

## 授課講師學經歷

|            |                                                             |
|------------|-------------------------------------------------------------|
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| 課程名稱(主題)   | 產前與產後的生理體質變化 產前與產後併發症                                       |
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## 授課內容摘要

孕婦體質的改變始於受孕，受到胎兒的刺激，孕婦在解剖、生理及生化方面有多項的調適。子宮的容積及重量倍增，子宮胎盤血流也相對增加；陰道黏膜增厚，較具彈性。皮膚出現妊娠紋，並有色素沉著、乳房變大、乳暈變深。體內水分留滯導致血清滲透壓下降，體重的上升來自於子宮、胎盤、羊水、胎兒、增加的血量。正常 BMI 的孕婦，體重增加控制在 11—16 公斤，由於胰島素及胎盤 lactogen 分泌增加，加上周邊組織對胰島素拮抗，孕婦空腹時有低血糖，飯後高血糖的現象。血量增加 40—50%，紅血球也增加 450ml，免疫力下降，白血球功能也受到影響，大多數的凝血因子也上升，全身血管阻力下降，使脈搏、心輸出量升高，在懷孕中期血壓是最低的。由於下肢靜脈壓升高，併血液回流受阻，導致下肢水腫、靜脈曲張。呼吸速率及潮氣量增加、腎絲球過濾率及腎臟血量上升，常有夜尿、頻尿、及尿失禁。腸胃排空變慢、胃酸逆流、心口灼熱，血中白蛋白下降，球蛋白上升，泌乳素上升 10 倍，游離 T4 在妊娠週 6—9 週達到高峯，第二孕期恢復正常。背部酸痛肇因於骨盆關節鬆動。孕婦常有注意力不集中，健忘與失眠。

生產後 6 週，婦女體內也有巨大變化，子宮外的血管管徑縮小，子宮內的大血管也被小血管取代。子宮頸管徑變小，然而兩側凹痕仍存。在產後 4 週子宮的體積也回復原來的大小，產後 2 週子宮下降到真骨盆腔，宮縮痛在哺乳時特別明顯，由於泌乳素刺激的緣故。惡露顏色由鮮紅變淡至米白色，持續至產後 4—8 週。子宮內膜再生在產後 3 週完成，留置胎盤及子宮發炎造成子宮恢復不完全，惡露持續排出及不正常的出血。膀胱容量變大及對膀胱內壓不敏感，導致排尿不完全。除了銀白色的妊娠紋，腹部在數週內恢復產前的形狀。心跳速率及心輸出量在產後 2 週恢復。體重在產後 6 個月內下降，接近孕前體重。

常見的產前併發症有子癇前症及妊娠糖尿病。懷孕中期開始的血壓過高，妊娠高血壓及子癇前症就佔了 70%，後者除了高血壓還併有蛋白尿。孕婦血管疾病、胎盤血管發育不良、基因及免疫問題等因素，導致子宮胎盤血流灌注不良，活化內皮細胞，進而導致血管痙攣，血管通透性增加，凝血系統活化。臨床表現如血小板過低、溶血、肺水腫、肝指數上升、小便減少、顱內出血、頭痛、視力模糊、胎盤早期剝離、胎兒生長遲滯、羊水過少等。目前並無有效預防或篩檢子癇前症的方法。處理的原則為早期偵測出嚴重的子癇前症，也就是對母胎作嚴密的監控。治療的目標是最適當生產的時機與方式，讓母胎均安。治療的方式是預防痙攣，控制血壓，生產是最終的治療。產後 6 週血壓大都恢復正常，如高血壓持續超過 12 週，表示演變成慢性高血壓。子癇前症的復發率是 20%。

妊娠糖尿病是懷孕中期血糖過高，盛行率 2—6%。併發症包括羊水過多、胎兒過大、早期破水、

早產、產後大出血、胎兒在生產時損傷、出生後低血糖、低血鈣、易黃疸等。篩檢在懷孕 24—28 週進行，50 公克耐糖試驗如超過 130—140mg/dl，進一步做 100 公克耐糖檢查，如超過標準值，就確立診斷為妊娠糖尿病。70%的妊娠糖尿病可經由飲食運動加以控制，只有 30%需要胰島素治療。產後哺餵母乳有助於血糖控制，建議在產後 6—12 週做 75 公克耐糖檢查。妊娠糖尿病的復發率為 33—50%。在下次懷孕前 3 個月，建議血糖宜控制良好並補充葉酸錠。

