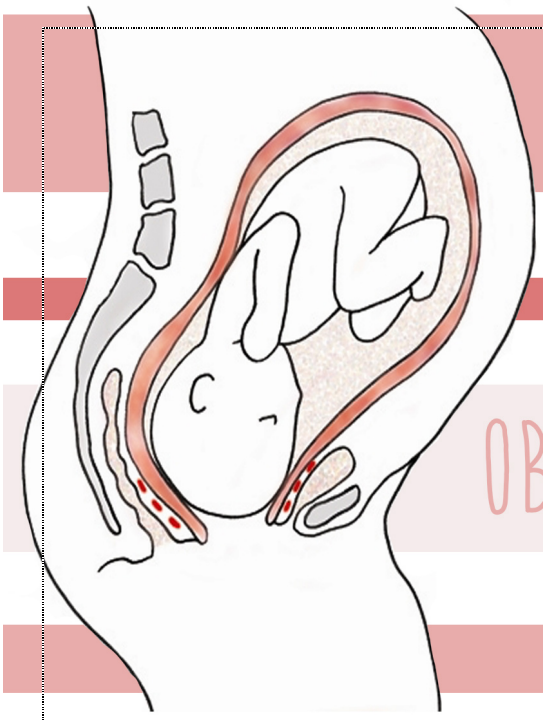

OBSTETRICS AND GYNECOLOGY

HYPERTENSIVE DISEASES OF PREGNANCY

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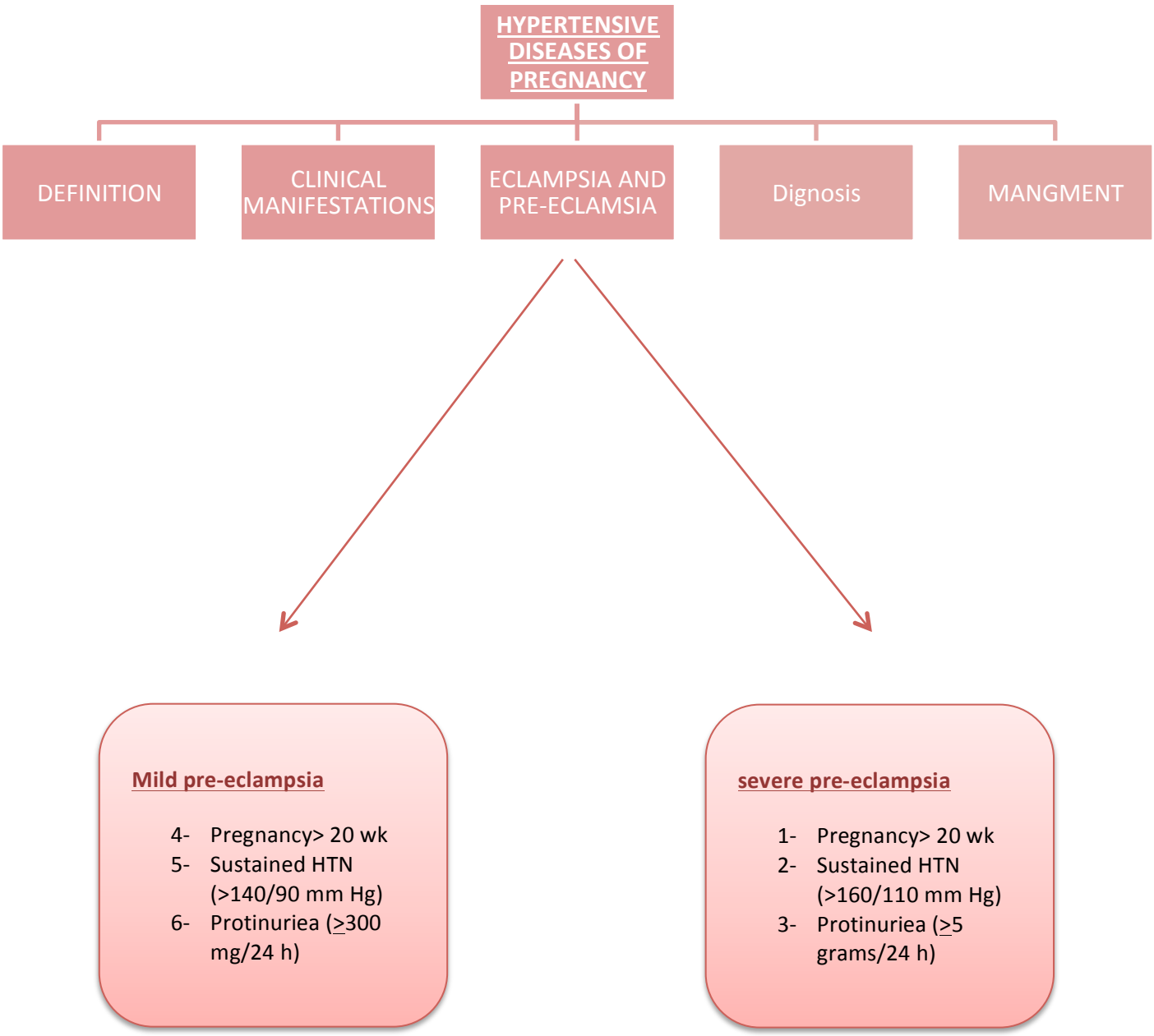
Doctor's note Team's note Not important Important 431 teamwork


