Obstetrics & Gynecology
TEAM

Postpartum haemorrhage

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Red = important
Blue = dr notes
Green = extra

Postpartum Haemorrhage

Definition

Any blood loss than has potential to produce or produces hemodynamic instability

Excessive bleeding from genitalia that produce symptoms

Incidence

About 5% of all deliveries

The most common causes of maternal death:

1-PPH 2-PE 3-HTN

Hemodynamic unstable symptoms:

Pale, SOB, palpitation, weakness, headach, dizziness

Primary PPH: from 3rd stage of labor to 24h of delivery

Secondary PPH : after 24 h of delivery to 6 weeks

PPH diagnosis: seeing the bleeding and patient symptoms

we should observe the patient 2 h after the delivery because can shock and die in this time

Definition

- >500ml after completion of the third stage, 5% women loose >1000ml at vag delivery
- >1000ml after C/S
- >1400ml for elective Cesarean-hyst
- >3000-3500ml for emergent Cesarean-hyst
- woman with normal pregnancy-induced hypervolemia increases blood-volume by 30-60% = 1-2L
- therfore, tolerates similar amount of blood loss at delivery
- hemorrhage after 24hrs = late PPH

The patient can tolerate to certain limit, what are these limits ?

Why she can tolerate this amount of bleeding although it's consider high, and some people can't tolerate 300 ml? Due to the physiological changes that happened during pregnancy, she is already has high volume of fluids and blood

Hemostasis at placental site

Where do the bleeding come from ?
The placental site as well as vagina and perineum specially those who have episiotomy

- At term, 600ml/min of blood flows through intervillous space
- Most important factor for control of bleeding from placenta site = contraction and retraction of myometrium to compress the vessels severed with placental separation.
- Incomplete separation will prevent appropriate contraction

Etiology of Postpartum Haemorrhage

4 Ts (very imp)

Tone	Uterine atony 95%
Tissue (Retained part of placenta, Placenta berivia)	Retained tissue/clots
Trauma (Laceration of the vagina or cervix, uterine rupture)	laceration, rupture, inversion
Thrombin (Coagulation cascade should start immediately after delivery)	coagulopathy

Predisposing factors- Intrapartum (bleeding during the delivery)

- Operative delivery (as c-section)
- Prolonged or rapid labour

Depend on the time that we know, e.g: 1st stage in labor is

- Induction or agumentation (increase medication for induction)
- Internal podalic version

Definitions: maneuver to deliver the fetus by inserting a hand into the uterine cavity, grasping one or both feet, and drawing them through the cervix; rarely indicated today except for the delivery of a second twin.

- Choriomnionitis (infection)
- Shoulder dystocia
- coagulopathy

Prolong labor can lead to uterine atony

Predisposing Factors- Antepartum

- Previous PPH or manual removal
- Abruption/previa
- Fetal demise = (fetal death)
- Gestational hypertension
- Over distended uterus
- Bleeding disorder

Postpartum causes

- Lacerations or episiotomy
- Retained placental/ placental abnormalities
- Uterine rupture / inversion
- Coagulopathy

Prevention

- Be prepared
- Active management of third stage
  - Prophylactic oxytocin
  - 10 U IM
  - 5 U IV bolus
  - 10-20 U/L N/S IV @ 100-150 ml/hr
  - Early cord clamping and cutting
  - Gentle cord traction with surapubic countertraction

Remember!

- Blood loss is often underestimated
- Ongoing trickling can lead to significant blood loss
- Blood loss is generally well tolerated to a point

Management

- talk to and assess patient
- Get HELP!
- Large bore IV access
- Crystalloid-lots!
- CBC/cross-match and type
- Foley catheter

Diagnosis

- Assess in the fundus (Palpate the abdomen to see if the uterus contacted or not)
- Inspect the lower genital tract (Check externally : vagina, episiotomy then internally: uterus, cervix)
- Explore the uterus
  - Retained placental fragments
  - Uterine rupture
  - Uterine inversion
- Assess coagulation

