Critical Care of the Obstetric Patient

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Outline

• Common causes of admissions to the ICU
• Physiologic changes of pregnancy
• Causes of obstetrical hemorrhage
• Review amniotic fluid embolus (AFE)
• Discuss pre-eclampsia/eclampsia
• ARDS in the pregnant patient

Case Report

29 yo G1P0 woman was admitted at 39 weeks gestation for induction of labor. She had a h/of IDDM since the age of six. She had not suffered any complications related to her diabetes. Her pregnancy had been uncomplicated. She had been able to maintain good blood sugar control.

Case Report
Following admission she was given prostaglandin intravaginal gel. 5 hours later she had spontaneous rupture of membranes followed by rapid progression of labor. A tetanic contraction of her uterus occurred and she was moved to the OR. She was noted to be fully dilated and the decision was made to proceed with delivery by vacuum extraction. The baby was born with Apgars of 5 and 8.


Case Report
Her delivery was complicated by a fourth degree vaginal tear. Following delivery of the placenta the patient received oxytocin, and the tear was repaired under local. Excessive vaginal bleeding was noted but uterine tone was felt to be good. The patient was resuscitated for ongoing hemorrhage despite good uterine tone.


Case Report
Coagulation studies revealed a PT of 27.5, a PTT of 149 and a fibrinogen of 60. Her Hgb was 8.6. She received embolization of the vaginal arteries and was transferred to the ICU. Over the next several hours the patient continued to bleed profusely despite ongoing correction of her coagulopathy. She was taken to the OR and eventually received a hysterectomy to control the bleeding.

Incidence and Mortality Rates

- 0.11-0.89% of deliveries result in ICU admissions

- Maternal mortality rates 2-20%

Gilbert et al., Obstet and Gynecol, 2003; 897-903
Kippatric et al., Chest, 1992; 1407-12
Loverro et al., Arch Gynecol Obstet, 2001; 265:195-8
Mahutte et al., Obstet and Gynecol 1999; 263-266

Obstetric Admissions to the Intensive Care Unit: Outcomes and Severity of Illness

Table 2. CU Admissions of All 231 Obstet Patients by Type of Admission (Antepartum, Postpartum and Delivery Type: Antepartum, Obstetric)

<table>
<thead>
<tr>
<th>Antepartum (n = 154)</th>
<th>Postpartum (n = 77)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication type</td>
<td>Diagnosis</td>
</tr>
<tr>
<td>Medical diagnosis</td>
<td>Diagnosis</td>
</tr>
<tr>
<td>Obstetric diagnosis</td>
<td>Diagnosis</td>
</tr>
</tbody>
</table>

- SAPS score

Obstetric Admissions to the Intensive Care Unit

- McGill University ICU services
- 131 OB admissions from 1991-1997
- Incidence: 0.3% of all deliveries
- 78% admitted postpartum

Mahutte et al., Obstet and Gynecol, 1999; 94: 263-266
Obstetric Admissions to the Intensive Care Unit

<table>
<thead>
<tr>
<th>Reason</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstetric hemorrhage</td>
<td>34 (26%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>28 (21%)</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>18 (14%)</td>
</tr>
<tr>
<td>Respiratory disorders</td>
<td>13 (10%)</td>
</tr>
<tr>
<td>Infection</td>
<td>13 (10%)</td>
</tr>
<tr>
<td>Other</td>
<td>25 (19%)</td>
</tr>
</tbody>
</table>

Mahutte et al, Obstet and Gynecol, 1999; 94: 263-266

Obstetric Admissions to the Intensive Care Unit

Hemorrhage → abnormal placentation, uterine atony, lacerations, retained products of conception, severe coagulopathy
Hypertension → preeclampsia/eclampsia with or without HELLP (Hemolysis, Elevated Liver enzymes, Low Platelet count)
Cardiac disease → valvular diseases, cardiomyopathy, arrhythmias
Respiratory disorders → pulmonary edema, asthma
Infection → pyelonephritis, chorioamnionitis