HYPERTENSIVE DISEASES OF PREGNANCY
DEFINITION
CLINICAL MANIFESTATIONS
ECLAMPSIA AND PRE-ECLAMSIA
Dignosis
MANGMENT
Mild pre-eclampsia
4- Pregnancy> 20 wk
5- Sustained HTN (>140/90 mm Hg)
6- Protinuriea (≥300 mg/24 h)
severe pre-eclampsia
1- Pregnancy> 20 wk
2- Sustained HTN (>160/110 mm Hg)
3- Protinuriea (≥5 grams/24 h)

DEFINITION:

Those who enter pregnancy with hyper tension (essential hypertension) or renal disease or connective tissue disease.

Coincidental presence of a medical problem e.g. phaeochormocytoma.

Those who develop hypertension during pregnancy which remits within a few months of delivery P.I.H. (Pregnancy induced hypertension)

Superimposed hypertension: لمحظا عم داز سبق لبق اهدنع ناك

 # imp to book the patient early in pregnancy to know if HTN is related to pregnancy or not.

HYPERTENSION AND PROTEINURIA

عظ اذا110 هتانعم ديكا ام طغض اهدنع ديعا جاتحي ءارقلا .

Classification by the ISSHP

Diastolic BP≥ 110 mmhg on any **one** occasion or

Diastolic BP ≥ 90 mmhg on any two or more **consecutive** occasions ≥ 4 hours apart

100 – 105 may lead to stroke.

SEVERE HYPERTENSION

Diastolic BP≥ 120 mmhg on any **one** occasion or

Diastolic BP≥ 110 mmhg on any **two or more** consecutive occasions ≥ hours apart

It also depends on the patient if she is underweight 90 will be sever and high.

PROTEINURIA

One 24 hrs urine collection ≥ 300 mg of proteinuria

Or (**over 4hrs apart**)Two MSU or catheter specimens with ≥ ++

protein or reagent strip(if you are expecting > admit > do 24 hrs test)

BLOOD PRESSURE

Fall in BP in the **1st trimester**-nadir in **2nd and goes back** to non-pregnant in **the 3rd**. (normally it will be law in first and second trimester and return back to normal in the third, if it is high in the second trimester > pathological)

Cardiac output increases by **40% in 1st trimester**

Decrease in BP is due to fall in **peripheral resistances**.

