حسب الفيديو 17

Numb disease
* Epid/ Body parts
* Findings
* DX
* TX, subs

Placental disease

Normally

- Trophoblast cells invade the spiral arterioles within the first 12 weeks of pregnancy and replace the smooth muscle of the wall of the vessels, thus converting them to wide bore, low resistance, large capacitance vessels
- This process is normally complete by 20 weeks gestation
- The maternal blood flow to the placenta increases throughout pregnancy from 50 mL/min in the first trimester to 500–750 mL/min at terms

In PET

- There is a complete or partial failure of trophoblast invasion of the myometrial segments of the spiral arteries.
- Spiral arteries retain some of their pre-pregnancy characteristics being relatively narrow bore and of low capacitance and high resistance and resulting in impaired perfusion of the fetoplacental unit

Definition

- **pre-eclampsia: hypertension of at least 140/90 mmHg** recorded on at least two separate occasions and at least 4 hours apart and in the presence of at least 300 mg protein in a 24 hour collection of urine, **arising de novo after the 20th week of pregnancy in a previously** normotensive woman and resolving completely by the sixth postpartum week

Pathophysiology

- Placental bed biopsies have demonstrated that trophoblast invasion is patchy in pre-eclampsia and the spiral arteries retain their muscular walls.
- This is thought to prevent the development of a high flow, low impedance uteroplacental circulation. The reason why trophoblast invades less effectively in these pregnancies is not known but may reflect an abnormal adaptation of the maternal immune system.
- It is widely believed that defective trophoblast invasion results in relative under-perfusion of the placenta and that this releases a factor(s) into the maternal circulation that targets the vascular endothelium .
- The target cell of the disease process; the vascular endothelial cell
- pre-eclampsia is a truly multisystem disease, affecting multiple organ systems

(every system)

Cardiovascular system

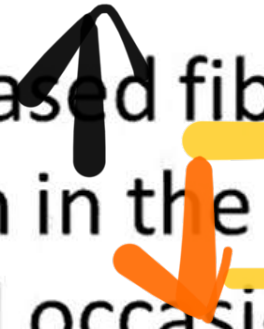
- Pre-eclampsia is characterized by marked peripheral vasoconstriction, resulting in hypertension. The intravascular high pressure and loss of endothelial cell integrity results in greater vascular permeability and contributes to the formation of generalized oedema.

Renal system

- A highly characteristic lesion called 'glomerulo/endotheliosis' 2 assoc. with
- Associated with impaired glomerular filtration and selective loss of intermediate weight proteins, such as albumin and transferrin, leading to proteinuria.
- Reduction in plasma oncotic pressure exacerbates the development of oedema.

Haematological system

- Endothelial damage; increased fibrin deposition and a reduction in the platelet count may accompany and occasionally predate the onset of disease.



Thrombocytopenia

↓

very bad prog.

Liver

- subendothelial fibrin deposition is associated with elevation of liver enzymes. This can be associated with haemolysis and a low platelet count due to platelet consumption (and subsequent widespread activation of the coagulation system), HELLP.
- HELLP syndrome is a particularly severe form of pre-eclampsia, occurring in just 2–4 per cent of women with the disease. It is associated with a high fetal loss rate (of up to 60 per cent)

*Some may come with normal BP & Absence of proteinuria → with time Completed pic of pet

CNS

Vasospasm and cerebral oedema have both been implicated in the pathogenesis of eclampsia. Retinal haemorrhages, exudates and papilloedema are characteristic of hypertensive encephalopathy and are rare in preeclampsia, suggesting that hypertension alone is not responsible for the cerebral pathology

Incidence

- Pre-eclampsia complicates approximately 2–3 per cent of pregnancies

Epidemiology

- Pre-eclampsia is more common in primigravid women
- the recurrence risk in a subsequent pregnancy is 20 per cent, but is much higher if severe pre-eclampsia developed at an extremely early gestation in the first pregnancy
- a three- to-four-fold increase in the incidence of pre-eclampsia in the first degree relatives of affected women

Risk factors

- First pregnancy
- Multiparous with pre-eclampsia in any previous pregnancy
- ten years or more since last baby
- Age 40 years or more
- Body mass index of 35 or more
- Family history of pre-eclampsia (in mother or sister)
- Booking diastolic blood pressure of 80 mmHg or more
- Booking proteinuria (of 1 on more than one occasion or quantifi ed at 0.3 g/24 hour)
- Multiple pregnancy
- Certain underlying medical conditions:
  - pre-existing hypertension
  - pre-existing renal disease
  - pre-existing diabetes
  - antiphospholipid antibodies

2ry : very bad.