Mahutte et al, Obstet and Gynecol, 1999; 94: 263-266

Respiratory Changes in Pregnancy

<table>
<thead>
<tr>
<th>Pulmonary Function</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Forced Expiratory Volume in 1 second</td>
<td>No change</td>
</tr>
<tr>
<td>Functional Residual Capacity</td>
<td>Decreased 10-25%</td>
</tr>
<tr>
<td>Total Lung Capacity</td>
<td>Mininal decrease</td>
</tr>
<tr>
<td>Minute Ventilation</td>
<td>Increased 20-40%</td>
</tr>
<tr>
<td>Alveolar Ventilation</td>
<td>Increased 50-70%</td>
</tr>
</tbody>
</table>

Lapinsky S., Crit Care Med, 2005 Vol. 33, No. 7 1616-1622
Lung Volume Changes in Pregnancy

Nonpregnant lung volumes
Pregnant lung volumes

Cardiovascular Changes in Pregnancy

<table>
<thead>
<tr>
<th>Hemodynamics</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate</td>
<td>Increased 10-30%</td>
</tr>
<tr>
<td>Pulmonary Artery Occlusion Pressure</td>
<td>No change</td>
</tr>
<tr>
<td>Cardiac Output</td>
<td>Increased 30-50%</td>
</tr>
<tr>
<td>Systemic Vascular Resistance</td>
<td>Decreased 20-30%</td>
</tr>
<tr>
<td>Pulmonary Vascular Resistance</td>
<td>Decreased 20-30%</td>
</tr>
</tbody>
</table>

Cardiovascular Changes in Pregnancy (continued)

- Blood volume increases up to 2 L (30-50% above normal intravascular volume)
  - High uterine and placental blood flow demand (up to 600 mL/min at term)
  - Increased pelvic venous capacitance
  - Protects mother against blood loss during delivery
- Body position alters hemodynamics after 20 weeks
  - 30% reduction in ejection fraction in supine position
  - Left lateral position improves venous return
Arterial Blood Gas in the Pregnant Patient

<table>
<thead>
<tr>
<th>Arterial Blood Gas</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO2*</td>
<td>No change</td>
</tr>
<tr>
<td>PaCO2</td>
<td>Reduced to 28-32 Torr</td>
</tr>
<tr>
<td>Serum Bicarbonate</td>
<td>Reduced to 18-21 mEq/L</td>
</tr>
</tbody>
</table>

*oxygen consumption increases nearly 20% at term but is offset by increase in cardiac output

Lapinsky S., Crit Care Med, 2005 Vol. 33, No. 7 1616-1622

Fetal Oxygen Delivery

- Determinants:
  - Maternal arterial oxygen content
  - Uterine Blood Flow
  - Hgb Concentration

Lapinsky S., Crit Care Med, 2005 Vol. 33, No. 7 1616-1622

Obstetric Admissions to the Intensive Care Unit

- Hemorrhage → abnormal placentation, atony, lacerations, retained products of conception, severe coagulopathy
- Hypertension → preeclampsia/eclampsia with or without HELLP (Hemolyisis, Elevated Liver enzymes, Low Platelet count)
- Cardiac disease → valvular diseases, cardiomyopathy, arrhythmias
- Respiratory disorders → pulmonary edema (ARDS), asthma
- Infection → pyelonephritis, chorioamnionitis

Mahutte et al, Obstet and Gynecol, 1999; 94: 263-266
Case Report

Diagnosis:

Post-partum hemorrhage secondary to amniotic fluid embolus leading to a coagulopathy (DIC)

