

Problem of gestoses

Gestosis is a syndrome defined as violated adaptation of a woman to pregnancy. Gestosis arises only in connection with pregnancy, is etiologically linked to fetal egg development, is characterized by various symptoms, complicates the course of pregnancy and usually disappears right after or in some time after the end of pregnancy.

Classification

- Early gestoses (1-st 3 months)
- Late gestosis (after 20 weeks)
- Rare forms of gestosis

Aetiopathogenesis (theories)

- toxemic
- allergic, immune
- corticovisceral
- hormonal
- neurogenic
- psychogenic
- genetic



The concept of "early gestosis" exists only in the practice of obstetricians - gynecologists of the ICU. In the obstetric practice of foreign countries, this concept does not exist, where these states are regarded as "minor" complications of pregnancy, or "unpleasant symptoms during pregnancy."

1. Early gestosis, often occurs - vomiting of pregnant women and ptyalism (hypersalivation)
2. Early gestosis, rarely occurs - dermatoses of pregnant women, cholestatic hepatitis of pregnant women, acute fatty hepatitis of pregnant women, chorea of pregnant women, osteomalacia during pregnancy.

Classification of vomiting:

- mild -3-5 times a day on an empty stomach or after meals, reduced appetite
- moderate- 10 times a day irrespective of food intake, weight loss, weakness, apathy, electrolyte imbalance
- severe – more than 10 times a day, no food is held, weight loss, low grade fever, icteric skin, acetonuria with oliguria, tachycardia, hypotension, hyperbilirubinemia, potassium reduction, hypoproteinemia, hematocrit increase

Differential diagnosis of vomiting in pregnant women should be carried out with the following diseases:

- food toxicoinfection
- gastritis
- Pancreatitis
- Pyelonephritis
- Cholelithiasis
- viral hepatitis
- Appendicitis
- Meningitis
- brain tumors

Treatment

- Psychotherapy: electrical sleep ,laser reflexotherapy, acupuncture, sedatives or tranquilizers (diazepam, seduxen)
- Antiemetic agents – droperidol, aminazine, etapirazin, cerucal
- Water-electrolytic balance correction
- Mild: dietary correction - fractional (5-6 times a day), balanced nutrition, plenty of drinks, vitamin therapy.
- In case of vomiting of pregnant women, moderate and severe, hospitalization and prescription of drug therapy are indicated.

Hypersalivation(ptyalismus)

The amount of saliva with hypersalivation can reach 1.0 l per day. Salivation does not cause severe irregularities in the body, causes maceration of the skin and mucous membrane of the lips.

In order to reduce the secretion of the salivary glands, intramuscular administration of atropine is prescribed in 0.5 ml 0.1% solution 2 times a day. atropine is prescribed in 0.5 ml 0.1% solution 2 times a day. It is advisable to rinse the mouth with infusion of sage, mint, chamomile, oak bark and other means that have astringent properties.

Rare forms of gestosis

- Dermatoses
- Chorea
- Itching
- Osteomalation

Rare forms of gestosis

- dermatoses of pregnant women



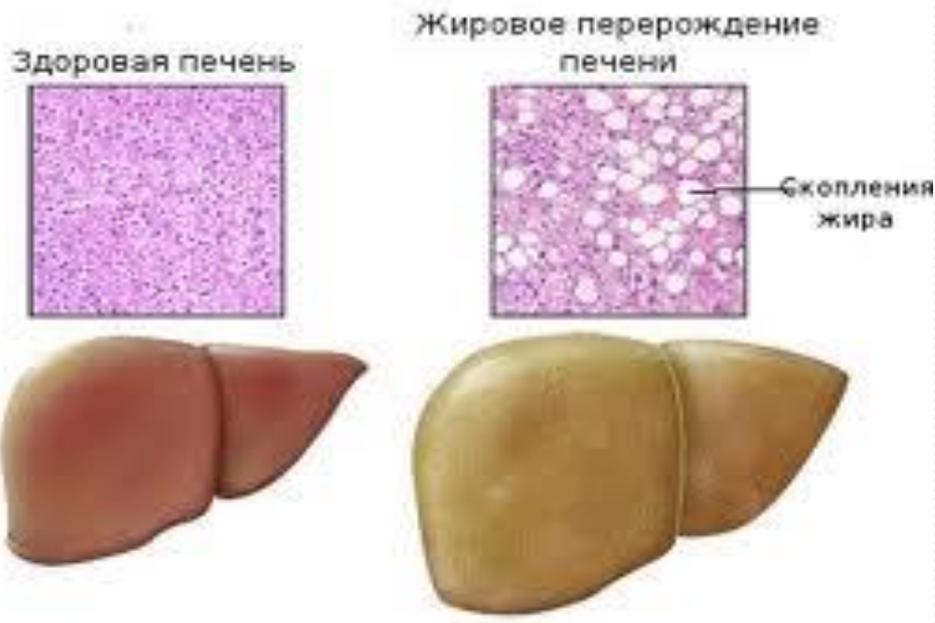
Rare forms of gestosis

- pemphigoid of pregnant women



Rare forms of gestosis

- Cholestatic hepatosis of pregnant women
- Acute fatty hepatosis of pregnant women



- **Chorea (tetany)** occurs in connection with a violation of calcium metabolism, due to the hypofunction of the parathyroid glands.

Clinically, it manifests convulsive uncoordinated muscle twitching of the upper, lower extremities, sometimes the face, very rarely the larynx or the stomach.

Rare forms of gestosis

- osteomalacia



Gestosis in modern obstetrics

- Late gestosis of pregnant women (LGP) is a symptom complex of polyhedral and polysystemic insufficiency that occurs during pregnancy.
- The frequency of LGP varies from 7% to 16% among all pregnant women. In the structure of mortality of pregnant women, parturient women and mothers, LG takes one of the first places.

- GESTOSIS is not an independent disease.
- this is a clinical manifestation of the failure of the adaptation mechanisms of the maternal organism to adequately meet the needs of the fetus that develops. This failure is realized through a varying degree of expressiveness of perfusion-diffusion insufficiency in the mother-placenta-fetus system.

