Number 1 displayed a LVEF = 63%, 2 a LVEF = 47%, 3 and 4 a LVEF = 50%, and 5 a LVEF = 13%.
Bromocriptine in PPCM

- Shown to be effective in 2 African studies with phenotypically and probably genetically different PPCM patients.
- German study was inconclusive.
- No information is available in the US.
Potential Down Sides of Bromocriptine Therapy

- It deprives women and their newborns from the many benefits of breast feeding.
- Associated with a risk of thromboembolism.
- Hypotension.
# The effects of bromocriptine in patients with chronic CHF

<table>
<thead>
<tr>
<th>parameter</th>
<th>Baseline</th>
<th>Bromocriptine</th>
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</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>87 +/- 16</td>
<td>78 +/- 17</td>
</tr>
<tr>
<td>SVR (dynes)</td>
<td>1494 +/- 361</td>
<td>1249 +/- 289</td>
</tr>
<tr>
<td>SVI (ml/m2)</td>
<td>27 +/- 7</td>
<td>33 +/- 10</td>
</tr>
<tr>
<td>Mean BP (mmHg)</td>
<td>87 +/- 9</td>
<td>73 +/- 9</td>
</tr>
<tr>
<td>Mean RA (mmHg)</td>
<td>10 +/- 4</td>
<td>7 +/- 4</td>
</tr>
<tr>
<td>Mean PCW (mmHg)</td>
<td>28 +/- 8</td>
<td>21 +/- 8</td>
</tr>
</tbody>
</table>

Francis G, Cohn J Am Heart J 1983;106:100-106
Hospital Course

Day 3:

Improvement of ventricular ectopy frequency

Trial of Bromocriptine causing hypotension

Day 12 (27 5/7 wks):

PVCs
BP 90/60s
PA pressure 60/36 mmHg
PCWP 35 mmHg
CO 4, CI 1.9-2.0 L/min/m² by FICK

Fetus: Monitored with non stress test and biophysical profiles
Steroids and Magnesium
18 YO G1 developed tachycardia and hypertension with severe hypoxemia (PO\textsubscript{2} 30%) and acidosis during C section which was not corrected with Inotropic support and 100% oxygen. LVEF 18%. ECMO was used successfully for 24 h, extubated on day 4 and was D/C home on day 12. 1 month after D/C LVEDD of 42 mm and LVEF 56%.

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Fig 1. Severe pulmonary edema was shown in the chest x-ray taken at admission.

Fig 2. Chest x-ray taken during an outpatient visit (1 month after discharge).
26 YO CS for placenta previa at 34 weeks hemodynamic instability requiring vasopresor. Severe hypoxemia requiring ECMO for 71 hrs, LVEF 20% improved to 55% 29 d after delivery.

Table 2. Summary of data from the literature regarding PPCM treatment with VA ECMO (*LVAD = left ventricular assist device).

<table>
<thead>
<tr>
<th>Author, year of publication</th>
<th>Patient’s (age in years), kind of delivery, condition, complications</th>
<th>Duration of ECMO run</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yang 2007^9</td>
<td>(18) cardiac failure and severe pulmonary edema immediately after a cesarean section</td>
<td>28 hours</td>
<td>discharged on day 12 with EF of 58%</td>
</tr>
<tr>
<td>Smith 2009^9</td>
<td>(19) low cardiac output and pulmonary edema just after a vaginal birth complicated by massive post partum hemorrhage and uterine atony</td>
<td>68 hours</td>
<td>discharged on day 19 with normal RV and LV function, no wall motion abnormalities</td>
</tr>
<tr>
<td>Palanzo 2009^10</td>
<td>(24) heart failure with severe cardiogenic shock (EF 15%) within four months of delivery</td>
<td>11 days</td>
<td>discharged on day 30, EF 47% after 5 months of follow-up</td>
</tr>
<tr>
<td>Gevaert 2011^11</td>
<td>(28) Initially not recognized heart failure at the end of pregnancy, cardiogenic shock after cesarean section</td>
<td>11 days of VA ECMO as bridge to LVAD^a</td>
<td>survived after heart transplant on day 78 of LVAD^a</td>
</tr>
<tr>
<td>Chen 2011^12</td>
<td>(27) dilated cardiomyopathy with EF of 21% on day 8 after cesarean section with recovery. Symptom recurrence 3 months after delivery</td>
<td>10 day</td>
<td>survived, second pregnancy and section delivery 2 years later with ECMO stand-by</td>
</tr>
<tr>
<td>Park SH 2014^13</td>
<td>use of VA ECMO in two full-term patients (both 34) for PPCM treatment and safe cesarean section on VA ECMO support</td>
<td>5 and 6 days</td>
<td>both mothers and both children survived without clinical irregularities</td>
</tr>
<tr>
<td>Kim HY 2014^15</td>
<td>two patients (27,32) after cesarean section delivery</td>
<td>119 hours 203 hours</td>
<td>one patient did not survived</td>
</tr>
</tbody>
</table>
# Impella in PPCM and Cardiogenic Shock