If there is still bleed check the 4Ts

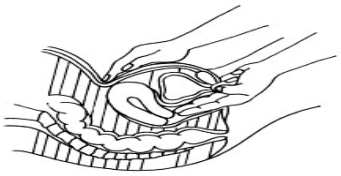
Then we check the uterine contraction and the pads for regular bleeding

Symptoms of placental delivery:
Gush of blood
Elongated umbilical cord
Change the uterus shape (contraction)

Management: 1- Assess the fundus

- If she still bleed we resuscitate
- Simultaneous with ABC's
- Atony is the leading case of PPH
- Bimanual massage
- Rules out uterine inversion
- May feel lower tract injury
- Evacuate clot from vagina and/ or cervix
- May consider manual exploration at this time

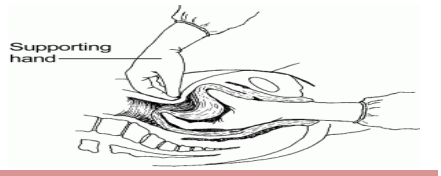
Management: 2- Bimanual Massage



Management- Manual Exploration

- Manual exploration will:
  - Rule out the uterine inversion
  - Palpate cervical injury
  - Remove retained placenta or clot from uterus
  - Rule out uterine rupture or dehiscence

Replacement of Inverted Uterus →



Management: 3- Oxytocin

- 5 units IV bolus
- 20 units per L N/S IV wide open
- 10 units intramyometrial given transabdominally

Management: 4-Additional Uterotonics

- Ergometrine (caution in hypertension)
  - .25 mg IM 0r .125 mg IV
  - Maximum dose 1.25 mg
- Hemabate (asthma is a relative contraindication)
  - 15 methyl-prostaglandin F2 alfa
  - O.25mg IM or intramyometrial
  - Maximum dose 2 mg (Q 15 min- total 8 doses)
- Cytotec (misoprostol) PG E1
  - 800-1000 mcg pr

TABLE 3

DRUG THERAPY FOR PPH

Drug	Dose	Side Effects	Contraindications
Oxytocin	10 units IM/IMM 5 units IV bolus 10 to 20 units/litre	Usually none painful contractions nausea, vomiting, (water intoxication)	hypersensitivity to drug
Methylergonovine maleate	0.25mg IM/0.125mg IV repeat every 5 mins as needed maximum 5 doses	peripheral vasospasm hypertension nausea, vomiting	hypertension hypersensitivity to drug
Carboprost (15-methyl PGF_2 alpha)	0.25 IM/IMM repeat every 15 mins as needed maximum 8 doses	flushing, diarrhea, nausea, vomiting bronchospasm, flushing, restlessness, oxygen desaturation	active cardiac, pulmonary, renal, or hepatic disease hypersensitivity to drug
Vasopressin	20 units diluted in 100 ml normal saline = (0.2 units/ml) inject 1 ml at bleeding site avoid intravascular injection	acute hypertension, bronchospasm nausea, vomiting, abdominal cramps angina, headache, vertigo death with intravascular injection	coronary artery disease hypersensitivity to drug

Management: 5- Bleeding with Firm Uterus

- Explore the lower genital tract
- Requirements
  - Appropriate analgesia
  - Good exposure and lighting
- Appropriate surgical repair
  - May temporize with packing