Increase in BP is due to **vasoconstriction**

Technique of taking BP K4 vs K5

PROTEINURIA

(**Vaginal discharge** is a common cause of proteinuria that's why try to educate the patient to clean it before passing urine and testing)

Indicative of severe disease

Increase proteinuria is related to increased perinatal mortality

However, eclampsia can occur without proteinuria

OEDEMA

Some patients will not have protinurea but will notice sudden weight gain, puffy eyes and her ring becomes tight

Severe pre-eclampsia/eclampsia can occur without oedema

In fact, perinatal mortality has been shown to be greater in pre-eclampsia without oedeam 70% of normal pregnancies have oedema

Hyperuricaemia precedes proteinuria (normally the uric acid will raise in pregnancy but in a normal limit if it exceed it something wrong is going on)

Serial platelets counts **(earliest and most significant sign)**

CLINICAL PRESENTATION:

A

Chronic Hypertension (1-1.5%) of pregnancies Persistently increase BP in 1st trimester If seen late may be confused with PIH-

plasma urate can be used to distinguish (uric acid and platelets count are two **important** tests)

Increased risk of super imposed PIH or preeclampsia and IUCR.

B

PIH and Eclampsia Increase BP in the 2nd half (PIH) Associated with significant proteinuria- pre-eclampsia Associated with variable systemic upset (coagulation renal and hepatic function)

Eclampsia:

C

Ultimate consequence of severe PIH characterized **by grand mal seizures** .. rule out epilepsy, etc.

15-20% may have no symptoms

38% - antenatal

44% post partum (w/in 48 hrs & up to 2-3/52) 12% - intrapartum

Prodromal symptoms – headache, visual disturbance and epigastric pain.

EPIDEMIOLOGY

Risk Factors

a. First pregnancy
b. Previous pre-eclampsia
c. Central obesity
d. Migraine
e. Age <20 yr and > 35 years
f. Maternal family history of pre-eclampsia
g. Diabetes
h. Congenital or acquired thrombophilia
i. Renal and connective tissue disease
j. Essential hypertension
k. Multiple pregnancy
l. Hydrops and molar pregnancy
m. Fetal trisomy

MATERNAL OUTCOME

Eclampsia and deaths from ARDS (from pulmonary oedema, fluid overload or over transfusion). Also cerebral edema and infaction DIC, renal failure, hepatic necrosis liver rupture. Metabolic changes cause hyper insulinaemia and increased triglyceridaemia, there are reports of increase I.H.D. in formerly pre-eclamptic women.

FETAL OUTCOME

IUGR, Intrauterine asphyxia and introgenic prematurity

In the longer term IUGR babies are at increased risk of cardiovascular diseases and diabetes later in life.

PATHOLOGY

- In normal pregnancy (by the 6th week)
- Spiral arteries are invaded by cytotrophoblast which breaks down the endothelium, internal elastic lamina and muscular coat of the vessel, which are largely replaced by fibrinoid.
- Virtually every spinal artery in the deciduas basalis has undergone these changes by the end of the 1st trimester.
- Another cytotrophoblast invasion occurs in early 2nd trimester and transforms the myometrial segments of the spiral arteries.
- The vascular supply thus changes from a high pressure low-flow system to low pressure high-flow system to meet fetal placental needs.
- These vessels are also unable to respond to vasomotor stimuli

IN PRE-ECLAMPSIA

- Only ½ to 2/3 of the vessels are affected in the 1st invasion and none for the 2nd invasion. Therefore there is restricted placental blood flow. Also the vessels maintain their muscular coats and remain sensitive to vasomotor stimuli (these changes can occur also in IUGR).

(time is important if she **is 13 weeks** pregnant this is not pre-eclampsia)

Primy gravida is at high risk to develop pre-eclampsia

pre eclampsia is a **multi-system Disease** if one system is affected you should reach the Dx.

LIVER

- Lesions include haemorrhages, periportal deposition, and areas of infarction and necrosis
- Subcapsular haematoma or hepatic rupture can occur
- Shoulder tip pain hepatic rupture
- Epigastric pain, tenderness, vomiting are indicative of fulminating pre-eclampsia

Kidney

- The glomeruli enlarge and protrude in the proximal tubule.
- the swelling narrows the lumen
- damage results in reduced glomerular filtration rate
- Protenuria occurs because of glomerulat dysfunction and it reflects disease severity
- While glomerular dysfunction is manifest as proteinuria, tubular dysfunction is associated with hyper uricarcaimia due to increased rescription of uric acid, which is coupled with tubular sodium re-absorption.
  You should have coagulation profile, DIC. Because it might lead to sudden abruption.