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Age/Race</th>
<th>Time of presentation</th>
<th>Reason for use of Impella</th>
<th>Other MCD</th>
<th>Duration of Impella use</th>
<th>LVEF before Impella (%)</th>
<th>LVEF after Impella (%)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25 y, AA</td>
<td>7 d PP</td>
<td>Severe HF</td>
<td></td>
<td>3 d</td>
<td>10-15</td>
<td>35</td>
<td>D/C home on d 6</td>
</tr>
<tr>
<td>2</td>
<td>27 y, AA</td>
<td>5 d PP</td>
<td>CS</td>
<td>IABP</td>
<td>4 d</td>
<td>20</td>
<td>20</td>
<td>LVAD HTx</td>
</tr>
<tr>
<td>3</td>
<td>24 y, AA</td>
<td>PP time NA</td>
<td>CS multiple codes</td>
<td>IABP</td>
<td>72 d</td>
<td>10</td>
<td>NA</td>
<td>LVAD</td>
</tr>
<tr>
<td>4</td>
<td>37 y, AA</td>
<td>1 d PP</td>
<td>CS</td>
<td>IABP</td>
<td>3 d</td>
<td>15</td>
<td>NA</td>
<td>Transferred with Impella for LVAD</td>
</tr>
<tr>
<td>5</td>
<td>25 y, NA</td>
<td>37 w</td>
<td>CS, VT, PEA</td>
<td></td>
<td>3 d</td>
<td>10-15</td>
<td>20-25</td>
<td>D/C home on dialysis</td>
</tr>
<tr>
<td>6</td>
<td>17 y, AA</td>
<td>6 m PP</td>
<td>CS</td>
<td>ECMO BiVAD</td>
<td>1 d</td>
<td>5</td>
<td>NA</td>
<td>Multi-organ failure, death on d 5</td>
</tr>
<tr>
<td>7</td>
<td>18 y, AA</td>
<td>23 w</td>
<td>CS, VT, cardiac arrest</td>
<td></td>
<td>1 d</td>
<td>30-40</td>
<td>55-60</td>
<td>D/C home on d 14</td>
</tr>
<tr>
<td>8</td>
<td>30 y, C</td>
<td>38 w</td>
<td>HF</td>
<td></td>
<td>3 d</td>
<td>10</td>
<td>35-40</td>
<td>D/C home on d 13</td>
</tr>
<tr>
<td>9</td>
<td>39 y, NA</td>
<td>5 d PP</td>
<td>CS, Bradycardia, PEA</td>
<td>NA</td>
<td>&lt;15</td>
<td>NA</td>
<td>NA</td>
<td>Transferred with Impella for LVAD</td>
</tr>
<tr>
<td>10</td>
<td>39 y, C</td>
<td>4 d PP</td>
<td>CS, severe MR</td>
<td></td>
<td>5 d</td>
<td>45</td>
<td>normal</td>
<td>D/C home on d 21</td>
</tr>
</tbody>
</table>
Importance of Hemodynamic Monitoring

- Hemodynamic optimization prior to delivery.
- Correct hemodynamic instability during delivery.
- Prevent and manage post delivery hemodynamic changes due to increased venous return.

- 1. relief of IVS obstruction
- 3. Transfer fluid from extra cellular space
With aggressive management of severe PPCM it is possible to stabilize patients and delay early delivery as well as prevent death and bridge patients to recovery or durable MAC/heart transplantation.
- Vitals: 96/82, P111, RR 25, 97% RA
- Gen: mild distress
- Neck: Supple, JVD 11-12cm
- Irregularly irregular rhythm, 3/6 systolic murmur at apex radiating to axilla
- Lungs: bilateral crackles at bases
- Abd: Soft, gravid
- Ext: Warm, trace edema
- Fetus: No significant compromise.
Palpitations and progressive dyspnea on exertion