Clinical presentation

- The classic symptoms of pre-eclampsia include a frontal headache, visual disturbance and epigastric pain.
- Majority of women with pre-eclampsia are asymptomatic or merely complain of general, vague 'flu-like' symptoms.
- Clinical examination should include a complete obstetric and neurological examination.
- Hypertension is usually the first sign, but occasionally is absent or transient until the late stages of the disease.
- Dependent oedema of the feet is very common in healthy pregnant women.
- Rapidly progressive oedema of the face and hands may suggest pre-eclampsia.

Clinical presentation

Epigastric tenderness is a worrying sign and suggests liver involvement.

Neurological examination may reveal hyperreflexia and clonus in severe cases.

Urine testing for protein should be considered part of the clinical examination

CO: ↓ Absorption

Screening and prevention

Unfortunately, there is currently no screening test for pre-eclampsia

The ability of Doppler ultrasound uterine artery waveform analysis to identify women at risk of pre-eclampsia (and other adverse pregnancy outcomes) has been investigated with varying success; useful in high but not in low risk cases.

Screening and prevention

- Low dose aspirin (75 mg); modestly reduces the risk of pre-eclampsia in high-risk women, and calcium supplementation may also reduce risk, but only in women with reduced dietary intake.
- Despite encouraging preliminary studies, vitamins C and E do not lower the risk of pre-eclampsia.

IUGR
< 34 w
early onset

* stop ACEI (Absolute contra), others are Relative

Tx

Management

The principles of management of pre-eclampsia are:

- early recognition of the asymptomatic syndrome;
- awareness of the serious nature of the condition in its severest form
- adherence to agreed guidelines for admission to hospital, investigation and the use of antihypertensive and anticonvulsant therapy
- well-timed delivery to avoid serious maternal or fetal complications
- post-natal follow up and counselling for future pregnancies.

Management

Same efficacy
differ in SE, cost, familiarity

- The aim of antihypertensive therapy is to lower the blood pressure and reduce the risk of maternal cerebrovascular accident without reducing uterine blood flow and compromising the fetus.
- There are a variety of antihypertensives used in the management of pre-eclampsia.
- Methyldopa is a centrally acting antihypertensive agent. It has a long established safety record in pregnancy. it can only be given orally, it takes upwards of 24 hours to take effect and has a range of unpleasant side effects; including sedation and depression.
- Labetalol is an alpha-blocking and betablocking agent. It too has a good safety record in pregnancy and can be given orally and intravenously.
- Nifedipine is a calcium-channel blocker with a rapid nset of action. It can, however, cause severe headache that may mimic worsening disease.

only

tachy, placental ↓BF

fantastic, in UK (Drug of choice)

→ CCB ↓SE
→ smooth (↓Rapid) ↓ in BP → ↓ placental BF (↑distress)

Management

- intravenous infusion of hydralazine or labetalol
- MgSO4 (5-6g IV slowly) (Drug of choice) → (as prophylax & tx) → Sedative / vasodilator / Anticonv
- intracranial haemorrhage; the most common cause of death
- In cases of serious multisystem complications, a multidisciplinary approach involving clinicians from other specialties (e.g. intensive care, haematology, nephrology) is essential.

CVS risk
pulm edema (100 ml/hr) low Rate
Myoc. depression
Maint. fluid
ICU
must be on observ
to Pr Mg toxicity
affect ↓ US RR Reflexes

Eg: Csec if: primi <34W, head is very high, cervix (long, thick closed) the only curative

Start with PG

of vagina

↳ for ripening

Mode of delivery

- Mode of delivery; less than 34 weeks, c-section. (Remember it's indication)
- Steroids
- Prophylactic anti-coagulation & stockings
- Epidural or spinal if normal clotting tests → must 80 or > platlet count
- ~~Ergometrine~~ is contra-indicated (cvz ↑ BP)
- If hypertension &/or proteinuria persist beyond 6 weeks, think of chronic hypertension or kidney disease
- Severe PET before 34 weeks; think of underlying causes

CV y confer