**Hypertensive disorders of pregnancy**

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**Objectives**

1. Define hypertensive disorders of pregnancy.
2. Classification of this disorder.
3. Maternal & fetal complications
4. Managements .

**Definition:**

Hypertension is defined as changes of BP recorded on at least 2 occasions of either:

* Diastolic BP >90 mmHg, or
* Systolic BP >140 mmHg, or
* A rise (compared to booking) in diastolic BP of at least 15 mmHg, or
* A rise (compare to booking) in systolic BP of at least 30 mmHg.

**Classification:**

* Pregnancy-induced hypertension (Gestational hypertension alone): hypertension arising for the first time in the second half of pregnancy &in the absence of proteinuria or any other features of pre-eclampsia.It is not associated with adverse pregnancy outcome.
* Chronic hypertension: pre-existing hypertension may be diagnosed before gestation or assumed when a women is found to be hypertensive in early pregnancy. It can predispose to the later development of superimposed pre-eclampsia.
* Pre-eclampsia: defined as hypertension of at least 140/90 mmHg recorded on 2 separate occasions at least 4 hours apart & in the presence of at least 300 mg protein in a 24-hour collection of urine, arising de nova after the 20th week of gestation in a previously normotensive women & resolving completely by the sixth postpartum week
* Eclampsia: is a serious & life-threatening complication of pre-eclampsia.It is defined as convulsions occurring in a woman with established pre-eclampsia,in the absence of any other neurological or metabolic cause. It is an obstetric emergency.
* It may occur:
	+ Antepartum 40%.
	+ Intrapartum 20%.
	+ Postpartum 40%.
* Imminent eclampsia (fulminating pre-eclampsia): is the transitional condition characterized by increasing symptoms & signs, it’s the sever form of pre-eclampsia

**Pre-eclampsia:**

**Incidence&Epidmiology:**

* It complicates approximately 3% of pregnancies.
* It is more common in primigravida (effect of fetal and hence paternal genome).
* Maternal genetic predispositions (3-4 folds increase in the first-degree relatives of affected women).
* Risk factors for pre-eclampsia(predisposing factors):

1. Conditions in which the placenta:-

 \*multiple gestation.

 \*diabetes.

 \*hydrops.

2. Pre-existing hypertension or renal disease.

3. Pre-existing vascular disease (such as in diabetes or autoimmune vasculitis).

**Clinical presentation:-**

* May be asymptomatic.
* Headache.
* Visual disturbances.
* Epigastric& right upper abdominal pain.

**Signs of pre-eclampsia:**

* Elevation of BP.
* Fluid retention (non-dependent oedema).
* Brisk reflexes.
* Ankle clonus (more than 3 beats).
* Uterus & fetus may feel small for gestational age.

**Etiology:**

* Trophoblastic tissue provides the stimulus for the disorder, so its only occurs in pregnancy ,but it has been described in pregnancy lacking a fetus(molar pregnancy)& in the absence of the uterus ( abdominal pregnancy) .
* Trophoblastic invasion is patchy& the spiral arteries retain their muscular walls which interne prevent the development of a high flow, low-impedance uteroplacental circulation, the reason for that is unknown.

**Organ-specific changes associated with pre-eclampsia:**

* **Central nervous system**
* Cerebral oedema.
* Cerebral hemorrhages.
* Retinal haemorrhage, exudates &papillodema are characteristic of hypertensive encephalopathy.
* **Cardiovascular**
* Generalized vasospasm.
* Increased peripheral resistance.
* Reduced central Venus/pulmonary wedge pressures.
* **Haematological**
* Platelet activation & depletion.
* Coagulopathy.
* Decreased plasma volume.
* Increased blood viscosity.
* **Renal**
* Proteinuria
* Decreased GFR(oliguria)
* Decreased urate excretion (increase serum uric acid).
* **Hepatic**
* Periportal necrosis
* Sub- capsular haematoma.
* Elevation of liver enzymes.
* **HELLP syndrome:** it is sever form of pre-eclampsia, occure in 2-4% of women with pre-eclampsia&is associated with fetal loss rate of up to 60% if occur antenatally& a maternal mortality of up to 24%.
* It may be associated with DIC&placental abruption.
* H=Haemolysis.
* EL=Elevated Liver enzymes.
* LP=Low Platelet count.

**DIAGNOSIS:**

A diagnosis of pre-eclampsia usually requires admission of the patient for more intensive investigations & monitoring of her condition.

1.) Mild form:

* BP mildly elevated i.e. diastolic BP of 90-95 mmHg.
* Minimal proteinuria.
* Normal haematological&biochemical parameters.
* Patient can be monitored as an outpatient, attending for regular fetal & maternal assessment.

2.) Moderate (95-105mmHg), it requires admission to the hospital for investigation &follow up.

3.) Sever pre-eclampsia is identified by

* symptoms of sever pre-eclampsia:-
* Frontal headache
* Visual disturbance
* Epigastric pain
* General malaise & nausea
* Restlessness
* Signs of sever pre-eclampsia:
* Agitation
* Hyper- reflexia(clonus)
* Facial &peripheral odema
* Right upper quadrant tenderness
* Poor urine output

**Investigation for pre-eclampsia:**

These investigations will be repeated at interval depending on the overall clinical picture.