產後併發症最常見是產後大出血，原因為子宮收縮不良、產道裂傷及留滯胎盤。處理的原則是穩定產婦的生理徵象，確定出血原因並加以治療。如施打子宮收縮藥物，修補傷口，取出殘餘胎盤，結紮供應子宮的血管，或行經動脈血管栓塞術等。

## **Prenatal & Postnatal Physiologic Changes**

### **Prenatal Complications**

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## **Outline**

- Maternal adaptations to pregnancy
- Puerperium changes
- Obesity in pregnancy: Complications
- Gestational diabetes
- Gestational hypertension & Preeclampsia

## **Maternal Adaptations to Pregnancy**

### **Maternal adaptation to pregnancy**

- Anatomical, physiological & biochemical
- Begin soon after fertilization & evolve through pregnancy
- In response to physiological stimuli by fetus
- Accommodate the need of maternal-fetal unit
- Return to the pre-pregnant state several weeks after delivery

## Uterus & vagina

- Uterine hypertrophy
  - stimulated by estrogen +/- progesterone, products of conception
  - weight: 70gm → 1100gm
  - volume: 10ml → 5-20 L
  - height: extrapelvis (gestation 12 weeks) → umbilicus (gestation 20 weeks)
- Vagina
  - hyperemia & with copious secretion
  - thickened mucosa, loosened connective tissue & hypertrophied smooth muscle cell

## Breast

- Increase in size
- Areola: broader & pigmented
- Clostrum
- Volume of milk production **not** correlated with prepregnancy breast size

## Skin

- Abdominal wall
  - reddish, glistening & silvery streak
  - diastasis recti
- Pigmentation
  - linked to increased melanocyte-stimulating hormone level
  - linea nigra, chloasma or melasma gravidarum, areolae & genital skin
- Vascular spiders & palmar erythema

## Weight gain

- Uterus, placenta, fetus & amniotic fluid(AF)
- Breast, blood volume & extracellular fluid
- Increased water retention
  - fall in plasma osmolality 10 mOsm/L
  - water content of fetus, placenta & AF: 3.5L
  - blood volume, breast and uterus: 3.0L
  - edema of ankle & leg: 1.0L

## Recommended weight gain for woman with singleton pregnancy

| Prepregnancy BMI      | Total gain (kg) |
|-----------------------|-----------------|
| Low ( BMI<19.8)       | 12.5-18         |
| Normal( BMI: 19.8-26) | 11.5-16         |
| High ( BMI: 26-29)    | 7-11.5          |
| Obese ( BMI>29)       | <u>≥7</u>       |

## Fat metabolism(1)

- Hypertriglyceridemia
  - increased TG synthesis (increased hepatic lipase activity)
  - decreased TG catabolism (decreased lipoprotein lipase in adipose tissue)
  - provide maternal fuels
- Apolipoproteins A-I, A-II, and B rise across gestation
- HDL-cholesterol levels initially increase, then fall in the third trimester
- Lipid stored in 2nd trimester is consumed in 3rd trimester

## Lipoprotein concentrations in pregnancy

| Type of lipoprotein | Nonpregnant* | Early pregnancy* | Late pregnancy* |
|---------------------|--------------|------------------|-----------------|
| Total triglycerides | 60           | 75 to 100        | 210             |
| Total cholesterol   | 170          | 175 to 200       | 250             |
| VLDL cholesterol    | 10           | 10               | 25              |
| LDL cholesterol     | 105          | 100 to 125       | 150             |
| HDL cholesterol     | 55           | 55 to 75         | 65              |

\* Units are mg/dL; divide cholesterol values by 38.5 and triglycerides by 88.6 to convert to mmol/L.  
Data from: Fahraeus, L, Larsson-Cohn, U, Wallentin, L. *Obstet Gynecol* 1985; 66: 668.

