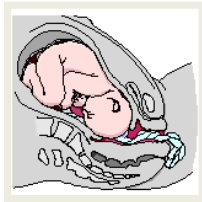


Umbilical Cord Prolapse

Defined as:

- The umbilical cord lies beside or below the presenting part
 - Funic
 - Occult
 - Complete



Incidence:

- 1/ 275 births =0.36
(Dildy & Clark, 1993)
- 1.4 and 6.2/ 1000 births = 0.14 – 0.62%
(Koonings PP, Campell K., 1990)
(Lin, Mg. 2006)
(Phelan & Holbrook, 2013)
- No significant change over time.
(Stable over last century.)

Risk factors - Preexisting:

- Abnormal placentation
- Long umbilical cord
- Polyhydraminos
- Uterine tumors
- Small or preterm fetus
- Malpresentations
- Multiple gestation
- Unengaged presenting part
- High parity

Risk factors - Iatrogenic:

- Amniotomy
- External cephalic version
- Fetal scalp electrode placement
- IUPC placement
- Forceps application
- Fetal pulse oximetry placement
- Fetal scalp blood sampling

Diagnosis:

- Most obviously ??

Differential Diagnosis:



Remember -

- Repetitive variable decelerations late in the contraction cycle may indicate a prolapse of the umbilical cord through a dehiscence of the uterine scar.

Management:

- Following ROM may rule out through vaginal exam
- If prolapse, relieve compression of the cord
- Continuously elevate the presenting part until delivery of the neonate, either through manual elevation or patient repositioning (knee chest or trendelenburg)
- Fill the woman's bladder with 500 – 700 mls of sterile saline to elevate presenting part (Katz, 1988)

**Other Supportive
Measures
to Prepare for Delivery**

- O2 at 10L/min by face mask
- IV fluid hydration bolus
- DC oxytocin
- Continuous fetal assessment
- Administration of tocolytic agent to decrease uterine activity
- Anticipate compromised neonate and the need for neonatal resuscitation

Amniotic Fluid Embolism

Anaphylactoid Syndrome of Pregnancy

Defined:

Historically --

- Amniotic fluid enters the maternal circulation, resulting in blockage of the pulmonary vasculature and tissue destruction.

???Lone cause???

Incidence:

- Varies from 1/ 8000 to 1/ 80,000 pregnancies worldwide
(Clark et,al., 1995)
- 1/8000 to 1/30,000 in US
(Tuffnell, D.J. 2005)
- California Study (1994-1995) – 1 million deliveries: 1/20,000
- True Incidence....

Mortality/ Morbidity :

- Accounts for approximately 10% of all maternal deaths
 - Maternal mortality is 20-30%
 - Survivors: 85% suffer long-term neurological sequelae
(15% neurologically intact)
- Fetal mortality: 21%
- Survivors: 50% neurologic injury

Time from onset of signs/symptoms to maternal death:

Risk factors:

Clinical Manifestations & Diagnosis:

- Appears to occur after maternal intravascular exposure to amniotic fluid or fetal tissue (supportive diagnosis)
- Development of profound shock – cardiovascular collapse associated with severe respiratory distress or DIC

Pathophysiology:

--incompletely understood
Intense reaction?
Benign?

--Leong AS, Reprod Sci 2008

Presenting Signs & Symptoms:

- Disorientation, decreased level of consciousness
- Sudden dyspnea, cyanosis, respiratory distress

3 Phases

1. Amniotic fluid enters maternal pulmonary vasculature.
2. Release of inflammatory mediators
 - Myocardial depression
 - Lung injury
 - Neurologic compromise
3. Immunologic activation of coagulation pathway

Further Signs and Symptoms

Differential Diagnosis

Survival – but...

