

Pathogenesis of preeclampsia

- Several factors seem to contribute to the development of preeclampsia, including defective trophoblast invasion of the spiral arteries and abnormal trophoblast differentiation.

Hypertension	Systolic arterial pressure ≥ 140 mmHg OR Diastolic arterial pressure ≥ 90 mmHg
and	
Proteinuria	≥ 300 mg protein in a 24 h collection

diagnostic criteria for pre-eclampsia

Warren phase V.
A rise in blood pressure above baseline and oedema are now not usually included.
A positive dipstick test for proteinuria should be confirmed by 24 h urine collection.

risk factors:

- Preeclampsia during previous pregnancy
- Advanced maternal age
- Multiple pregnancies
- High body-mass index
- Conception before age 20
- Connective tissue disorders
- Protein C and protein S deficiencies
- Factor V Leiden mutation
- Hyperhomocysteinemia

Blood pressure	Systolic arterial pressure ≥ 160 mmHg Diastolic arterial pressure ≥ 110 mmHg
Renal	Proteinuria ≥ 2 g / 24 h Oliguria < 500 ml / 24 h Serum creatinine ≥ 0.09 mmol/l
Hepatic	Epigastric or right upper quadrant pain Elevated bilirubin and/or transaminases
Neurological	Persistent headaches Visual disturbances Convulsions (eclampsia)
Haematological	Thrombocytopenia Deranged coagulation tests Haemolysis
Cardiac/respiratory	Pulmonary oedema Cyanosis

clinical features:

- Development of new-onset hypertension (systolic BP > 140 mm Hg or diastolic BP > 90 mm Hg) during the third trimester or late second trimester in a previously normotensive pregnant woman is the first sign of preeclampsia.
- Development of proteinuria (> 300 mg/24 hours) usually follows.
- Preeclampsia may present with unusual clinical manifestations. Transient cortical blindness, frequent headaches, and transient scotomata may occur

clinical features of severe preeclampsia

laboratory features:

- The serum creatinine measurement may be within normal laboratory range, because the preeclampsia-induced decrease in filtration fraction is compensated by the pregnancy-induced increase in glomerular filtration rate.
- Hyperuricemia and hypocalciuria also may accompany preeclampsia.
- Consumption of platelets in microcoagulation, resulting in thrombocytopenia is a frequent feature of preeclampsia. The prothrombin time, partial thromboplastin, time and fibrinogen concentration are unaffected by preeclampsia, unless complicated by abruption placentae or severe hepatic involvement.

treatment:

- The definitive therapy for preeclampsia is delivery of the fetus.
- The use of antihypertensive agents to control mildly elevated systolic BP in preeclampsia has not been shown to alter the course of maternal disease; however, there is a general consensus, that systolic BP of at least 160 mmHg and diastolic BP of at least 105 mm Hg should warrant initiation of antihypertensive therapy.
- Prolonged antenatal therapy with methyldopa has stood the test of time. Calcium-channel blockers also have been used as long-term therapy with increasing confidence.
- Peripartum acute hypertensive episodes should be treated with intravenous hydralazine or labetalol. The goal is to lower systolic BP to approximately 140 to 150 mm Hg and diastolic BP to about 90 to 100 mm Hg
- Magnesium sulfate ($MgSO_4$) is the preferred drug for preventing seizures.
- The safety and efficacy of $MgSO_4$ have been demonstrated in the MAGPIE trial, in which 10,000 pregnant women with BP of at least 140/90 mm Hg and proteinuria of at least 1+ were treated with intramuscular $MgSO_4$ or placebo. Major findings include the following:
 - (1) a significantly reduced risk for convulsions in $MgSO_4$ recipients, regardless of the severity of preeclampsia, gestational age, or parity; and
 - (2) a trend toward decreased maternal mortality rates

magnesium dosage in eclampsia

Intravenous regimen	Loading dose: 4–6 g over 20 min Maintenance: 1–3 g/h
Intramuscular regimen	4 g every 4 hours
Reduce dose and monitor serum concentrations in oliguria or renal failure. (1 g magnesium sulphate = 98 mg = 4.06 mmol = 8.12 mEq elemental magnesium).	

preeclampsia & eclampsia

hypertensive emergencies in pregnancy
[created by Paul Young 06/10/07]

general

- Development of hypertensive disorders is the most common medical complication during pregnancy, affecting 10% to 20% of all pregnancies worldwide.

- Causes include:

- pregnancy-induced hypertension
- preeclampsia,
- eclampsia, and
- hemolysis, elevated liver enzyme levels, low platelet count (HELLP) syndrome.

general:

- characteristics of HELLP are:

- microangiopathic haemolytic anaemia
 - elevated liver function tests (AST, ALT, LDH)
 - low platelets
 - normal APTT, PT and fibrinogen
- HELLP syndrome should not be considered a variant of disseminated intravascular coagulation (DIC), even though microangiopathic hemolytic anemia is the hallmark of both conditions. Important differences exist between HELLP syndrome and DIC. Prothrombin time, partial thromboplastin time, and serum fibrinogen levels are normal in HELLP syndrome, whereas these parameters are usually abnormal in DIC.
- Immunofluorescence studies usually reveal fibrin microthrombi and fibrinogen deposits in necrotic and normal hepatic sinusoids.

clinical manifestations:

- No clinical signs or symptoms are unique to HELLP syndrome.
- Epigastric pain and right upper quadrant pain are the most common symptoms. Significant weight gain and generalized edema are the most prominent signs.
- Severe hypertension is not a universal finding.
- Occasionally, patients may present with polyuria caused by nephrogenous DI.

differential diagnosis:

- Acute fatty liver of pregnancy
- Acute cholecystitis
- Hemolytic uremic syndrome
- Thrombotic thrombocytopenic purpura
- Perforating peptic ulcer
- Acute pyelonephritis
- Hepatic encephalopathy
- Acute viral hepatitis

treatment:

- Diagnosis of HELLP syndrome should be considered a serious complication of pregnancy. Patients should be admitted to monitored beds in the hospital.
- Administration of intravenous corticosteroids is associated with rapid improvement of laboratory and clinical parameters in some patients after delivery. Although the results are less dramatic, intravenous corticosteroid use before delivery may be beneficial.
- Plasma exchange after delivery has been used in patients with persistent HELLP syndrome and end-organ damage with variable response.

complications:

- HELLP syndrome is associated with several complications (eg, abruption placentae [16%], acute renal failure [8%], subcapsular hematoma of liver [1%])

HELLP syndrome