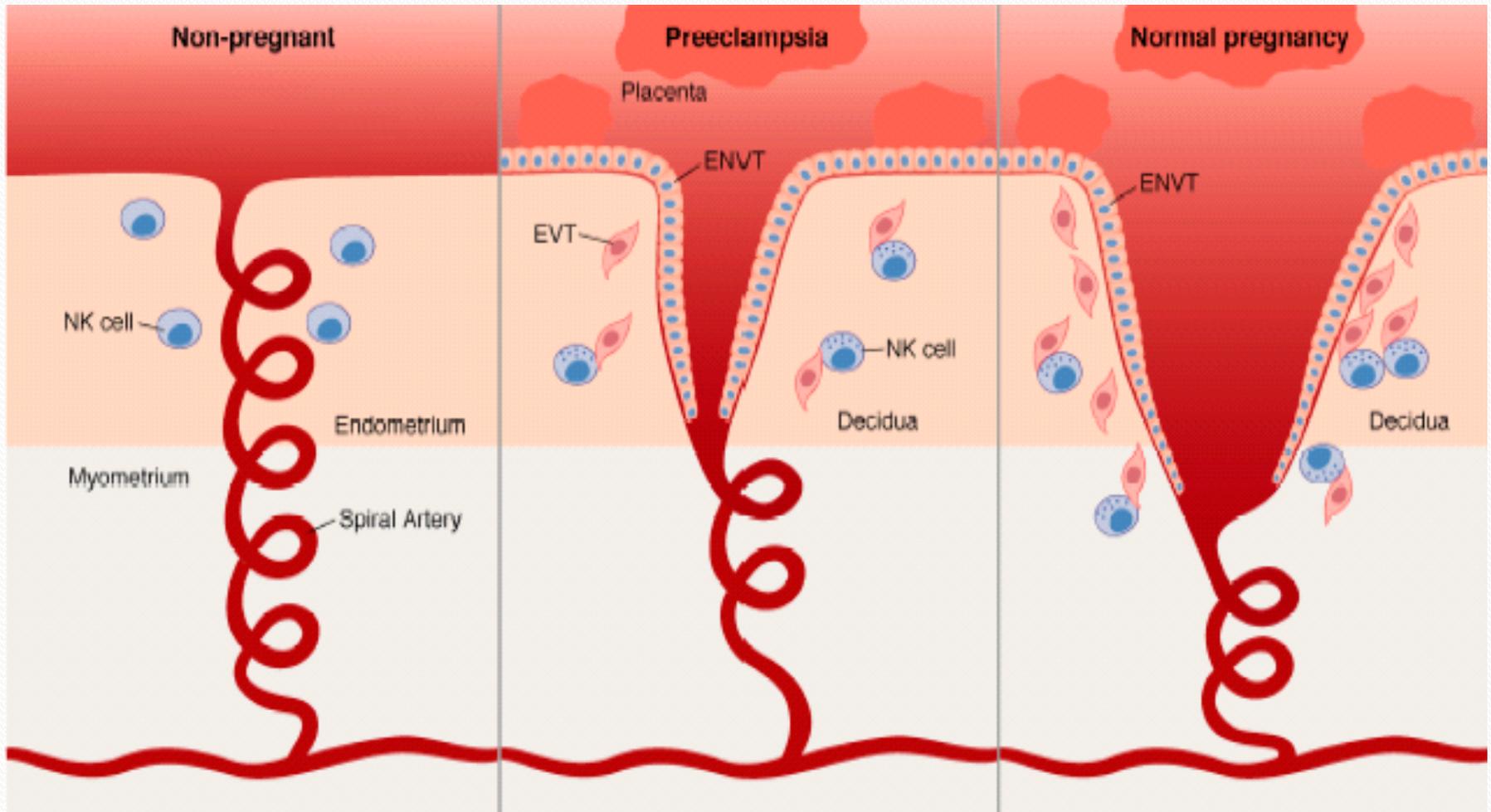
Aetiology of gestosis

- The etiology of preeclampsia is not fully understood.
- There are about 30 different theories. But the definition of preeclampsia as a disease of adaptation is most consistent with the ideas about it. Of particular importance is immunological changes during pregnancy.

Aetiology and Pathogenesis

- Basic etiology is *abnormal placentation* : failure of trophoblast invasion
- Failure of second wave of endovascular trophoblast migration resulting in reduction of blood supply to fetoplacental unit.
- 2 main things we should remember :
 - Endothelial Dysfunction* due to oxidative stress and inflammatory mediators, *Vasospasm* due to imbalance b/w vasodilators(PGI₂, NO) & vasoconstrictors (TxA₂, angiotensin 2, endothelin).

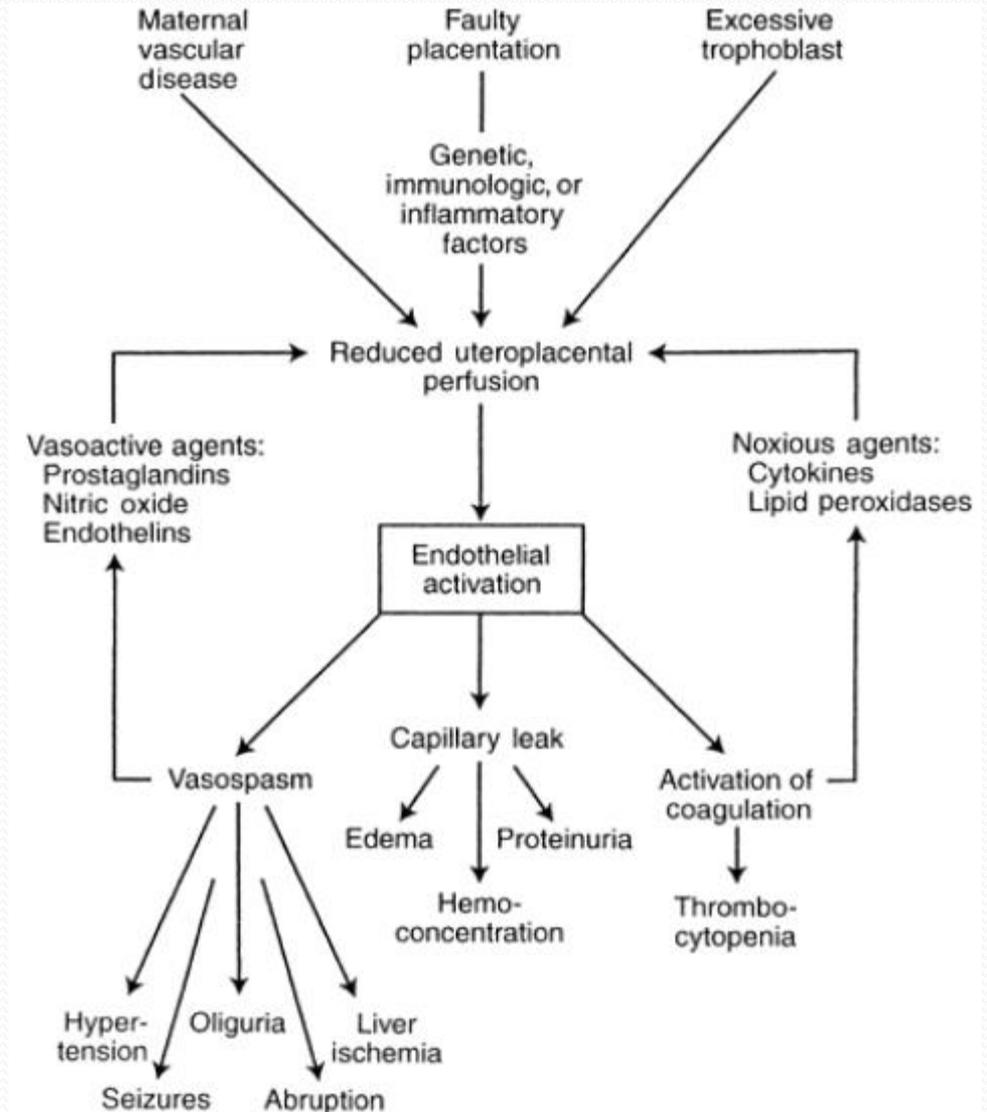
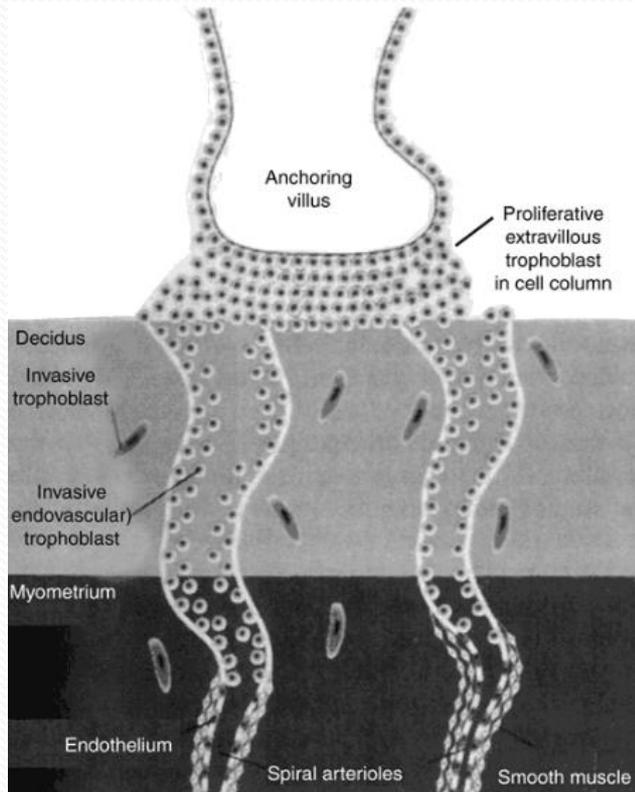
Aetiology and Pathogenesis



