

Hypertensive Disorders in Pregnancy

BY

PROF. DR. YASSER A. HELMY

**Prof. of OB/GYN,
Faculty of Medicine- Sohag
University .**

Hypertension is diagnosed when the blood pressure is 140/90 mm Hg or more on measured under basal condition at least on two occasions 6 hours apart.



Terms

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Classification

****Gestational hypertension**

****Preeclampsia**

****Eclampsia**

****Chronic hypertension**

****Superimposed preeclampsia**

Gestational hypertension:

**BP \geq 140/90 mmHg for first time during pregnancy

**No proteinuria

**BP returns to normal $<$ 12 weeks' postpartum, so the final diagnosis made only postpartum

Preeclampsia:

**BP \geq 140/90 mmHg after 20 weeks' gestation

**Proteinuria \geq 300 mg /24 hrs.

** Lower limb edema is not essential criteria for the diagnosis

Preeclampsia:

- Preeclampsia is considered severe if there is
- the systolic bl pr > 160 mmHg or the diastolic bl pr > 110 mmHg
 - proteinuria exceed 5 g/24 h
 - urine output < 400 ml/24 h
 - abnormal liver function tests
 - platelet count < 100,000
 - neurological symptoms (headache, visual blurring, altered consciousness)
 - epigastric pain (congested liver or subcapsular hepatic hematoma)

Eclampsia

Eclampsia = preeclampsia + convulsions

** Generalized convulsions that may appear before, during, or after labor and cannot be attributed to other causes in a woman with preeclampsia

Chronic hypertension

** BP \geq 140/90 mmHg before pregnancy
or diagnosed before 20weeks'
gestation

OR

**Hypertension first diagnosed after
20weeks' gestation and persistent after
12weeks' postpartum

Chronic hypertension

Underlying causes:

- Essential hypertension (90%)
- Endocrine disorders (Cushing syndrome, pheochromocytoma, thyrotoxicosis)
- Glomerulonephritis
- Connective tissue disease (systemic lupus, periarteritis nodosa)

Preeclampsia superimposed on Chronic Hypertension

New-onset proteinuria ≥ 300 mg/24 hours in hypertensive women who previously have no proteinuria after 20 weeks' gestation

Preeclampsia

Incidence and Risk Factors:

Preeclampsia constitutes about 75% of the hypertensive disorders in pregnancy. It affects about 5% of pregnancies (wide variation among different populations)

The risk factors include:

**Nulliparity (Exposed to chorionic villi for the first time)

Incidence and Risk Factors:

**Twins or molar pregnancy (Exposed to superabundance of chorionic villi)

**Maternal age

**Ethnicity

**Obesity

**Diabetes

**¹⁴Chronic hypertension

Etiology :

Vascular endothelial damage with vasospasm, and ischemic and thrombotic sequelae which may result from:

.

****Abnormal trophoblastic invasion of Uterine vessels**

****Immunological intolerance between maternal and fetoplacental tissues**

Etiology :

****Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy .**

****Dietary deficiencies .**

****Genetic predisposition**

Etiology : Abnormal trophoblastic invasion of Uterine vessels

In normal implantation, endovascular trophoblast invades the uterine spiral arteries, while in preeclampsia there is incomplete trophoblastic invasion. The magnitude of defective trophoblastic invasion of the spiral arteries is correlated with the severity of the preeclampsia.

Etiology : Abnormal trophoblastic invasion of Uterine vessels

By using electron microscopy there are:

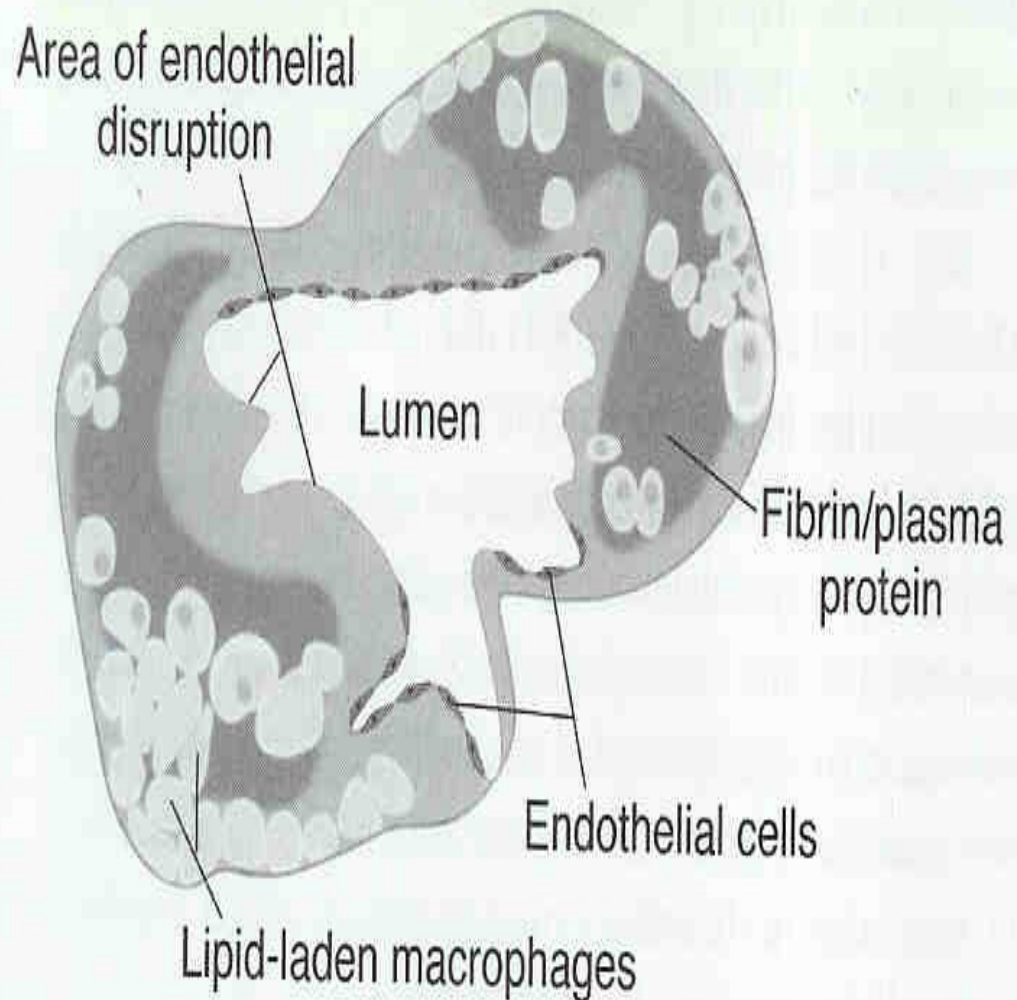
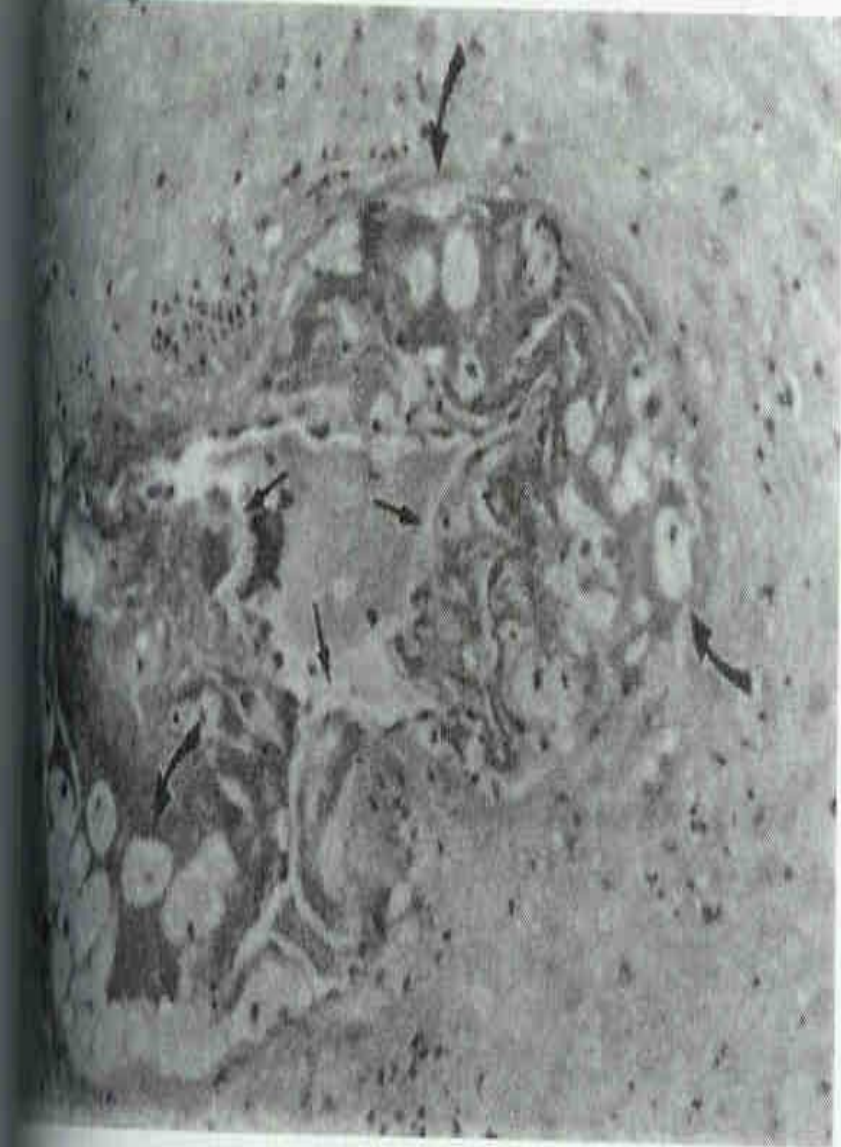
- **Endothelial damage .
- **Transudation of plasma constituents into vessel walls .
- **Proliferation of myointimal cells .