HELLP SYNDROME (Hemolysis elevated liver enzyme levels and low platelet count)

- Haemolysis, abnormalities of liver function and thrombocytopenia lactate dehyrogenases >600 units/1 aspartate amino transferase >70 units/1 low platelets (<100 x 109/L).
- Means severe disease even when BP is not too high
- Common symptoms – **epigastric** and **right hypochondrial pain**, **nausea** and **vomiting** and **visual disturbance > RED FLAGS**
- Maybe misdiagnosed as viral hepatitis, cholestasis of pregnancy, cholecystitis, hyperemesis or acute fatty liver of pregnancy
- Delivery is the safest option for the mother
- High dose **steroid** therapy

THE BRAIN

- Eclampsia – extreme clinical manifestation of pre-eclampsia
- Cerebral vasospasm
- Pathological features include oedema. Cerebral haemorrhage, petechial haemorrhages thrombotic lesions and fibrinoid necrosis secondary endothelial dysfunction and vascular damage.
- Cortical blindness

Pre-eclampsia may cause retardation
due to the less perfusion coming to the fetus > IUGR

VASOMOTOR FUNCTION

What is the mechanism? (MCQS)

- In normal pregnancy, there is acquired vascular insensitivity to angiotension II, maximal in 2nd trimester and slowly returns towards the non pregnant situation and is associated with down regulation of II receptors (angiotension)
- In pre-eclampsia, there is a loss of this acquired insecsitivity to AII which antedates clinical disease, also, there is an increase in AII receptors.

ENDOTHELIUM – dependent vascular relaxation is reduced

COAGULATION AND PLATELETS

- Endothelial dysfunction will trigger activation of the haemostatic system.
- There is widespread deposition of fibrin
- The trigger for coagulation disturbances is expression of tissue factors on endometrial cells.

Pregnancy is hyper-coagulable state > may cause DVT
Sudden abruption > at the level of placenta
Genetics play a role in pre-Eclampsia

PATHOPHYSIOLOGICAL PROCESS

- an abnormal maternal response to pregnancy
- placental trigger causing placental damage in case of IUGR alone, or over expressed as increased placental mass (twin or molar pregnancy)
- Maternal response to trigger is dependent on her genotype/phenotype
- Pre-eclampsia is an attempt by the fetus to provoke the mother to compensate for poor placentation by increasing blood pressure to improve perfusion – all these lead to vascular damage in the mother.
- IUGR alone the mother's phenol type allows her to neglect the growth restricted fetus.

Management: IMP

- Prediction and Prevention
- Doppler USS at 22/52 persistent notching
- pattern (reflects failure of the 2nd invasion)
- Prophylactic aspirin (60-75mg/day) especially for those with history of pre-eclampsia before 30-32/52
- Anti oxidant therapy with Vit. C and E

Anti-hypertensive therapy

- Delivery of the baby will abolish the disease
- Drug therapy is required in severe hypertension >170/110 mhg
- First line is methyl dopa or adreceptor antagonist

Risk of cardiovascular disease and diabetes later in life

ANTAGONISTS

- Such as labeta, atenolol or oxprenelol. Second line is a vasodilator e.g. nifedifine. Third line adrenceptor antagonist or methydopa.

METHYDOPA

- Side effects – tiredness, loss of energy, dizziness, depression, flashes, headache, vomiting and palpitation.

HYDRALLAZINE

(most common Drug used in Dilevery Room for emergency not to take it at home)

For severe and acute hypertension IM, IV boluses. IV infusion orally as second line therapy

Onset of action 20-30 mins.

Causes tachycardia (baroreceptor mediated reflex tachycardia and prolonged stimulation of nor adrenaline release)

Calcium – channel blocking agents

Potent vasodilators and rapid onset of action even orally

Block calcium influx into smooth muscles cells so interfering with excitation contraction coupling

Prophylactic magnesium sulpate

Volume expansion - with plasma protein solutions

DELIVERY

- Expedited by the most suitable route
- Epidural analgesia if there is no coagulopathy
- G.A may be
- Laryngeal oedema may make intubation difficult
- Larygoscopy may provoke extreme hypertension
- Avoid ergometrine

ECLAMSPIA AND MANAGEMENT

Classical prodrome or symptomatic
Alert senior staff
IV mg So4 – toxity Rx with 10 ml of 10% cal. Gloconate
Hypertension – IV labetlol or hyrauazine

Magnesium Sulfate > used for sever pre-eclampsia also used for seizures and it is a prophylactic too.