Aetiology and Pathogenesis

- Abnormal trophoblast invasion – failure of the trophoblast invasion to myometrial segments of the spiral arteries.
- Spiral arteries retain their muscular walls and prevent the development of the high blood flow and increase impedance in the utero-placental circulation.
- Diffuse vasospasm caused by reduced sensitivity to vasodilators (nitrous oxide and prostacyclin) and enhanced sensitivity to vasoconstrictors (angiotensin).

Late gestosis



Pathology

- Diminished plasma volume
- Increase extracellular fluid
- Vasospasm
- Disseminated intravascular coagulation in very severe cases ischaemic changes in various organs, when the disease is severe. General vasospasm lead to changes of reologic properties of blood: appear stasis, acedosis and formation of polyorganic insufficiency. Hypovolemey lead to decrease of protein in plasma, as a result-disorders of leaver functions, low of osmotic pressure and liquid part of blood go away in itraorganic space, then oedema, centralisation of blood flow, increase of BP

Changes in organs with preeclampsia:

- Cardiovascular system: general vasospasm, increased peripheral vascular resistance, hypovolemia.
- Hematological changes: activation of platelets, followed by consumption coagulopathy, decreased plasma volume, increased blood viscosity, hemoconcentration.
- Urinary System: proteinuria, reduced glomerular filtration rate, decreased excretion of uric acid.
- Liver: necrosis, subcapsular hematoma.
- CNS: brain edema, intracranial hemorrhage.

Risk factors for preeclampsia:

1. Extragenital pathology: kidney, liver, hypertension, chronic pulmonary and bronchial diseases, heart disease, diabetes, obesity and other manifestations of endocrinopathy.

2. Obstetric risk factors:

- Family history of arterial hypertension (family history);
 - Availability of pre-eclampsia in previous pregnancy;
 - Age pregnancy (up to 19 years and over 30 years);
 - Polyhydramnios, multiple pregnancy;
 - Anemia of pregnant;
 - Izosensitization by Rh-factor and ABo system.
3. Social and domestic factors:
- Bad habits;
 - Occupational hazard;
 - Unbalanced diet.

Risk factors for pre-eclampsia

- **Moderate**
- ● First pregnancy
- ● Age ≥ 40 years
- ● Pregnancy interval > 10 years
- ● BMI ≥ 35 kg/m² at first visit
- ● Family history of pre-eclampsia
- ● Multiple pregnancy

- **High**
- ● Hypertensive disease during previous pregnancy
- ● Chronic kidney disease
- ● Autoimmune disease such as systemic lupus erythematosus or antiphospholipid syndrome
- ● Type 1 or type 2 diabetes
- ● Chronic hypertension

Late gestosis classification

1. Gestational hypertension- appeared after 20 weeks of pregnancy and is not accompanied by proteinuria up to delivery
 - transient - normalisation of BP during 12 weeks after delivery
 - chronic - continues during 12 weeks after delivery
2. Proteinuria during pregnancy- protein content of 0.3 g/l
3. Edema during pregnancy-local or generalized, pathologic weight gain

Pre-eclampsia –hypertension, which appeared after 20 weeks of pregnancy with proteinuria, with/without edema

4. Mild - diastolic pressure 90-99 mm Hg,proteinuria <0.3 g/L

5. Moderate-diastolic pressure 100-109 mm Hg,proteinuria 0.3-5 g/L ,edema of the face, hands, s.t. headache

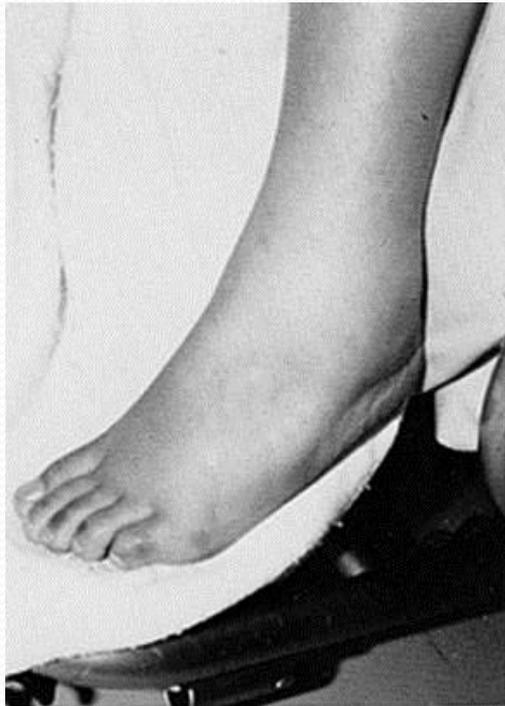
6. Severe –diastolic pressure ≥ 110 mm Hg, proteinuria > 5.0 g/L ,generalized edema , headache,visual impairment ,hyperreflexia, pain in the epigastrium , oliguria (<500 ml/day).thrombocytopenia.

7. Eclampsia (any term)- convulsive attack in the pregnant with pre-eclampsia

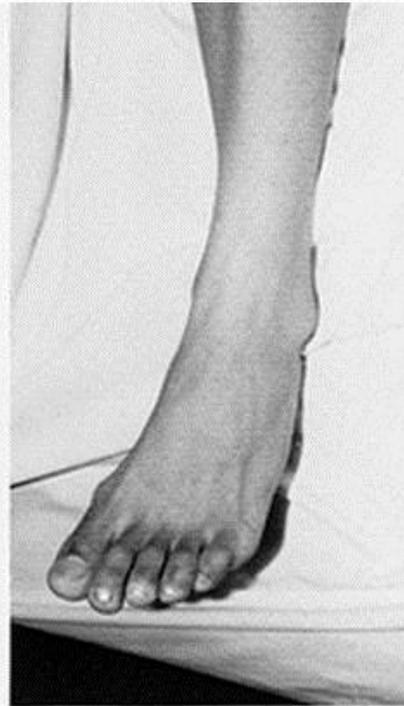
Clinical manifestations:

- The classic triad of symptoms of preeclampsia (edema, proteinuria, hypertension), described in 1913 by the German obstetrician Tsangmeister.
- Headache, blurred vision, epigastric pain and right upper quadrant are the clinical manifestations of severe forms of preeclampsia.

