

O & G Summary Notes

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| 1. Changes in Pregnancy | 5. PID |
| 2. Bleeding in 1st Trimester | 6. Hypertension in Pregnancy |
| 3. Bleeding after 1st trimester | 7. Pelvic pain in non-pregnant patient |
| 4. Bleeding in non-pregnant patient | |

Changes in Pregnancy

Airway

Breathing

- There is increased maternal carbon monoxide in the first 10 weeks

Circulation

- Blood volume increases by 45% by the end of the third trimester
- Increased resting heart rate by 15-20 bpm by end of 3rd trimester
- Decrease in systolic and diastolic blood pressure by 10-15 mmHg by the second trimester then an increase
- There may be IVC obstruction after 20 weeks

Labs

FBC

- Leucocytosis in the third trimester
- Dilutional Anaemia
- Decrease in haematocrit by 35% by the end of pregnancy
- An increase in ESR by the third trimester

EUC

- A decrease in the buffer and the capacity of the blood
- **beta hCG**
 - o Formed from the outer layer of cells of the gestation sac.
 - o Detected at nine days after fertilisation, $t_{1/2}=48$ hrs
 - o Increases ~ 1.66 times / 48 hours, early in pregnancy. then plateaus and falls at ~12 weeks.

LFT

- There is relative bilirubinaemia

COAG

- Increase in coagulation factors and increased risk of venous thrombosis

Urine

- **urinary hCG**

Urine pregnancy tests are sensitive when the beta hCG level is > 25 IU/L

ECG

- ECG changes may include t wave inversion and t wave flattening

Bleeding in the 1st Trimester

This affects approximately 25% of all diagnosed pregnancies. The cause of this bleeding ranges from physiological bleeding, to threatened miscarriage, failed pregnancy, ectopic pregnancy and molar pregnancy.

Ectopic Pregnancy

This is any pregnancy that is implanted outside of the normal uterine cavity. The most common location of an ectopic pregnancy is the fallopian tube. It can also occur in the cervix, ovary, abdomen and rarely in uterine scar.

The rate of ectopic in subsequent pregnancies is 25 to 30%.

Heterotopic pregnancy

This is when an intrauterine pregnancy coexist with the ectopic pregnancy.

The incidence is 1:3889 In the general population, But increases to 1:100-1:500 In those that have had fertility treatment.

Ultrasound in ectopic

Transvaginal ultrasound.

A gestation or sac is identified at 31 days (4 weeks) gestation. A yolk sac within the gestational sac is seen at 5 to 6 weeks, when the beta hCG is 1500 IU/L

Cardiac activity is seen at 39 days (5.5 weeks) gestation. ***Trans abdominal ultrasound***

It identifies the pregnancy at about six weeks, when the beta HCG is approximately 6500 IU per litre.

Haemodynamically unstable ectopic pregnancy

Operating room

Haemodynamically stable ectopic pregnancy

These patients can be observed, especially if they have a low beta hCG e.g. (<1000 IU/L) And it is falling.

Medical management involves the use of intramuscular methotrexate. Selection criteria for methotrexate use include:

1. A non-tubal ectopic pregnancy
2. Tubal ectopic pregnancy less than 3 cm
 - No cardiac activity
 - bhCG < 5000 IU /L

Failed Pregnancy (Miscarriage)

This is defined as an embryo greater than 6 mm with no cardiac activity. Foetal death is where a foetus of more than eight weeks has no cardiac activity. A Pregnancy with no embryo i.e., blighted ovum, is a gestational sac of greater than 20mm with no embryo. A failed pregnancy may progress to an incomplete or complete miscarriage, or may remain in the uterus.

Approximately 50% of patients with bleeding in early pregnancy will proceed to term.

Patients with early pregnancy bleeding but a normal ultrasound confirming live intrauterine gestation have an 85 to 90% chance of pregnancy progressing to term. Indicators of a poor prognosis include advanced maternal age, ultrasound findings of a large sack and foetal bradycardia after seven weeks gestation.