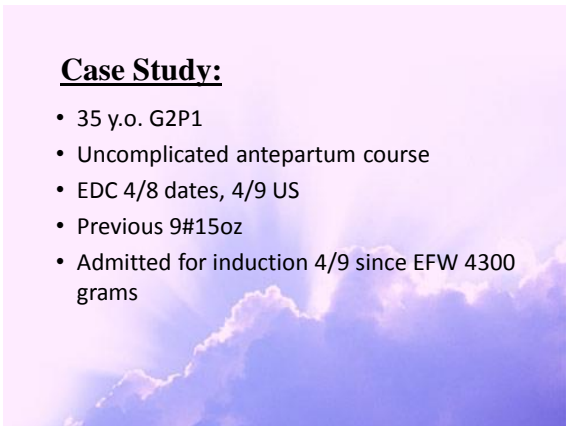
- Development of coagulopathy
- Noncardiogenic pulmonary edema
- Acute respiratory distress syndrome
- Acute tubular necrosis (Clark, 1990)

Management

- Maintain maternal central hemodynamic stability.

Case Study:

- 35 y.o. G2P1
- Uncomplicated antepartum course
- EDC 4/8 dates, 4/9 US
- Previous 9#15oz
- Admitted for induction 4/9 since EFW 4300 grams



Management:

- 2nd IV line started
- Blood readied
- Hespan
- Dopamine infusion
- Uterine massage

Patient:

- BP remained 70 systolic
- Uterus continued to bleed heavily

Massive Transfusion Protocol

Management Goals

Pathology:

- Respiratory
- Cardiovascular system
- Uterus – within deep myometrial vessels
- Cervix

- Embolism
- Platelet activation and degranulation
- Pulmonary hypertension due to serotonin and thromboxane
- Systemic hypotension and bradycardia due to reflex vagal stimulation
- Death

A-OK Protocol

- Atropine 1 mg for vagolysis
- Ondansetron 8 mgs to block serotonin receptors and for vagolysis
- Ketorolac 30 mg to block thromboxane production
- Off-label recommendations

New Insight

- United States National Registry of AFE
 - United Kingdom
- analysis of data for further understanding

DIC

Disseminated Intravascular
Coagulation

Predisposing Conditions:

- Anaphylactoid syndrome of pregnancy (AFE)
- Abruptio placentae (most common cause)
- Severe preeclampsia
- HELLP
- Septicemia
- Severe hemorrhage
- Retained IUFD

Clinical Manifestations & Diagnosis:

- Unusual bleeding
- Tachycardia
- Diaphoresis
- Symptoms of shock (late finding)

Nursing Assessment & Care:

- Assess and monitor...

Laboratory:

- Decreased platelets, fibrinogen, proaccelerin, antihemophilic factor, prothrombin (factors consumed during coagulation)
- Fibrinolysis increases initially (later severely depressed)
- Degradation of fibrin leads to accumulation of FDPs in blood
- FDPs – anticoagulant properties – prolong PT, PTT time

Blood Components & Fluid Volume:

“Massive Transfusion Protocol”

- Packed RBCs through one IV line
- FF Plasma through a second IV line
- Platelets
- Recheck serum fibrinogen level and platelet count every 30 – 60 minutes

Blood Components / Clotting Factors:

- Cryoprecipitate
each 40-50 ml bag contains approximately the same amount of fibrinogen as a unit of FFP, plus some other clotting factors (including Factor VIII)
- Fresh Frozen Plasma
Regular: 200-250 ml bag contains fibrinogen + all other clotting factors (higher level of Factor VIII) -
or
Prepared by plasmapheresis: 400-600 ml bag
- Platelets 5-10 units

REACT

- R= Resuscitation
- E= Evaluation
- A= Arrest Hemorrhage
- C= Consult
- T= Treat Complications

(Knuppel & Hatangadi, 1995)

Uterine Rupture

Defined:

- The separation of the uterine myometrium or previous uterine scar, with rupture of the membranes and extrusion of the fetus or fetal parts into the peritoneal cavity.

Classified as complete or incomplete

- Complete: laceration directly into the peritoneal cavity
- Incomplete: laceration is separated from the peritoneal cavity by the visceral peritoneum

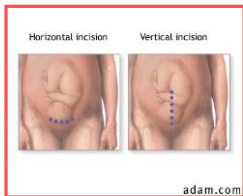
Dehiscence: previous scar begins to separate

Incidence:

- Rare
- Risk of rupture is 0.2% to 1.5% with prior low transverse Cesarean scar (ACOG, 1999b)
- 0.32% overall
0.02% elective repeat C.S
0.12% indicated repeat C.S.
(OB/GYN 2007)
- Incidence of asymptomatic scar dehiscence is 1.1% to 2% (Phelan, Korst & Settles, 1998)
- A normal uterus with no prior surgery contracting spontaneously is unlikely to rupture unless