Lapinsky S., Crit Care Med, 2005 Vol. 33, No. 7 1616-1622

Hemorrhage

- Accounts for ~17% of maternal deaths
- Causes:
  - Abnormal placentation
  - Uterine atony
  - Cervical/Vaginal laceration
  - Coagulopathy (Amniotic fluid embolus, Abruptio placentae, HELLP, Retained dead fetus)
- Treatment:
  - Drugs (uterine atony → ergot derivatives, prostaglandin analogues, oxytocin)
  - Coagulopathy → Factor VIIa
  - Extreme cases of hemorrhage: embolization of uterine or iliac arteries or surgical exploration +/- hysterectomy

Lapinsky S., Crit Care Med, 2005 Vol. 33, No. 7 1616-1622

Amniotic Fluid Embolism

- Exceedingly rare
- United States registry and United Kingdom registry
  - US: 46 in 1995
  - UK: 44 in 2005
- Estimated to occur in 1 in 8,000 to 1 in 80,000 deliveries
- High mortality (60% in older reports; 27% in newer population based study; 37% in 2005 UK registry)

Gist et al, Anesthesia and Analgesia, 2009; 108: 1599-1602
Amniotic Fluid Embolus

- Neonatal outcome poor with mortality rate of 20-25%
- Occurs intrapartum or immediate postpartum period
- NO PROVEN RISK FACTORS
- ONSET CANNOT BE PREDICTED

Gist et al, Anesthesia and Analgesia, 2009; 108: 1599-1602

Amniotic Fluid Embolism

<table>
<thead>
<tr>
<th>Signs or symptoms</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>100%</td>
</tr>
<tr>
<td>Respiratory distress</td>
<td>100%</td>
</tr>
<tr>
<td>Pulmonary edema or ARI</td>
<td>93%</td>
</tr>
<tr>
<td>Cardiopulmonary arrest</td>
<td>92%</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>83%</td>
</tr>
<tr>
<td>Coagulopathy</td>
<td>83%</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>49%</td>
</tr>
<tr>
<td>Nausea</td>
<td>48%</td>
</tr>
<tr>
<td>Uterine atony</td>
<td>23%</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>17%</td>
</tr>
<tr>
<td>Transient hypertension</td>
<td>11%</td>
</tr>
<tr>
<td>Cough</td>
<td>7%</td>
</tr>
<tr>
<td>Headache</td>
<td>7%</td>
</tr>
<tr>
<td>Chest pain</td>
<td>2%</td>
</tr>
</tbody>
</table>

ARDS = adult respiratory distress syndrome
IAH = intrapulmonary hypertension

Amniotic Fluid Embolism

1. Amniotic fluid enters maternal circulation through endocervical veins, placental insertion site, or uterine trauma site
2. Acute pulmonary hypertension
   - right heart failure
   - hypoxia
   - cardiac arrest
3. Left ventricular failure (mechanism unclear)
   - Hypoxic injury during initial phase
   - Release of inflammatory mediators
   - Direct depressant effect of amniotic fluid on myocardium
4. Pulmonary edema

Gist et al, Anesthesia and Analgesia, 2009; 108: 1599-1602
Amniotic Fluid Embolism

- Treatment is supportive
  - Correct hypoxemia
  - Correct hypotension
  - Improve inotropy
  - Consider blood products/Factor VIIa
  - Determine whether fetus should be delivered

- Consider rescue therapies
  - Inhaled nitric oxide
  - Ventricular assist devices
  - ECLS/ECMO

Gist et al, Anesthesia and Analgesia, 2009; 108: 1589-1602

Pre-eclampsia/Eclampsia

- Proteinuria and hypertension occurring after the 20th week of pregnancy
  - + seizures = eclampsia