The unstable patient Resuscitation techniques must commence as normal in the unstable patient. Perspective on examination needs to be conducted in these patients, to remove any

products of conception that cause dilatation of the cervix. Products of conception dilating the cervix can result in neurocardiogenic or cervical shock.

In those patients where products of conception have been removed and bleeding continues, uterine contraction may be induced by administering ergometrine 20mcg IM. Patients who continue to bleed will need surgical evacuation of the uterus.

The stable patient These patients require surgical intervention and removal of the retained products of conception. Conservative management has been proposed, where there is a spontaneous expulsion. An alternative approach is to use misoprostol to induce uterine contraction.

In patients who are discharged home advise should include:

- no sexual intercourse
- no tampon use
- bed rest has not been shown to alter the course.

In patients with an ultrasound finding of gestational sac < 20 mm and no yolk sac should have serial beta hCG and a repeat ultrasound to exclude a pseudo sac.

Rh(D) Immunoglobulin

In early pregnancy bleeding

In a Rh-negative woman with a

- singleton pregnancy 25IU of Rh(D) immunoglobulin should be given
- multiple pregnancies or gestation >13 weeks, 625 IU of Rh(D)

The Kleihauer test can be used later in pregnancy to identify numbers of foetal cells in maternal circulation. It is unreliable in early pregnancy

Bleeding after the first trimester

4% of gravid females have bleeding after 20 weeks gestation.

Antepartum Haemorrhage

Antepartum haemorrhage is bleeding that occurs after 20 weeks gestation.

Causes include bleeding from the **lower genital tract** which may include traumatic bleeding post coitus or other bleeding from physiological cervical erosion or from cervical polyps or malignancy or vaginal infections.

- **Placenta praevia** 0.5% of pregnancies. It is usually associated with painless bleeding but can bring on labour type contractions. It occurs when the placenta is in the lower uterus and thus before the presenting part.
- **A marginal bleed** can occur when there is bleeding from the edge of the placenta.
- **Placental abruption** is the separation of the placenta.

Placental abruption may follow relatively minor blunt trauma such as a fall onto the abdomen. It can also occur spontaneously in patients with disorders such as hypertension or coagulation issues or cocaine use. Bleeding from the central separation occurs from behind the placenta. A retroplacental clot can contain up to 4 L of maternal blood without any vaginal loss. The rate of fetal compromise and fetal death in a significant placental attachment approach is 30%.

Only 50% seen on ultrasound, so it is a clinical diagnosis.

Vasa praevia is the presence of foetal vessels that run in the amniotic membrane distal to the placenta. If these vessels rupture, they usually do show in association with rupture of the amniotic membranes. Bleeding from these vessels is foetal bleeding and can result in rapid foetal compromise. Cardiotocography is very sensitive in picking this up, especially through foetal bradycardia.

Physiological bleeding is usually called the 'show'. It is vaginal blood mixed with mucus. It results from the mucus plug or operculum within the endocervical canal dislodging as the cervix begins to dilate. This occurs around the time of labour and is not significant unless the pregnancy is pre term or associated with rupture of the membranes.

Post Partum Haemorrhage(PPH)

It is defined as blood loss of >500mL after fetus delivery.. It can also be defined clinically as blood loss resulting in haemodynamic compromise of the patient, or the patient becoming symptomatic of hypovolaemia or hypotension ie., lightheaded, syncope etc..

Causes include:

- **Retained products of conception**
- **Uterine atony-**
 - use oxytocin 5U IV then an infusion of 20-40U in 1L Normal Saline at 250mL/h.
 - Can also use misoprostol 800-1000mcg PR and/or ergometrine 250 mcg iv/im
 - In refractory uterine atony intramyometrial PF F2a 250-500mcg to a maximum of 2mg is successful in up to 85% of cases.
 - If persists may need exploration of uterine cavity
- **Soft tissue laceration**
- **Coagulopathy**
- **Uterine Rupture- Associated with severe abdominal pain**
- **Uterine Inversion**

Those at greatest risk include

- those with a previous history of PPH, or antepartum haemorrhage
- Those with multiple pregnancies and large placenta site
- Those with retained placenta

- Those with a distended uterus- polyhydramnios, multiple pregnancy
- Grand Multiparity(reduced muscular tissue)
- Where the labour is prolonged or induced.
- Where tocolytics have been used

Surgical Management of PPH

If simple procedures and medications don't work then a laparotomy is required and uterine artery ligation, or uterine compression sutures may be used. Hysterectomy may be required in atony, rupture or placenta praevia, but it is the procedure of last resort.