(IF the mother GOES IN the wrong direction gethe baby out, if she is stable keep the baby admit and treat her)

SUMMARY

PREGNENCY HYPERTENSIVE: Those who enter pregnancy with hyper tension (essential hypertension) or renal disease or connective tissue disease.

Dx: Classification by the ISSHP

Diastolic BP≥ 110 mmhg on any **one** occasion or

Diastolic BP ≥ 90 mmhg on any two or more **consecutive** occasions ≥ 4 hours apart

One 24 hrs urine collection ≥ 300 mg of proteinuria

MAY BE ASSOCIATED WITH OEDEMA

(uric acid and platelets count are two **important** tests)

ECLAMPSIA ; Ultimate consequence of severe PIH characterized **by grand mal seizures** .. rule out epilepsy, etc (HAPPENS AT DELEVRY)

COULD LEAD TO FETAL ASPHYXIA AND PREMATURI

PRE-ECLAMPSIA :

(time is important if she **is 13 weeks** pregnant this is not pre-eclampsia)

Primy gravida is at high risk to develop pre-eclampsia

pre eclampsia is a **multi-system Ds** if one system is affected you should reach the Dx.

HELLP SYNDROME RED FLAGS :

epigastric and **right hypochondrial pain**, **nausea** and **vomiting** and **visual disturbance**

MANGMENT(IMP)

Anti-hypertensive therapy – ANTAGONISTS - METHYDOPA

HYDRALLAZINE : **(most common Drug used in Dilevery Room for emergency not to take it at home) -**

Magnesium Sulfate > used for sever pre-eclampsia also used for seizures and it is a prophylactic too.

ECLAMSPIA AND MANAGEMENT

- Alert senior staff - IV mg So4 – toxity Rx with 10 ml of 10% cal. Gloconate
- Hypertension – IV labetlol or hyrauazine

Case: (from Kaplan's)

SEVERE PREECLAMPSIA

A 21-year-old primigravida is seen in the outpatient prenatal clinic for a routine

visit. She is at 32 weeks' gestation, confirmed by first trimester sonogram. For the

past 24 h she had experienced severe, unremitting occipital headache, and mid
epigastric pain not relieved by acetaminophen, and she has also seen light flashes

and spots in her vision. She has gained 10 pounds since her last visit .2 weeks ago

On examination her BP is 165/115. She has 2+ pedal edema, and her fingers appear

swollen. Fundal height is 29 cm. Fetal heart tones are regular at 145 beats/min. A

.spot urine dipstick shows 4+ protein

Diagnostic Tests. The diagnosis is made on the basis of the finding of at least mild preeclampsia

:plus anyone of the following

.Sustained BP elevation of ~160/110 •

.Proteinuria on dipstick of 3-4+ or ~5 g on a 24-h urine collection •

Evidence of maternal jeopardy. This may include symptoms (headache, epigastric •

pain, visual changes), thrombocytopenia (platelet count <l00,000/mL), elevated liver

.enzymes, pulmonary edema, oliguria (<750 mL124 h), or cyanosis

.Edema mayor may not be seen •

Risk Factors. These are the same as mild preeclampsia with the addition of diseases with small

vessel disease such as systemic lupus and longstanding overt .diabetes

Etiology/Pathophysiology. Pathophysiology is the same as mild preeclampsia but involves

severe diffuse vasospasm and more intense capillary injury to where the ischemia demonstrates

.itself in overt, usually multiorgan system injury

Presenting Symptoms. Presence of new onset of persistent headache, epigastric pain, or visual