* Urinalysis by dipstick (quantitatively inaccurate).
* 24-hour urine collection for total protein &creatinine clearance).
* Full blood count (platelets &haematocrit).
* Blood chemistry (renal function , protein concentration ).
* Plasma urate concentration.
* Liver function.
* Coagulation profile.
* Ultrasound assessment :
	+ Fetal size.
	+ Amniotic fluid volume.
	+ Doppler.

**Complications:**

***MATERNAL COMPLICATIONS***

**Increase maternal morbidity &mortality because of:**

* Cerebral oedema, cerebral haemorrhage& retinal haemmorrhage.
* Heart failure & pulmonary oedema.
* Sub-capsular haematoma, periportal necrosis& elevated liver enzymes.
* Hematological complications:
	+ Decrease platelets count.
	+ Haemolysis.
	+ Coagulopathy & DIC.
* Renal failure.
* HELLP syndrome.
* Increase risk of thrombosis (DVT, pulmonary embolism).
* Increase risk of APH & PPH.
* Increase risk of surgical interventions(c/s, instrumental delivery).
* Eclampsia.
* Adult Respiratory Distress Syndrome (ARDS).

***Fetal complications:***

* Increase perinatal morbidity & mortality.
* Preterm delivery (iatrogenic).
* IUGR.
* IUD.
* Birth asphyxia.

**Treatment:**

* The mainstay of treatment is ending the pregnancy by delivering the fetus & placenta; this can be significant problem at 24-32 weeks.
* The aim of antihypertensive therapy is to lower the BP & reduce the risk of maternal cerebrovasular accident without reducing uterine blood flow & compromising the fetus.
* **Antihypertensive drugs are:**
* Methyldopa: centrally acting antihypertensive agent, safe, can only giving orally ,need at least 24 hours to work, & it is the drug of choice antenatally.
* Labetolol: is an alpha & beta- blocking agent ,it can be given orally or IV, safe , can be given antenatally&intrapartum to control BP in sever pre-eclampsia .
* Nifedipine : calcium-channel blocker with a rapid onset of action.It can, however , cause sever headache that mimic worsening disease.
* Hydralazine : arterial vasodilator , used IV in sever pre-eclampsia.
* So sever form of pre-eclampsia , IV infusion of hydralazine/labetolol can be titrated rapidly against changes in the BP.
* **Management of eclampsia :**
* Maintain an open air way by mouth piece & oxygen .
* maintain an 2 IV line & take blood samples for :
	+ Blood group &Rh.
	+ CBC & Blood film.
	+ LFT
	+ RFT
	+ Serum uric acid.
	+ Coagulation profile.
* Control fit by giving magnesium sulphate which is given IV as bolus dose directly & maintenance dose over 24 hours after last fit.
* control BP by hydralizin / labetolol IV .
* Close observation of vital sign (PR,RR,BP,Temp.), urine output ,patellar reflex&clonus.
* Assessment of fetal condition & immediate delivery.

**Magnesium sulphate (MgSO4)**

\*Centrally acting anticonvulsant drug.

\* act as membrane stabilizing agent.

\* can be given iv or im but preferable iv

\* Is the drug of choice (1st drug of choice) in the acute phase treatment of eclamptic fit.

\*4-6 g given iv slowly over at least 10 min to arrest fit, then maintain on 1g / hr iv in drip for at least 24 hr from the last fit.

\*should be monitored carefully while giving it because of its toxicity by:

 1.measuring its level in the blood

 2.monitering the following

 a) respiratory rate.

 b) urine output.

 c) patellar reflexes (1st sign to disappear in MgSO4 toxicity).

\* antidote of MgSO4 toxicity is calcium gluconate10%, 10 ml over 10 min given iv.

**Chronic Hypertension :**

* Essential hypertension is the underlying cause in 90% of cases.
* Before a diagnosis of essential hypertension is made,other causes of chronic hypertension should be excluded which are :
* Renal disease:
	+ glomerulonephritis.
	+ Polycystic disease.
	+ Diabetic nephropathy.
	+ Renal artery stenosis.
* Collagen vascular disease :

 -SLE

 - scleroderma.

* Coarctation of the aorta.
* Endocrine causes:

 -phaeochromocytoma.

 - conn s syndrome.

* Irrespective of the underlying cause , the principal concern is that these women may develop superimposed pre-eclampsia(1/3).

**Treatment:**

* **1.mild(BP<150/100):** no need for immediate treatment,however , the pregnancy should be monitored carefully to detect any rise in BP or features of pre-eclampsia or IUGR.
* **2. BP>150/100:**antihypertensive medication is recommended which includes:
* Methyldopa
* Labetolol
* Nifedipine.
* Aim of treatment is to maintain the BP < 160 mmHg &100-110 mmHg diastolic.

It is reasonable to await spontaneous labour or attempt vaginal delivery by induction at 38 weeks

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