Edemas



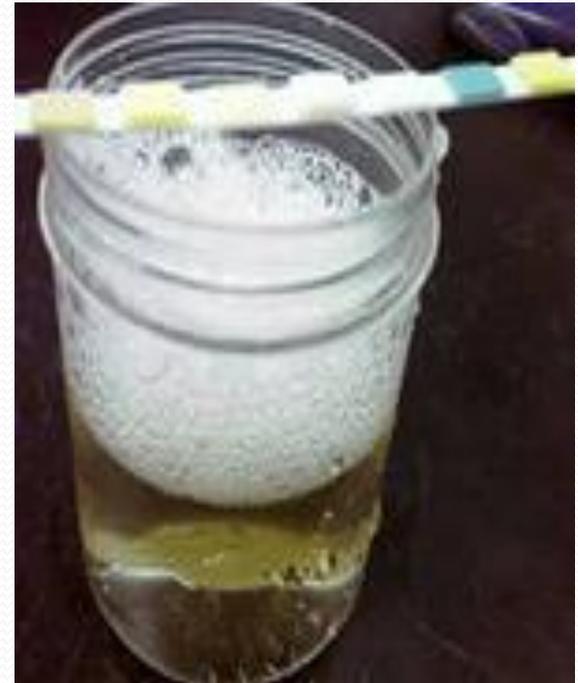
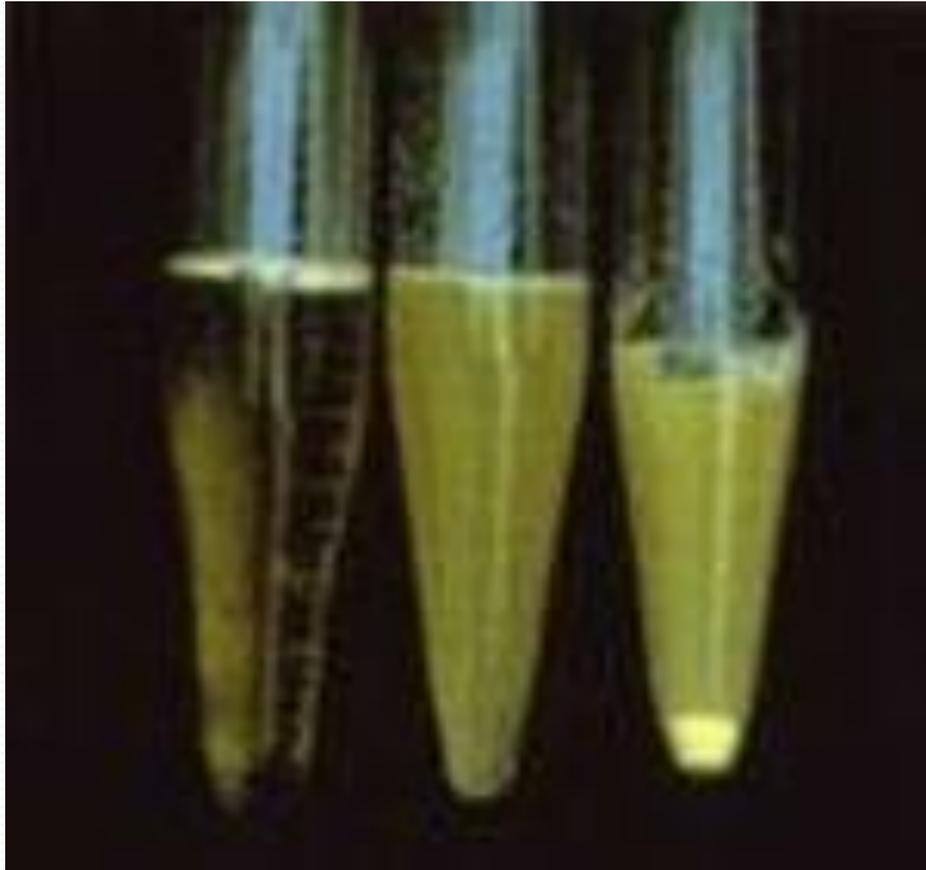
A



B



Proteinuria



Diagnostic criteria for the severity of preeclampsia / eclampsia

Diagnosis	Diast.BP mm.Hg	Proteinu ria, g/day	Other signs
Gestational hypertension or mild preeclampsia	90-99	<0,3	–
Moderate preeclampsia	100-109	0,3-5,0	Swelling on the face, hands Sometimes headache

Diagnostic criteria for the severity of preeclampsia / eclamp

Diagnosis	Diast.BP mm.Hg	Proteinuria, g/day	Other signs
Severe preeclampsia	≥110	>5	Generalized swelling, severe headache Visual impairment Epigaster pain or / and in the right hypochondrium Hyperreflexia Oliguria (<500 ml / day) Thrombocytopenia
Eclampsia	≥90	≤0,3	Convulsive seizure (one or more)

Additional clinical and laboratory criteria for pre-eclampsia

Signs	Mild Pre-eclampsia	Moderate preeclampsia	Severe preeclampsia
Uric acid, mmol / L	< 0,35	0,35-0,45	> 0,45
urea, mmol / L	< 4,5	4,5–8,0	> 8
Creatinine, μmol / L	< 75	75–120	> 120 or oliguria
Thrombocytes $\cdot 10^9$ /L	> 150	80–150	< 80

Mild preeclampsia

Indications for hospitalization: the appearance of at least one sign of moderate preeclampsia, a violation of the fetus.

In the case of a stable state of a woman within the criteria of mild pre-eclampsia, the management of pregnancy is expectant.

Childbirth is conducted according to obstetric situation.

Moderate preeclampsia

Planned hospitalization of the pregnant woman in the hospital.

Initial laboratory examination: complete blood count, hematocrit, platelet count, coagulogram, ALT and AST, blood group and Rh factor (in the absence of accurate information), urinalysis, determination of daily proteinuria, creatinine, urea, plasma uric acid, electrolytes (sodium and potassium), assessment of the condition of the fetus.

Guard mode - half-bed mode, limiting physical and mental stress.

Moderate preeclampsia

Delivery

The method of delivery at any time of gestation is determined by the readiness of the birth canal and the condition of the fetus. With the ineffectiveness of the preparation of the birth canal with prostaglandins a cesarean section have to performed.

If the cervix is mature enough, labor induction is performed and labor is carried out through the birth canal.

The clinical diagnosis of severe preeclampsia is also based on the classic symptoms:

- Hypertension - displays the degree of vasospasm and is the basis of the diagnosis.
- Weight gain and swelling; An increase in the body weight of a pregnant woman that is more than 900.0 grams per month can be the first sign of preeclampsia.
- A headache in the forehead and occiput, resistant to analgesics, may indicate a swelling of the brain, often preceded by cramps.

Features of severe pre-eclampsia

- Severe hypertension and proteinuria or mild or moderate hypertension and proteinuria with at least one of:
 1. severe headache
 2. problems with vision such as blurring or flashing
 3. severe pain just below ribs or vomiting
 4. papilloedema
 5. signs of clonus(≥ 3 beats)
 6. Liver tenderness
 7. HELLP syndrome
 8. platelet count falls to $< 100 \times 10^9$ /litre
 9. abnormal liver enzymes(ALT or AST rises to > 70 iu/litre).