EKG
Echo
Hospital Course

- Admission: Esmolol and lidocaine (Mexilitine), LMWH, Furosemide.
- Furosemide
- Day 3: prevention of ventricular ectopy, failed trial of bromocriptine due to hypotension. Fetus monitored with non stress test and bioprofile.
- Day 12: PVC’s, BP 90/60, PA pressure 60/35 mmHg, PCW 35, CO-4, CI 1.9.
Delivery Plan

- Anticipated post delivery hemodynamic changes: increased venous return due to
  1. relief of IVS obstruction
  2. Autotransfusion.
  3. Transfer fluid from extra cellular space
Delivery Plan

- Anticipated hemodynamics:
  - Increased RA and LA pressure
  - Enlargement of the ventricles – Increased MR and TR.
  - Increased wall tension and myocardial O2 consumption – Ischemia, arrhythmias
Recommended Delivery Management

- Hemodynamic optimization prior to deliver.
- Hemodynamic monitoring during delivery.
- Inotropes not a good choice because of tachycardia and arrhythmias.
- Intraaortic ballon pump-ideal, arrhythmias may interfere.
- Impella.
- Continue hemodynamic monitoring after delivery.
- Aggressive diuresis after delivery
Delivery

- Patient was delivered on day 13 at 28 weeks.
- General anesthesia.
- Impella CR placed plus sheath for a potential emergency ECMO.
- Flow 3.5 l/min, PA 32/16, RA 9.
- C section with left uterine displacement.
Delivery

- Extubated after several hours
- Impella weaned after 36 hours.
- Day 15 Heart failure regiment optimized
- Bromocriptine x1 wk on UFH
- Warfarin
- D/C on day 21
Impella CP

- Pump Motor
- Blood Outlet
- Blood Inlet
- Pigtail

- Catheter Diameter: 9Fr
- Flow Rate Up to: 4.0L/min
Impella

- EDV & EDP
- Mean arterial pressure
- Pulmonary capillary pressure
- Diastolic pressure
- Cardiac output
- Systemic perfusion
- RV afterload
- Oxygen demand
- LV work
- Coronary blood
- Independent of EKG stability
5 months Post Partum

- Mother severe LV dysfunction still on life vest
- Baby pneumothorax and infections D/C home after 3 months
Methamphetamine Induced Cardiomyopathy

- Dilated cardiomyopathy.
- Characterized by severe LV dilatation and reduced ejection fraction.
- High incidence of intra cardiac thrombi.
- Can be reversible with discontinuation of methamphetamine use.
- Presenting with severe symptoms in the 2\textsuperscript{nd}/3\textsuperscript{rd} trimester.
Methamphetamine Induced Cardiomyopathy

A 29 y/o Hispanic F, G4P3, 33+5 weeks. Referred to our program with complaints of increasing shortness of breath.

- Methamphetamine abuse – 4 years, until 3 months ago.
- No functional limitations prior to pregnancy.
- 3 normal previous pregnancies and deliveries. CS x 3.
- No home medications.
Methamphetamine Induced Cardiomyopathy

- Symptoms began 1 month ago and progressed.
- NYHA class III-VI on presentation.
- BP 98/65, HR 110 regular, O2 sat 91%
- Volume overload.
Methamphetamine Induced Cardiomyopathy

- **CTA** – no evidence of PE; signs of congestion.
- **Echo** – EF 25%, enlarged LV, normal RV, moderate MR, SPAP 42 mmHg.
- **RHC** – RA 13, RV 60/12, PA 59/36, PCWP 35
Why Hemodynamic Monitoring?

- Most accurate information.
- Allows optimization of pressures before delivery.
- Provides continuous monitoring during labor, delivery and the post delivery period.
Hemodynamic Changes After Delivery

- Sudden shift in fluid balance due to Cessation of the inferior vena cava compression by the fetus
- “Auto transfusion” from the contracting uterus.
- Movement of pooled blood from the lower limbs into the venous system.
Hemodynamic Changes After Delivery

- Increase in afterload to the left ventricle due to the loss of the low resistance placenta.

- Increased LA pressure → pulmonary edema and arrhythmias.
Patient That Should be Monitored in the CCU During Delivery

- Patients with severe arrhythmias.
- Patients with severe heart failure, especially on inotropes or mechanical circulatory support.
- Patients who are hemodynamically monitored for heart failure due to myocardial disease, severe valvular disease.
- Patients with severe pulmonary hypertension.
Heart Failure in Pregnancy

Thank You