Conditions associated with uterine rupture:

- Prior uterine surgery
- Previous cesarean delivery – (or myomectomy)



Type of Scar:

Low transverse:

Classical incision:

- Short interdelivery interval (<18 months)
- Oxytocin use
- Prostaglandin preparations
- Parity greater than 4
- Abruptio placentae
- Midforceps delivery
- Breech version and extraction
- Trauma
- CPD

Presentation:

- Depends on specific type and timing of rupture
- Dehiscence of a prior low-segment cesarean scar is initially asymptomatic
- Prolapse of the umbilical cord can occur through the scar tissue

As rupture continues:

- Contractions ???
- Significant association between dysfunctional labor and arrest of dilatation (causality is unclear)
- Hypotension and shock

Complete uterine rupture:

- Perimetrium and myometrium are involved
- Clinical signs immediate
- Uterine contractions cease and fetal parts are palpable through the abdominal wall

Remember:



Signs and symptoms:

- Sudden fetal distress is the most common sign/ symptom even prior to the onset of abdominal pain or vaginal bleeding.

Case Study:

- Previous C. Section at 37 weeks gestation, with c/o ?early labor
- Irregular contractions per EFM
- FHR reactive
- SVE by nurse: cervix closed, intact membranes

Nursing Diagnoses

1. Maternal shock related to blood loss secondary to uterine rupture.

Interventions:

2. Fetal distress related to impaired uteroplacental blood flow.

Interventions:

3. Fear related to life-threatening complication and threatened loss of babe.

Interventions:

Management:

- Maternal stabilization and immediate cesarean birth
- Repair uterine defect (if possible)

Uterine Inversion

Presentation:

- Hemorrhage
- Shock
- Pain
- Attempts to massage the fundus are unsuccessful.
- The fundus has inverted into the uterus, the vaginal vault, or the introitus.

Prepare for surgery:

- Begin blood volume expansion
- If placenta is still attached, do not remove it until blood volume expansion fluids are being given

Management:

- Combat shock (out of proportion to blood loss)
- Withhold oxytocin until the uterus has been repositioned
- Attempt to manually replace the uterus (tocolytic or general anesthesia)
Magnesium sulfate: 4-6 Gm IV over 5-10 minutes
Terbutaline: 0.25 mg IV
- If manual replacement fails, be prepared for abdominal or vaginal surgery to reposition the uterus
- Blood replacement therapy as indicated
- Broad spectrum antibiotic therapy
- Nasogastric tube to minimize paralytic ileus

- Nursing assessments and interventions for uterine inversion are the same as for any obstetrical hemorrhage
- Suspect an incomplete inversion ???

SEPSIS

In OB?...

...think about it!

Part 1:

SIRS – Systemic Inflammatory Response

The body's response to severe clinical insult manifested by 2 or more symptoms listed below:

- Temperature > 38 degrees C or < 36 degrees C >100.4 x 24 hrs
- HR > 90 BPM (>110 if laboring or recently delivered)
- Respiratory rate > 20/ minute or PaCO2 < 32 mmHg (1st sign – Count for full 60 secs.)

- WBC > 12,000, < 4000, or > 10% immature (band) forms (non-pregnant value)
- **Preg:** 5 K – 12 K
- **Labor:** up to 14 – 16 K
- **Late labor and PP:** up to 25 K
- **Post C/S:** up to 30 K
- Acute change in LOC
- Glucose > 120mg/ dl in non-diabetic

Part 2:

Infection suspected or confirmed

Part 3:

Acute (new) Organ Dysfunction

Part 4:

Severe/ Septic Shock

50 / 50 Life or Death.

Multiorgan Dysfunction

Presence of altered organ function requiring medical intervention to maintain hemostasis.

Management:

Early Goal Directed Therapy

To be completed within 3 hours

1. Measure lactate level
2. Obtain blood cultures prior to administration of antibiotics
3. Administer broad spectrum antibiotics
 - * 7.6% increase in mortality for every hour delay in Abx. Administration (Kumar, 2006)
4. Administer 30 ml/kg crystalloid for hypotension or lactate ≥ 4 mmol/L