Mushambi et al, Br J Anaesth, 1996; 76: 133-148

Pre-eclampsia/Eclampsia

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Definition of pre-eclampsia [38]</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.  Gross</td>
<td>Gestation &gt; 20 weeks</td>
</tr>
<tr>
<td>2.  Arterial pressure</td>
<td>Diastolic &gt; 130 mm Hg on any one occasion or Diastolic &gt; 90 mm Hg on 2 or more occasions at least 6 h apart</td>
</tr>
<tr>
<td>3.  Proteinuria</td>
<td>&gt; 300 mg in 24 h on 2-4 samples at least 4 h apart with:</td>
</tr>
<tr>
<td>2.  Albuminuria</td>
<td>(1) 1 g albuminuria in 24 h on 2 or more urinalysis strips</td>
</tr>
<tr>
<td>2.  Urine output</td>
<td>(2) 0.5 g hemoglobin in 24 h on urinalysis strips if urine pH &gt; 8 or specific gravity is &lt; 1.090</td>
</tr>
<tr>
<td>4.  Cerebral</td>
<td>Not essential for diagnosis</td>
</tr>
</tbody>
</table>

Mushambi et al, Br J Anaesth, 1996; 76: 133-148
Multi-system Changes in Pre-eclampsia

- Placental Abnormality
- Vasospasm
- Reduced Organ Perfusion
- Multiorgan Dysfunction

Pre-eclampsia/Eclampsia

Complications
- Refractory hypertension
- Neurologic dysfunction (seizures, intracranial hemorrhage)
- Elevated ICP
- Renal Failure
- Liver rupture or failure
- HELLP syndrome (Hemolytic Anemia, Elevated Liver enzymes, Low Platelet count)
- Pulmonary edema
- DIC

Pre-eclampsia/Eclampsia

- Management:
  - Delivery → removal of fetus and placenta
  - Anti-hypertensive therapy is used only to prevent maternal hypertensive complications and does not alter the natural history
  - Careful volume expansion
  - Magnesium infusion for seizure prophylaxis
ARDS in Pregnancy

Table 1. Pathogenesis of acute respiratory distress syndrome

<table>
<thead>
<tr>
<th>Tissue injury</th>
<th>Right heart failure</th>
<th>Unique to pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct injury</td>
<td>Pulmonary edema</td>
<td>Alveolar-capillary (\text{membrane}) disruption</td>
</tr>
<tr>
<td>Septic</td>
<td></td>
<td>Alveolar-capillary (\text{membrane}) disruption</td>
</tr>
<tr>
<td>Ischemic</td>
<td>Disease of capillaries</td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>Tumor</td>
<td></td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>Focal bleeding</td>
<td></td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>Local infection</td>
<td></td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Pulmonary edema</td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>Trauma</td>
<td>Disease of capillaries</td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>Acute pancreatitis</td>
<td></td>
<td>Acute alveolar (\text{injury})</td>
</tr>
<tr>
<td>TRALI</td>
<td>Disease of capillaries</td>
<td>Acute alveolar (\text{injury})</td>
</tr>
</tbody>
</table>

ARDS, acute respiratory distress syndrome; TRALI, transfusion-related acute lung injury.

Critical Care of the Obstetric Patient

- Common causes of admission to the ICU are hemorrhage, hypertension, cardiac disease, respiratory disorders, and infection
- Consider the physiologic changes in pregnancy
- Remember left uterine displacement!
- Consider rare causes of cardiovascular collapse and DIC
- Attempt low tidal volume ventilation in pregnant patients with ARDS but consider the effects on the fetus

Low Tidal Volume Ventilation and Hypercapnia

- Adequate fetal oxygenation requires a \(\text{PaO}_2 > 70\) mm Hg
- No human data on uteroplacental and umbilical blood flow with hypercapnia
- Fetal \(\text{PaCO}_2\) must be \(10\) mm Hg higher than maternal to allow exchange across the placenta
- Acidosis shifts oxygen Hgb dissociation to the right → limiting ability of fetal Hgb to bind oxygen
- Limited data to suggest that a \(\text{PCO}_2\) range between 45 and 60 can be tolerated by the fetus
- Maternal \(\text{PaCO}_2 \approx 45\) mm Hg may be safe limit
- Insufficient evidence to know if \(\text{HCO}_3\) is transferred efficiently across the placenta

Bandi et al, Crit Care Clin, 2004; 20: 577-607
Cole et al, Crit Care Med, 2005; 33: S269-S278