.disturbances is characteristic of severe preeclampsia

Laboratory Abnormalities. Evidence of hemoconcentration will be more severe. Proteinuria is

described under diagnostic tests. Evidence of DIC and hepatocellular injury is characteristic of

.severe preeclampsia

Management

Aggressive prompt delivery is indicated for severe preeclampsia at • any gestational age

.with evidence of maternal jeopardy or fetal jeopardy

Administer IV MgSO 4 to prevent convulsions. Give a 5-g loading dose, then continue

.maintenance infusion of 2 gIh

Lower BP to diastolic values between 90 and 100 mm Hg with N hydralazine and/or

.labetalol

Attempt vaginal delivery with N oxytocin infusion if mother and .fetus are stable

Conservative inpatient management may rarely be attempted in • absence of maternal

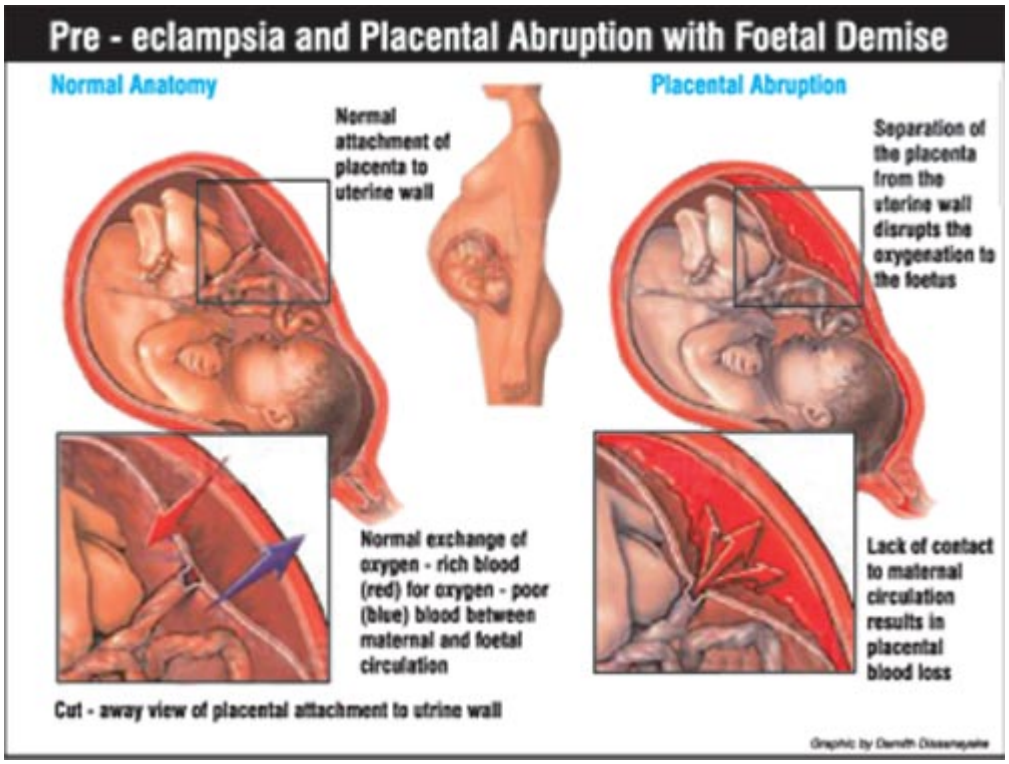
and fetal jeopardy with gestational age 26-34 weeks if BP can be brought below

mm Hg. This should take place in an intensive care unit 110/160 (ICU) tertiary-care

setting. Continuous IV MgSO 4 should be administered, and maternal betamethasone

.should be given to enhance fetal lung maturity

Complications. Progression from severe preeclampsia to eclampsia .may occur


Pre - eclampsia and Placental Abruption with Foetal Demise
Normal Anatomy
Normal attachment of placenta to uterine wall
Placental Abruption
Separation of the placenta from the uterine wall disrupts the oxygenation to the foetus
Normal exchange of oxygen - rich blood (red) for oxygen - poor (blue) blood between maternal and foetal circulation
Lack of contact to maternal circulation results in placental blood loss
Cut - away view of placental attachment to utrine wall