The clinical diagnosis of severe preeclampsia is also based on the classic symptoms:

- Pain in the epigastrium or in the upper right abdomen of the abdomen as a sign of edema or hemorrhage in the liver. This symptom is a symptom of severe preeclampsia, may be a precursor to seizures.
- Visual impairment - from the flashing of the flies and the grid in front of the eyes to complete blindness. They are associated with vasospasm, ischemia and petechial hemorrhages in the cerebral cortex, as well as spasm of retinal arterioles, its ischemia and edema preceding retinal detachment.
- Sensation of nasal congestion (perivascular edema).

Severe preeclampsia. Tactics of conducting.

A pregnant woman is hospitalized in the anesthesiology and intensive care unit of a level III hospital to assess the risk of pregnancy for the mother and fetus and to choose a method of delivery within 24 hours. An individual ward with intensive round-the-clock supervision of medical personnel is distinguished. Immediate consultation with a therapist, neuropathologist, ophthalmologist.

Severe preeclampsia.

management tactics.

Treatment.

- ✓ Security mode (strict bed).
- ✓ In gestational periods up to 34 weeks, corticosteroids for the prevention of RDS.
- ✓ Antihypertensive therapy.
- ✓ Infusion therapy
- ✓ Monitoring the state of the pregnant

Severe preeclampsia.

Management tactics

Delivery is carried out taking into account the obstetric situation.

Preference is given to childbirth through the natural birth canal with adequate anesthesia.

When birth canal is ready-amniotomy has been spent with subsequent labor induction.

When the cervix is unavailable and there is no effect from the preparation with prostaglandins, or in the case of progression of hypertension, the threat of a convulsive seizure, deterioration of the fetus, delivery is performed by cesarean section.



Pre - eclampsia when complicated with convulsions and/or coma is called **eclampsia**.

Cause of convulsion : Cerebral irritation may be provoked by

- Anoxia-spasm of the cerebral vessels following hypertension -> increased cerebral vascular resistance -> fall in cerebral oxygen consumption -> anoxia
- Cerebral oedema – may contribute to irritation
- Cerebral dysrhythmia – increases following anoxia or oedema

Hematoma of tongue from laceration during an eclamptic convulsion.



- Headaches, pain under the breasts, blurred vision may be harbingers of seizures.
- Others can go after the first convulsions; they can be from 10 to 100 or more in severe cases of eclamptic status.
- Death can occur from massive bleeding in the brain. With cerebral hemorrhages, hemiplegia may develop.
- In the absence of adequate treatment, an eclamptic coma develops.
- Loss of consciousness may be sudden without the onset of convulsions “eclampsia without eclampsia”. A differential diagnosis of eclampsia with encephalitis, meningitis, aneurysm, rupture of the cerebral vessels, and hysteria should be made.

Eclamptic seizure

- The patient may have 1 or more seizures.
- Seizures generally last 60-75 seconds.
- The patient's face initially may become distorted, with protrusion of the eyes.
- The patient may begin foaming at the mouth.
- Respiration ceases for the duration of the seizure.

Eclamptic convulsions or fits: The fits are epileptiform and consist of four stages.

- Premonitory stage: The patient become unconscious. There is twitching of the muscles of the face, tongue and limbs. Eye balls roll or turned to one side and become fixed. (30 sec.)
- Tonic stage: The whole body goes into a tonic spasm – the trunk-opisthotonus, limbs are flexed and hands clenched. Respiration ceases and tongue protrudes between the teeth. Cyanosis. Eye balls become fixed (30 sec.)
- Clonic stage :All the voluntary muscles undergo contraction and relaxation .Biting of the tongue occurs. Breathing is stertorous and blood stained frothy secretions fill the mouth; cyanosis gradually disappears (1-4 min)
- Stage of coma

- A coma or a period of unconsciousness follows phase 2.
 - Unconsciousness lasts for a variable period.
 - Following the coma phase, the patient may regain some consciousness.
 - The patient may become combative and very agitated.
 - The patient has no recollection of the seizure.
- A period of hyperventilation occurs after the tonic-clonic seizure. This compensates for the respiratory and lactic acidosis that develops during the apneic phase.

First aid for the development of seizures and coma.

- The patient is placed on a flat surface, avoiding damage.
- They release the airways, open the mouth with a spoon or spatula, draw the tongue forward and, if possible, aspirate the contents of the oral cavity.
- With renewed spontaneous respiration, oxygen is supplied. With prolonged apnea, assisted ventilation immediately begins.
- With the discontinued cardiac activity, in parallel with the ALV, a closed heart massage is performed and all methods of cardiovascular resuscitation are performed.
- To stop seizures in / in except for 20 ml of a 25% solution of magnesium sulphate, 0.002 g of sibazone is injected and administration is repeated after 10 minutes. 0.01g.

- Magnesium sulphate is considered to be the drug of choice for anticonvulsant therapy. It has anticonvulsant and sedative effects and does not cause a marked suppression of consciousness, which makes it possible to remove the problem of differentiation between the depth of drug allergies by neuroleptics, sedatives and narcotic drugs. Magnesium sulfate is also a diuretic and hypotensive effect, and also reduces intracranial pressure. The dose depends on the woman and blood pressure levels.

Complications of Eclampsia

- Tongue biting,
- Head trauma
- Broken bones
- Pulmonary edema from aspiration pneumonitis or heart failure
- Death from massive cerebral hemorrhage
- Hemiplegia from sublethal hemorrhage
- Blindness from retinal detachment or occipital lobe ischemia & edema
- Rarely eclampsia followed by psychosis

HELLP-syndrome:

- H (hemolysis) - microangiopathic hemolytic anemia
- EL (elevated liver enzymes) – the increase of liver enzymes concentration in blood plasma
- LP (low platelet quantity) – the decrease of thrombocytes level

HELLP Syndrome

- Hemolysis
- Elevated Liver enzymes
- Low Platelets
- < 36 wks
- Malaise (90%), epigastric pain (90%), N/V (50%)
- Self-limiting
- Multi-system failure

- 
- The disease most often occurs during pregnancy, at 35 weeks. In 10% of cases in a period of less than 27 weeks, and in 31% - in the first week after birth.

CLINICAL SIGNS:

- HEADACHE, VOMITATION, PAIN IN THE abdomen, MOSTLY IN THE Right upper quadrant (RUQ);
- INCREASING JAUNDANTS, HEPATIC INSUFFICIENCY;
- CONVULSIONS, COMA;
- UNCERTAKABLE LIVER TEAR AND INTRAABDOMINAL BLEEDING;
- COAGULOPATHY, BLEEDING IN THE POSTBIRTH PERIOD;
 - FREQUENTLY TOTAL PONRP + MASSIVE BLEEDING;
 - Hepatic and renal insufficiency.