Management: 6- ABC's

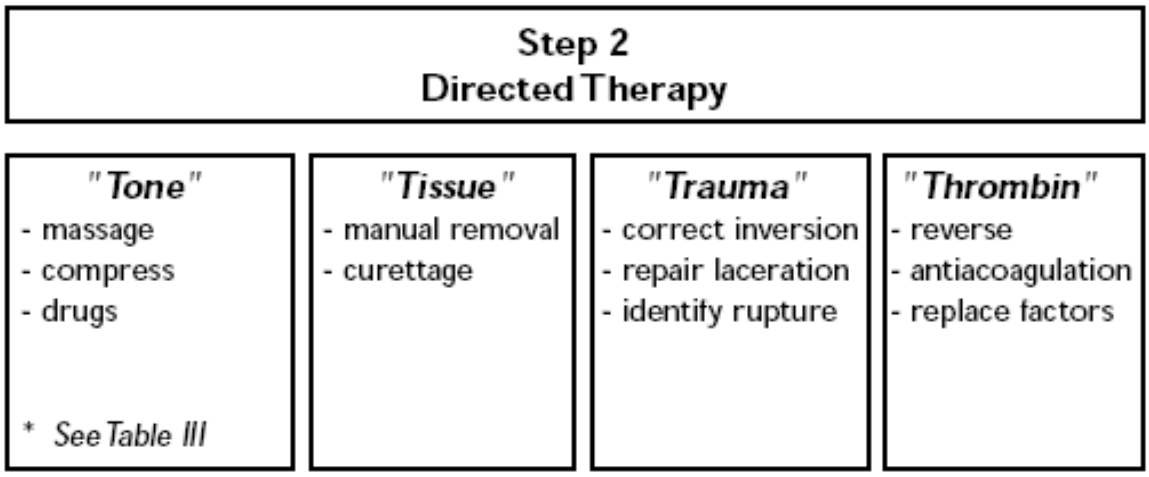
ENSURE THAT YOU ARE ALWAYS AHEAD WITH YOUR RESUSCITATION!!!!

- Consider need for Foley catheter, CVP, arterial line, etc.
- Consider need for more expert help

Management: 7- Evolution

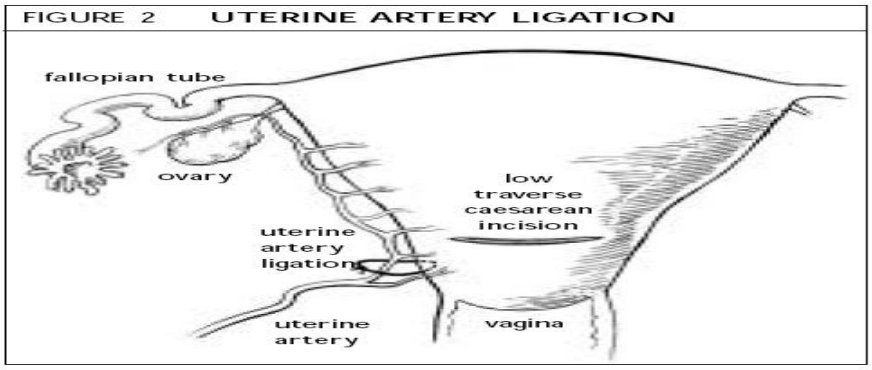
- Panic
- Panic
- Hysterectomy
- Pitocin
- Prostaglandins
- Happiness

MANAGEMENT OF PPH



Management- Continued Uterine Bleeding

- Consider coagulopathy
- Correct coagulopathy
  - FFP, cryoprecipitate, platelets



- If coagulation is normal:
  - Consider embolization
  - Prepare for O.R

Surgical Approaches

- Uterine vessel ligation
- Internal iliac vessel ligation
- Hysterectomy

Conclusions

- Be prepared
- Practice prevention
- Assess the loss
- Assess the maternal status
- Resuscitate vigorously and appropriately
- Diagnose the cause
- Treat the cause

Summary: Remember 4 Ts

- Tone
- Tissue
- Trauma
- Thrombin

TONE: Rule out Uterine Atony :

- Palpate fundus.
- Massage uterus.
- Oxytocin
- Methergine
- Hemabate

Tissue: R/O retained placenta:

- Inspect placenta for missing cotyledons.
- Explore uterus.
- Treat abnormal implantation.

TRAUMA: R/O cervical or vaginal lacerations.