Etiology : Abnormal trophoblastic invasion of Uterine vessels

By using electron microscopy there are:

- **Medial necrosis
- **Lipid and macrophage accumulates in myointimal cells
- **Obstruction of the spiral arteriolar lumen by atherosclerosis may impair placental blood flow
- **Diminished placental perfusion

Etiology : Abnormal trophoblastic invasion of Uterine vessels



Etiology : Immunological Factors:

Theories:

****The formation of blocking antibodies of placental antigenic sites might be impaired**

****Number of antigenic sites provided by the placenta is unusually great compared with the amount of antibody, as with multiple fetuses**

****Effective immunization by a previous pregnancy is lacking, as in first pregnancies**

Etiology : Immunological Factors:

Preeclamptic women have Lower proportion of helper T cells (Th1) with (Th2) dominance. TH1 cells secrete specific cytokines that promote implantation, and their dysfunction may favor preeclampsia.

Etiology : Vasculopathy and the Inflammatory changes :

****The decidua contains an abundance of cells that, when activated, can release noxious agents that can provoke endothelial cell injury.**

****Cytokines TNF- α & interleukin may contribute to the oxidative stress associated with preeclampsia with the formation of highly toxic radicals which injure the endothelial cells**

Etiology : Nutritional factors :

Dietary deficiencies and excesses over the centuries have been blamed as the cause of eclampsia & supplementation with various elements such as zinc, calcium, and magnesium has been used to prevent preeclampsia.

Prediction :

Many biochemical and biophysical tests based on the postulated pathophysiological processes involved in the pathogenesis of preeclampsia have been used for its prediction but with low sensitivity:

Prediction :

--Angiotensin II infusion

--Roll over test

--Fibronectin (marker of endothelial cell injury)

--Markers of oxidative stress

Prediction :

--Cytokines

--Uric acid

--Urinary calcium level

--Doppler velocimetry

Prevention :

- Dietary manipulation (salt restriction & calcium supplementation)
- Low dose aspirin
- Antioxidants

Management :

The objectives of treatment are:

--Prevention of complications such as renal failure, abruptio placenta, cardiovascular accidents, pulmonary edema, e.t.c.

--Prevention of convulsions which is associated with increased maternal and perinatal mortality and morbidity.

--²⁹Delivery of healthy baby

Management :

- Antihypertensive drug therapy
- Termination of pregnancy
- Seizure prophylaxis

Eclampsia

Eclampsia is a preventable life-threatening complication of pregnancy, characterized by the appearance of tonic-clonic seizures, in a patient who has preeclampsia.

Eclampsia is associated with 10 folds increase in the maternal mortality and morbidity. The causes are:

--cerebral hemorrhage

--pulmonary edema

--renal failure

--HELLP syndrome

--hepatic rupture & internal
hemorrhage

The principles of eclampsia management:

- prevention further convulsions
- control the elevated blood pressure
- termination of pregnancy

Chronic hypertension

Chronic hypertension in pregnancy carries risks both for the mother & the fetus:

--superimposed preeclampsia

--IUGR

--Abruptio placenta

--preterm labor

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--IUGR

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--Ideally these patients are to be managed before being pregnant but unfortunately most cases are seen after conception. BI pr limit of 160/110 mmHg is used to differentiated mild from severe hypertension

--severe hypertension carries a worse prognosis

--in mild hypertension the presence or absence risk factors determine the need for antihypertensives

Risk factors for complications & poor outcome:

--history of severe hypertension in previous pregnancies

--history of IUGR, stillbirth or abruptio placenta

--age > 35 or more than 15 years of hypertension

--marked obesity

Management plan:

--control of hypertension

--monitoring of fetal growth and activity

--lab investigations

--delivery



**Thank
you**