Uterine tamponade may be another short term procedure that involves the Bakri tamponade balloon.(500mL capacity) If this is not available a Sengstaken-Blakemore tube can be used.

Secondary Post Partum Haemorrhage

Excessive or prolonged bleeding from 24hrs to 6 weeks post partum.

Normal lochia (blood, mucus and uterine tissue) is initially moderately heavy for some days and settles to light bleeding by 2-4 weeks. In some cases there is a brownish vaginal discharge for up to 8 weeks.

Common Causes include:

- retained products of conception
- endometritis

Less common causes

- Uterine AVM
- Trophoblastic disease
- Bleeding restarts form episiotomy etc.

Ultrasound to look for retained products and just incase an AVM is found.

Treat empirically with Augmentin for 5-7 days.

Small amounts of retained products can be treated conservatively. If uterine curette is needed it is associated with risk of uterine perforation or Asherman Syndrome(intrauterine adhesions or fibrosis).

Abnormal Vaginal Bleeding in Non-Pregnant Patient.

Its good to think of this in terms of anatomy ie., where the bleeding is coming from, external genitalia, vagina, cervix or uterus. The causes can be traumatic, infective, physiological or pathological.

Physiological Uterine Bleeding

This is the menstrual cycle, controlled by the hypothalamic-pituitary-ovarian axis. It occurs at regular intervals for 3-7 days. Average blood loss is 30-40mL. When the blood loss is >80mL it is defined as menorrhagia.

Oestrogen acting on the pituitary results in the production of FSH(follicle stimulating hormone) and LH(lutenising hormone) which act on the ovary to result in ovulation. The corpus luteum(CL) produces progesterone>oestrogen. If fertilization doesn't occur, there is involution of the CL, a decrease in progesterone and oestrogen and the resultant vasoconstriction of the endometrium, which results in ischaemia and a shedding of the endometrium, which is normal menstrual bleeding.

Pathological Uterine Bleeding

This can be infective, or due to fibroids or malignancy, or arteriovenous malformations.

The most common cause is menorrhagia occurring in ovulatory menstrual cycles.

Treatment includes Tranexamic acid 1g tds for 3-4 days.

Mefenamic acid 500mg tds, Naproxen 250mg tds and ibuprofen 400mg tds.

NSAIDs block PGE2- a vasodilator and found in excess in patients with menorrhagia.

When it occurs due to anovulatory menstrual cycles (dysfunctional uterine bleeding), the patient presents with irregular bleeding of variable volume. Anovulatory menstrual cycles are usually due to high oestrogen in comparison to low progesterone. This results in hyperplasia/metaplasia of the endometrium and it is unstable resulting in erratic sloughing.

Treatment includes:

- progestin therapy to stabilize the endometrium
 - o norethisterone 5-10mg- up to tds, tapering to 5-10 mg per day over 2-3 weeks.
 - o Medroxyprogesterone acetate 10-30mg daily, reducing to 10mg daily over 2-3 weeks.
- This can be combined with tranexamic acid and/or NSAID
- Combined OCP can be used to decrease blood loss in ovulatory cycles and regulate anovulatory cycles.
 - o OCP with ethinyloestradiol and a progestin

In patient > 35yo take an endometrial sample prior to commencing hormones.

Investigate with betahCG, coagulation profile(in all adolescents and those with heavy uterine bleeding). Consider TFT's in women with menorrhagia and anovulatory bleeding.

Get an ultrasound to look for fibroids and AVM or polyps etc.

Most patients can be discharged home.

Pelvic Inflammatory Disease (PID)

It is a clinical syndrome resulting from inflammation or infection. Usually caused by ascent of organisms via the vagina to the upper genital tract. It includes endometritis, salpingitis, tubo-ovarian abscess and/or pelvic peritonitis.