- Obtain good exposure.
- Inspect cervix and vagina.
- Worry about slow bleeders.
- Treat hematomas

THROMBIN

- Check labs if suspicious**.**

CONSUMPTIVE COAGULOPATHY (DIC)

- A complication of an identifiable, underlying pathological process against which treatment must be directed to the cause

Pregnancy Hypercoagulability

dr didn't read the rest

- ↑ coagulation factors I (fibrinogen), VII, IX, X
- ↑ plasminogen; ↓ plasmin activity
- ↑ fibrinopeptide A, b-thromboglobulin, platelet factor 4, fibrinogen

-Pathological Activation of Coagulation mechanisms

- Extrinsic pathway activation by thromboplastin from tissue destruction
- Intrinsic pathway activation by collagen and other tissue components
- Direct activation of factor X by proteases
- Induction of procoagulant activity in lymphocytes, neutrophils or platelets by stimulation with bacterial toxins

-Significance of Consumptive Coagulopathy

- Bleeding
- Circulatory obstruction→organ hypoperfusion and ischemic tissue damage
- Renal failure, ARDS
- Microangiopathic hemolysis

-Causes

- Abruptio placentae (most common cause in obstetrics)
- Sever Hemorrhage (Postpartum hge)
- Fetal Death and Delayed Delivery >2wks
- Amniotic Fluid Embolus

Septicemia

- Treatment
- Identify and treat source of coagulopathy
- Correct coagulopathy
    - FFP, cryoprecipitate, platelets

Fetal Death and Delayed Delivery

- Spontaneous labour usually in 2 weeks post fetal death
- Maternal coagulation problems < 1 month post fetal death
- If retained longer, 25% develop coagulopathy
- Consumptive coagulopathy mediated by thromboplastin from dead fetus
- tx: correct coagulation defects and delivery

Amniotic Fluid Embolus

- Complex condition characterized by abrupt onset of hypotension, hypoxia and consumptive coagulopathy
- 1 in 8000 to 1 in 30 000 pregnancies
- “anaphylactoid syndrome of pregnancy
- Pathophysiology: brief pulmonary and systemic hypertension→transient, profound oxygen desaturation (neurological injury in survivors) → secondary phase: lung injury and coagulopathy
- Diagnosis is clinical
- Management: supportive
- Prognosis:60% maternal mortality; profound neurological impairment is the rule in survivors
- fetal: outcome poor; related to arrest-to-delivery time interval; 70% neonatal survival; with half of survivors having neurological impairment

Septicemia

- Due to septic abortion, antepartum pyelonephritis, puerperal infection
- Endotoxin activates extrinsic clotting mechanism through TNF (tumor necrosis factor)
- Treat cause

Abortion

Coagulation defects from:

- Sepsis (*Clostridium perfringens* highest at Parkland) during instrumental termination of pregnancy
- Thromboplastin released from placenta, fetus, decidua or all three (prolonged retention of dead fetus)

Kaplan notes:

1- Uterine atony :

- Most common cause of PPH
- **Risk factors :** rapid or protracted labor, chorioamnionitis, medication ($MgSO_4$, B-adrenergic agonists), and overdistended
- **Clinical findings:** soft uterus palpable above the umbilicus
- **Management:** uterine massage and uterotonic agents (e.g: oxytocin, methylergonovine, carboprots)

2- Lacerations :

- **Risk factors :** uncontrolled vaginal delivery and operative vaginal delivery.
- **Clinical findings:** identifiable laceration (cervix, vagina, perineum) in the presence of contracted uterus.
- **Management :** surgical repair

3- Retained placenta:

- **Risk factors:** accessory placental lobe and abnormal trophoblastic uterine invasion (cervix, vagina, perineum)
- **Clinical findings:** missing placenta cotyledons in the presence of contacted uterus
- **Management :** manual removal or uterine curettage under US guidance.

4- Disseminated intravascular coagulation:

- **Risk factors:** abruption placenta, sever preeclampsia, aminiotic fluid emberiolism, and prolonged retention of dead fetus.
- **Clinical findings:** generalized oozing or bleeding from IV sites or lacerations in the presence of contracted uterus
- **Management:** removal of pregnancy tissue from the uterus, ICU support, and selective blood- product replacement

5- Uterine inversion:

- **Risk factors:** myometrial weakness and previous uterine inversion
- **Clinical findings :** beefy appearing bleeding mass in the vagina and failure to palpate the uterus abdominally
- **Management:** uterine replacement by elevating the vaginal fornices and lifting the uterus back into its normal anatomic position, followed by IV oxytocin