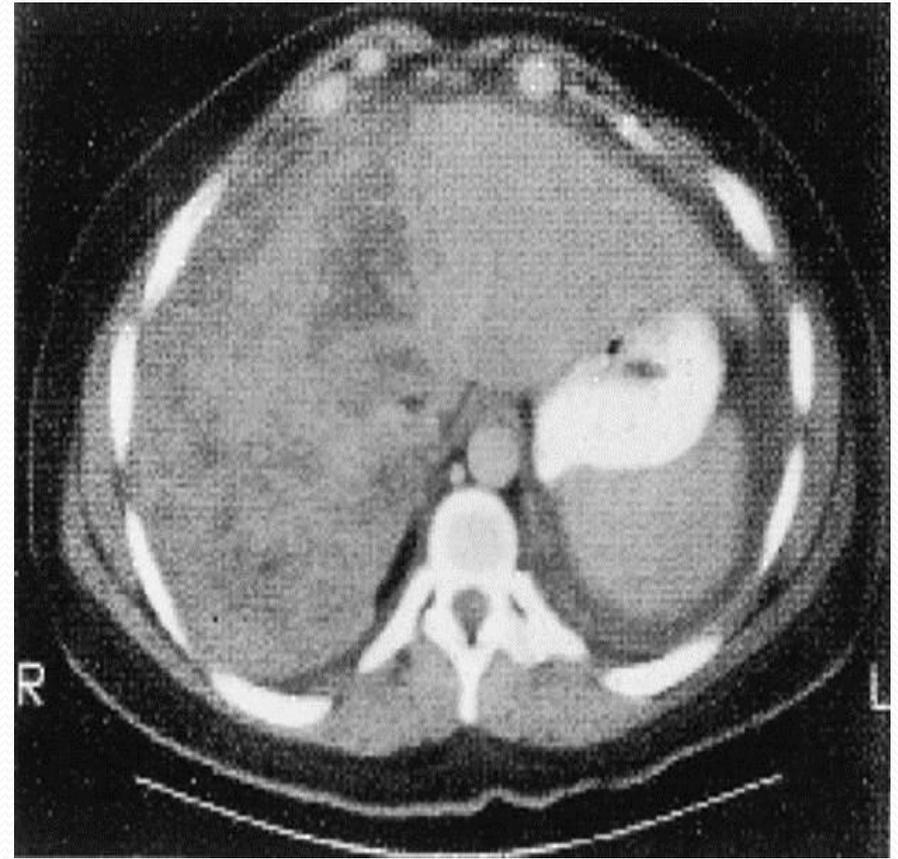
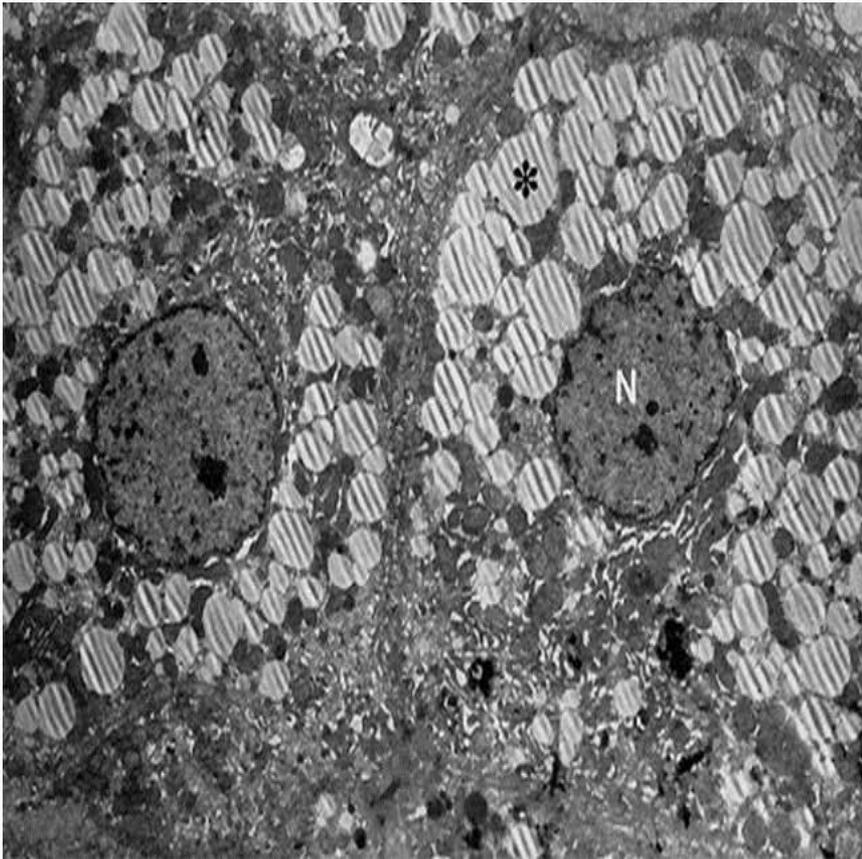
HELLP syndrome

- Epigastric or right upper quadrant pain in a woman with preeclampsia often represents hepatic involvement. This is called '**Pre eclamptic Angina**'. The pain responds poorly to analgesia but both the pain and associated increases in liver enzymes (AST, ALT) may subside (albeit temporarily) after blood pressure lowering, particularly with vasodilators.
- Thrombocytopenia is the commonest hematologic abnormality seen in preeclampsia; the lower limit of the normal platelet count in pregnancy is approximately $140 \times 10^9/L$ but the risk of spontaneous bleeding is not significantly increased until the count falls below $50 \times 10^9/L$. Even so, there are concerns with central neuraxial anesthesia and analgesic techniques at higher levels ($50-75 \times 10^9/L$), and surgical bleeding may be increased even with moderate thrombocytopenia

HELLP syndrome

- Hemostasis is *not problematic* unless $PLT < 40,000$
- Rate of fall in PLT count is important
- Regional anesthesia - contraindicated → fall is sudden
- PLT count → normal within 72 hrs of delivery
- Thrombocytopenia may persist for longer periods.
- Definitive cure is delivery

CHANGES IN THE LIVER WITH HELLP syndrome



Preeclampsia: Definition

- Hypertension
 - > 140/90
 - relative ↑ no longer considered diagnostic
- Proteinuria
 - > 300 mg/24 hours or ≥ 1 or 2+ on urine dipstick
 - **Proteinuria** is defined as urinary excretion 0.3 g protein or greater in a 24-hour+2 or greater on urine dip specimen
- Edema (non-dependent)
 - 'dry' pre-eclampsia has been recognised hence the exclusion of oedema from the diagnostic criteria.

Severe Preeclampsia :- Criteria (one or more)

- **Blood Pressure:** >160 systolic, >110 diastolic
- **Proteinuria:** >5gm in 24 hours, over 3+ urine dip
- **Oligurea:** less than 400ml in 24 hours
- **CNS:** Visual changes, headache, scotomata, mental status change
- **Pulmonary Edema**
- **Epigastric or RUQ Pain:** Usually indicates liver involvement
- **Impaired Liver Function tests**
- **Thrombocytopenia:** <100,000
- **Intrauterine Growth Restriction:** With or without abnormal doppler assessment
- **Oligohydramnios**

Physical Findings in Preeclampsia

- Blood Pressure ↑
- Proteinuria
- Retinal vasospasm or Retinal edema
- Right upper quadrant (RUQ) abdominal tenderness stems from liver swelling and capsular stretch
- Brisk, or hyperactive, reflexes are common during pregnancy, but clonus is a sign of neuromuscular irritability that raises concern.
- Among pregnant women, 30% have some lower extremity edema as part of their normal pregnancy. However, a sudden change in dependent edema, edema in nondependent areas such as the face and hands, or rapid weight gain suggests a pathologic process and warrants further evaluation