The organisms involved are:

- N.gonorrhoeae and
- C. trachomatis

Risk factors include sexually transmitted diseases and anything that disrupts the normal cervical barrier ie., dilatation and curettage and childbirth.

Chlamydia is the most common cause of sexually transmitted PID.

Abdominal pain of less than 3 weeks duration, abnormal vaginal discharge, dyspareunia are important findings in history. Adnexal tenderness is the most sensitive examination finding(95%) Other findings that are important are; lower abdominal tenderness, uterine tenderness and cervical motion tenderness.

Investigations include basic bloods and endocervical swabs. The chance of PID is low if there is clear cervical discharge and there are no WBC on the wet slide preparation.

Ultrasound cannot diagnose moderate to OIS, but is useful in identifying tubo-ovarian abscess and looking for other causes of pelvic pain.

If the patient is not septicaemic or unwell, outpatient treatment is considered appropriate.

Treatment (in proven cases of PID treat partners)

Sexually acquired PID

Mild to moderate infection: Azithromycin 1g po(single dose) + doxycycline 100mg bd for 14 days + metronidazole 400mg po bd for 14 days + if suspect gonorrhoea add ceftriaxone 250mg IV/IM as single dose.

Severe infection: doxycycline 100mg po/iv bd + metronidazole 500mg iv bd + ceftriaxone 1g iv daily

Non-sexually acquired PID

Mild to moderate infection: Augmentin 875/125 bd 14 days + doxycycline 100mg bd, 14 days

Severe infection: Amoxicillin/ampicillin 2g iv qid + gentamycin 4-6 mg/kg daily + metronidazole 500mg iv bd

What is Fitz-Hugh-Curtis syndrome ? Also called peri-hepatitis.

It is a peri-hepatitis secondary to transcoelomic spread of inflammatory peritoneal fluid to the sub-phrenic and sub-diaphragmatic spaces.Suspect it in a patient with PID, no known biliary disease and right upper quadrant pain.

Hypertension and Pregnancy

Renin-angiotensin-aldosterone system is activated in normal pregnancy. The result is vasoconstriction. However small vessels to vasopressors have reduced sensitivity to vasopressors and there is the release of systemic and renal vasodilators.

Blood pressure falls after conception and returns to normal in third trimester. The BP falls again after delivery and returns to pre-pregnancy levels by 5th post-partum day.

During this time, plasma volume increases by 50%.

In the pre-eclamptic pregnancy

- -placental vascularization is incomplete and abnormal, where there is a risk of utero-placental hypoperfusion. and
- -maternal circulation is volume depleted and has a high vasomotor tone resulting in hypo-perfusion of target organs. This is the direct result of the Renin- Angiotensin-Aldosterone System not being activated and the release of factors such as endothelin and thromboxane that lead to increased vasomotor tone.

Pre-eclampsia and eclampsia

It is one of the hypertensive disorders of pregnancy and is characterised by hypertension, proteinuria or other evidence of end organ dysfunction.

Note that patients may have gestational; hypertension in pregnancy without proteinuria or end organ dysfunction.

Pre-eclampsia and eclampsia increase maternal morbidity and mortality by 400 times, compared to normal pregnancy.

Foetal mortality is 2% in pre-eclampsia, but increases to up to about 12% in eclampsia.

Pre-eclampsia: Typical time of onset

-20/40 and 48hrs post partum

HT + proteinuria and/or organ dysfunction

DBP of 90mmHg on 2 occasions or single 110mmHg

Proteinuria 1+ on dipstick correlates with >300mg per 24h

Oedema- no longer a defining feature

Remember that in about 16% with pre-eclampsia symptoms there is no hypertension.

Severe-Pre-eclampsia: At least one of following is present:

-severe proteinuria > 5g/24hr

-SBP > 160mmHg

-DBP >110mmHg

-Fetal growth retardation

Eclampsia: This is the seizures in pre-eclampsia are secondary to cerebral ischaemia, secondary to vasospasm.

What are the risk factors for eclampsia?

- Gestational HT
- Primiparity
- Past history of same
- Renal disease
- Diabetes
- Hypercoagulable states: antiphospholipid synd with anticardiolipin antibodies, lupus anticoagulant or both, Factor V Leiden Deficiency
- Large placenta

- Multiple gestations

There isn't much of a role for prevention, although in patients admitted for severe pre-eclampsia with symptoms ie., headache and blurred vision, it is advisable to treat with Magnesium Sulphate, as number needed to treat is 16. If no symptoms, NNT is 185.

Manifestations of Eclampsia:

Neurological: Headache, blurred vision, hyper-reflexia and altered conscious state. Generalised tonic clonic seizures occur mostly(90%) after 29 weeks. 44% occur in the post partum period.

Pulmonary Oedema: APO occurs secondary to pulmonary capillary leakage

Hepatic Syndrome: There is a range of damage, from simple raised liver enzymes, to subcapsular bleeding to elevated liver enzymes and low platelets, which is HELLP Syndrome. HELLP syndrome defines severe pre-eclampsia and is accompanied by severe HT, renal failure and DIC.

(HELLP Syndrome = Haemolysis, elevated liver enzymes, low platelet count.)

Renal Dysfunction: In normal pregnancy the creatinine falls as the GFR increases. In renal involvement, the GFR falls by up to 40% and results in a rise in creatinine, uric acid and calcium. Serum uric acid, is a marker for pre-eclampsia as abnormally high levels, predate the clinical manifestations of pre-eclampsia.

Treatment

Beware when treating not to cause hypoperfusion as there is no autoregulation of uteroplacental circulation and there is a volume depleted maternal circulation.

Ranges to aim for 140-160/90-110, although no consensus..

No real consensus on where to start buy SBP>170 or DBP > 110

Hydralazine (peripheral arteriolar vasodilator that improves uterine blood flow. 2.5-5mg doses every 15 min up to max of 10 mg.

Treatment of eclampsia is with Magnesium sulphate 6g iv (24mmol)over 15 min then a maintenance dose of 2g/hr. Magnesium lowers BP by vasodilatation.

Delivery of fetus and placenta is considered essential in eclampsia and pre-eclampsia complicated by HELLP Syndrome.

Pelvic Pain in the Non- Pregnant patient.

Adnexal mass or cyst

Rupture of cyst

Corpus Luteum cyst rupture occurs between 20-26 days of menstrual cycle and is associated with intra-peritoneal bleeding.

Intra-ovarian haemorrhage- includes haemorrhage into a cyst or tumour. Haemorrhagic ovarian cysts can be managed conservatively.

Torsion of adnexae (ovary and fallopian tubes). Occurs in 3rd decade of life. Adnexal torsion is associated with cystic tumours or simple cysts of the ovary in >90% of cases.

Cyclic Pelvic Pain

In up to 50% of women. Usually related to ovulation and menstruation.

- **Mittelschmerz**- transient mid-cycle pain, at or after ovulation
- **Endometriosis**- Caused by ectopic endometrial glands and stroma outside the uterine cavity. Initially pain is cyclic, but may develop into constant pain. Affects women 20-45 yo. >70% are nulliparous. Pain commences a few days prior to menses and continues beyond it.
- **Adenomyosis**; Benign. Ingrowth of endometrial glands and stroma into the myometrium. In >80% of cases, women are multiparous. Presenting complaints are menorrhagia and dysmenorrhea.
- **Leiomyomata** (fibroids): Benign, with origin in the myometrium.
- **Dysmenorrhoea**
 - o Primary: Painful menstruation and no pathology.
 - o Secondary Dysmenorrhoea: Painful menstruation associated with pelvic pathology.

Acyclic Pelvic pain

- **Pelvic Adhesions**: Associated with PID, endometriosis, abdominal surgery, perforated appendix and IBD.
- **Pelvic Congestion Syndrome**: It is associated with multiparity, polycystic ovarian syndrome tubal ligation and lower limb varicosities. Patients have pelvic and lower back pain, dyspareunia, dysfunctional uterine bleeding and mucoid vaginal discharge. It is caused by dilatation, congestion and stasis of the pelvic veins.