Laboratory Tests :- Uric acid

- Decreased renal urate excretion in preeclampsia
- Serum uric acid exceeding 5.9 at 24 wk (PPV 33%)
- Not useful in differentiating GHT from preeclampsia
- usually \uparrow ($> 6\text{mg/dl}$)

Coagulation activation

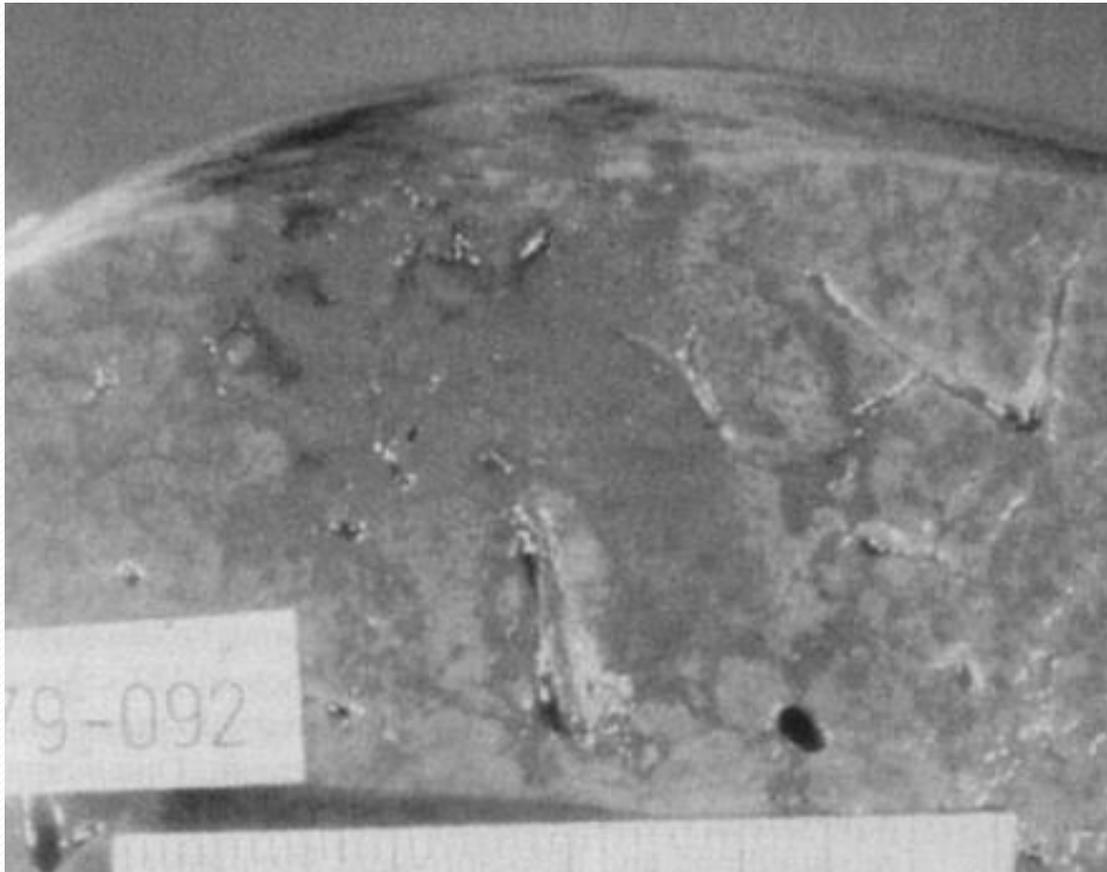
- Thrombocytopenia and platelet dysfunction
- Increased destruction cause platelet volumes increase (younger platelet)
- Preeclampsia : PAI-1 increase increased relative to PAI-2 because of endothelial cell dysfunction

Instrumental Investigations

- Uterine artery doppler
- Fetal Sonography
 - Fetal size
 - Amniotic fluid volume
 - Fetal and maternal dopplers
- CT Scan (Brain)
- ECG
- Doppler sonography
- Cerebral angiography

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- Differential diagnosis :
 - epilepsy , encephalitis , meningitis , cerebral tumor , cysticercosis , ruptured cerebral aneurysm

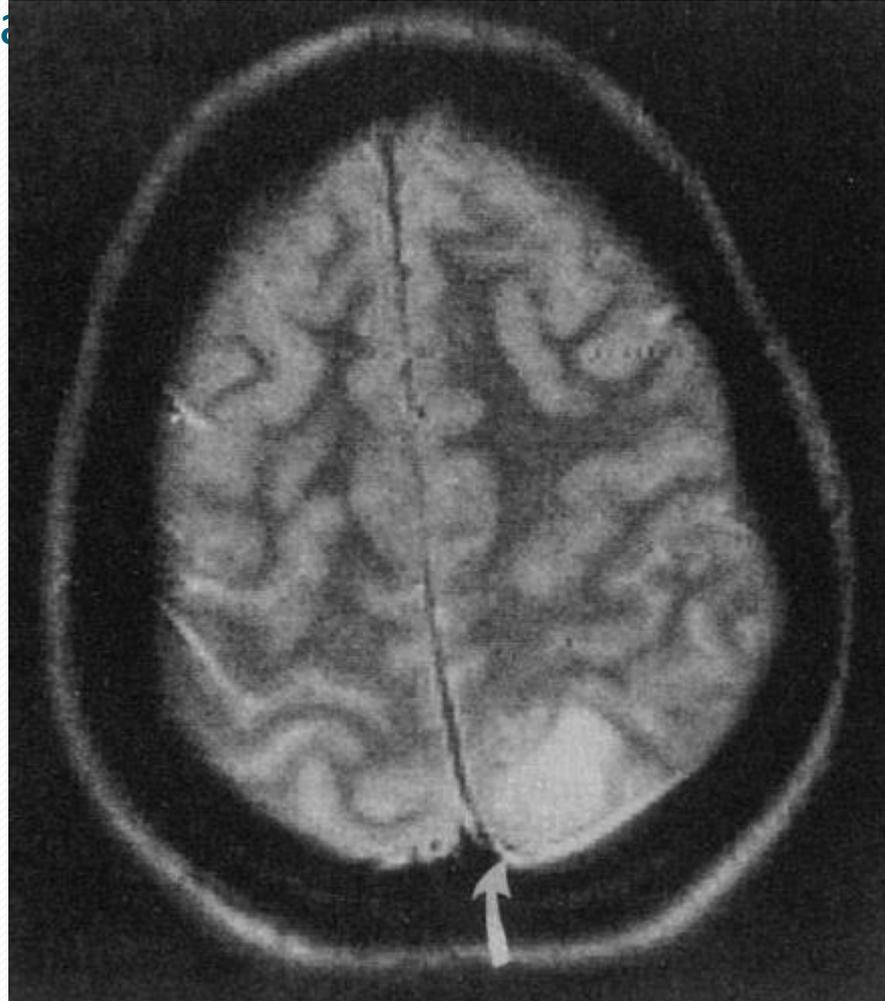
Gross liver specimen from a woman with preeclampsia who died from severe acidosis and liver failure. Periportal hemorrhagic necrosis was seen microscopically.



Computed tomographic scan of liver showing a subcapsular hematoma with a peripheral hyperdense rim corresponding to more recent hemorrhage.



Magnetic resonance imaging in a 22-year-old woman with eclampsia who had cortical blindness for 96 hours. A high-signal lesion (*arrow*) is a



At postnatal review (6–8 weeks after birth)

1. Offer medical review.
2. Offer specialist referral if antihypertensive treatment still needed.
3. Repeat platelet count, transaminases and serum creatinine measurements if indicated.
4. Carry out a urinary reagent-strip test. If proteinuria \geq 1+:
 - – offer further review at 3 months to assess kidney function
 - – consider offering referral for specialist kidney assessment.

- Three short-acting antihypertensive agents

- hydralazine

- short-acting [sublingual or orally administered] nifedipine

- labetalol

→ commonly used control acute very high blood pressure in women with severe hypertension in pregnancy who may require emergency cesarean section and often receive magnesium sulfate

- First line drugs include
 - methyldopa
 - labetolol
- Second line agents are
 - hydralazine,
 - nifedipine
 - prazosin

Hydralazine

- Dose: 5-10 mg every 20 minutes IV
- Onset: 10-20 minutes
- Duration: 3-8 hours
- Side effects: headache, flushing, tachycardia, lupus like symptoms
- Mechanism: **peripheral vasodilator**
- **First Choice for severe PIH**

Labetalol

- Dose:
 - IV: 20mg, then 40, then 80 every 20 minutes, for a total of 220mg
 - Oral 100 mg bid to be increased up to 200 mg qid. (maximum 2400mg daily)
- Onset: 1-2 minutes
- Duration: 6-16 hours
- Side effects: hypotension
- Mechanism: **Alpha and Beta blocker**
- Used extensively in pregnancy. Has favorable effects.

Nifedipine

- Dose: 10 mg po, not sublingual
- Onset: 5-10 minutes
- Duration: 4-8 hours
- Side effects: chest pain, headache, tachycardia
- Mechanism: **CA channel block**
- Required by midwives and nurses in the absence of a doctor

Clonidine

- Dose: 1 mg po
- Onset: 10-20 minutes
- Duration: 4-6 hours
- Side effects: unpredictable, avoid rapid withdrawal
- Mechanism: Alpha agonist, works centrally

Nitroprusside

- Dose: 0.2 – 0.8 mg/min IV
- Onset: 1-2 minutes
- Duration: 3-5 minutes
- Side effects: cyanide accumulation, hypotension
- Mechanism: direct vasodilator

Alpha-methyl Dopa

- The most commonly used and presumably the safest with pregnancy.
- The usual dose starts with 250mg tds to be increased up to 2 grams per day.
- It blocks the adrenaline release at post synaptic sites.

Moderate and high risk of pre-eclampsia

Antenatal care and fetal monitoring

- 
- If previous severe eclampsia /pre-eclampsia needing birth before 34 weeks pre-eclampsia **OR** with baby's birth weight < 10th centile **OR** intrauterine death **OR** placental abruption , Carry out
 - ultrasound fetal growth and amniotic fluid volume assessment + umbilical artery
 - doppler velocimetry.
 - Start at 28–30 weeks, or at least 2 weeks before previous
 - gestational age of onset of hypertensive disorder if earlier than 28 weeks.
 - Repeat 4 weeks later.

- 
- If at least **two moderate** risk factors or at least **one high risk factor** for pre-eclampsia,
 - Advise woman to take aspirin 75 mg/day from 12 weeks until birth.
 - If fetal activity abnormal, carry out cardiotocography.

Pre-eclampsia

Management in various
periods of pregnancy



Antenatal care

Mild hypertension (BP 140/90–149/99 mmHg)

1. Do not treat hypertension.
2. Measure BP at least 4 times a day.
3. Test kidney function, electrolytes, FBC, transaminases, bilirubin 2 times a week.

Moderate hypertension (BP 150/100–159/109 mmHg)

- Treat with first-line oral labetalol to keep BP < 150/80–100 mmHg.
- Measure BP at least 4 times a day.
- Test kidney function, electrolytes, FBC, transaminases, bilirubin 3 times a week.

Timing of birth

□ Before 34 weeks

1. Manage conservatively (do not plan same-day delivery of baby).
2. Consultant obstetric staff to:
 - – document maternal (biochemical, haematological and clinical) and fetal indications for elective birth before 34 weeks
 - – write plan for antenatal fetal monitoring.
3. Offer birth (after discussion with neonatal and anaesthetic teams and, if required, course of corticosteroids completed) if:
 - – severe refractory hypertension
 - – maternal or fetal clinical indication develops as defined in plan.

Mild and moderate hypertension (140/90–159/109 mmHg)

1. Measure BP hourly.
2. Continue antenatal hypertensive treatment.
3. Carry out haematological and biochemical monitoring according to criteria from antenatal period, even if regional analgesia being considered.
4. Do not routinely limit duration of second stage of labour if BP stable



Management of severe hypertension (Eclampsia)

1. Measure BP continually.
2. Continue antenatal hypertensive treatment.
3. If BP controlled within target ranges, do not routinely limit duration of second stage of labour.
4. If BP does not respond to initial treatment, advise operative birth.
5. Treat women admitted to critical care during pregnancy or after birth immediately with one of:
 - labetalol (oral or intravenous)
 - hydralazine (intravenous)
 - nifedipine (oral).
6. Monitor response to treatment to:
 - ensure blood pressure falls
 - identify adverse effects for woman and fetus
 - modify treatment according to response.
7. Consider using ≤ 500 ml crystalloid fluid before or at same time as first dose of hydralazine in antenatal period.
8. Aim to keep BP $< 150/80$ – 100 mmHg.



Anticonvulsants

- Give intravenous **magnesium sulphate** if woman with severe hypertension or severe pre-eclampsia has or previously had eclamptic fit.
- Consider giving intravenous magnesium sulphate if birth planned within 24 hours in woman with severe pre-eclampsia.
- **Do not use diazepam, phenytoin or lytic cocktail as alternatives to magnesium sulphate* in women with eclampsia.**



Regimen for magnesium sulphate

Loading dose

4 g given intravenously slowly over 5-10 minutes, followed by infusion of 1 g/hour for 24 hours.

OR

10gm deep IM 5gm in each buttock

Maintenance dose

5gm deep IM, 2.5gm in each buttock 4hrly.

Continue 24hrs after last convulsion or delivery

(Further dose of 2-4 g given over 5 minutes if recurrent seizures.)



Corticosteroids



For fetal lung maturation

- If birth likely within 7 days in woman with pre-eclampsia:
 1. give 2 doses betamethasone 12 mg intramuscularly 24 hours apart between 24 and 34 weeks
 2. consider giving 2 doses betamethasone 12 mg intramuscularly 24 hours apart at 35–36 weeks.
- **For HELLP syndrome**

Do not use dexamethasone or betamethasone to treat HELLP syndrome

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- **Caesarean section versus induction of labour**
 - Choose mode of birth according to clinical circumstances and woman's preference.



Diuretics & hyperosmotic agents



- Diuretics : deplete intravascular volume , compromise placental perfusion , limited used to pulmonary edema
- Hyperosmotic agents e.g. Mannitol , Isosorbide :-
leaks of agents through capillaries into lungs & brain
promote accumulation of edema

MANAGEMENT OF HELLP SYNDROME

Antenatal management

- If the platelet count is sufficiently low to present a hazard for operative delivery, a platelet transfusion should be considered



Thank you for attention!