## Fat metabolism(2)

- Leptin
  - secreted by adipose tissue & placenta
  - maternal serum level peaks in 2nd trimester & remained elevated till delivery
  - linked to preeclampsia, gestational diabetes, fetal growth restriction

## Carbohydrate metabolism(1)

- Fasting hypoglycemia
  - increased storage of tissue glycogen
  - increased peripheral tissue utilization
  - decreased hepatic glucose production
  - continued glucose consumption by fetus
- Postprandial hyperglycemia
- Hyperinsulinemia
  - hyperplasia of insulin-secreting pancreatic beta cell

## Carbohydrate metabolism(2)

- Peripheral resistance to insulin
  - diabetogenic hormones e.g. growth hormone, corticotropin-releasing hormone, chorionic somatomammotropin (also called human placental lactogen )
- Insulin resistance and relative hypoglycemia resulting increased lipolysis, allow mother
  - preferentially use fat (free fatty acid, ketones, triglyceride)
  - preserve glucose & amino acid for fetus
  - lower protein catabolism
- Placenta is permeable to glucose, amino acid and ketone body, but not large lipid

## Blood

- Increased blood volume 40-45%
  - to meet the demand of hypertrophied uterus & vascular system
  - to protect the mother & fetus against impaired venous return in supine position
  - to protect the mother against blood loss during parturition
- Increased RBC for 450ml

## Iron metabolism

- Total iron content (adult woman): 2.0-2.5gm
- Total iron required (normal pregnancy): 1000mg
  - 300mg (actively transfer to) uterus & placenta
  - 200mg : obligatory loss
  - 500mg: for increase in erythrocytes
- Iron replacement is necessary during pregnancy
  - 30mg/day

Normal iron indices during pregnancy

|                                    |                |
|------------------------------------|----------------|
| Plasma iron                        | 40-175 mcg/dL  |
| Plasma total iron-binding capacity | 216-400 mcg/dL |
| Transferrin saturation             | 16-60 percent  |
| Serum ferritin                     | >10 mcg/dL     |

## Immunological & leukocyte function

- Depressed
  - humoral & cellular immunity
  - antibody against HSV, influenza
  - polymorphonuclear WBC chemotaxis & adherence function
- Improved: autoimmune disease
- WBC count: 5000-12000/ $\mu$ L
- CRP: gestation age independent

## Heart & circulation

- Decrease: systemic vascular resistance
- Increase: pulse rate, stroke volume, cardiac output
- BP lower to nadir at midpregnancy
- Retarded blood flow & increased lower venous pressure, resulting in
  - dependent edema
  - varicose vein in legs, vulva & hemorrhoid

## Respiratory tract

- Increase
  - respiratory rate
  - tidal volume
  - minute ventilatory volume
  - minute oxygen uptake
  - vital capacity
  - inspiratory capacity
- Decrease
  - residual volume
  - functional residual capacity

## Kidney

- Increase in glomerular filtration rate & renal plasma flow, →
  - decrease BUN & Cr
  - increase excretion of protein, amino acid & glucose
- Decrease
  - renal bicarbonate threshold →
    - serum  $\text{HCO}_3^-$  4-5mEq/L lower
    - serum  $\text{Pco}_2$  10mm Hg lower
  - threshold for AVP release & thirst →
    - serum osmolality decrease 10 mOsm/L

## Digestive system

- Slower gastric emptying & intestinal transit
- Regurgitation of acidic gastric contents
- Hemorrhoid
- Alkaline-P : doubled
- GOT, GPT, GGT, bilirubin: decreased
- Albumin decreased, globulin increased
- Impaired contraction of gallbladder resulting cholestasis

## Pituitary glands

- Increase in size of pituitary gland
- Increased prolactin secretion stimulated by estrogen and TRH (thyrotropin-releasing hormone)

## Thyroid glands

- Free T4: plateau at gestation 6-9th weeks, return to normal after first trimester
- T3 plateau since 18th weeks
- Increase TBG (thyroxine binding globulin)

## Puerperium Changes

## Uterus & vagina

- Reduction in size of uterus & vagina
- Vaginal rugae reappear (3rd week)
- Relaxed vaginal outlet due to
  - extensive laceration
  - stretching of the perineum
  - changes in pelvic support

## Peritoneum & abdominal wall

- Broad & round ligaments
- Abdominal wall soft and flaccid due to
  - rupture of elastic fibers
  - prolonged distention by pregnant uterus
- Silvery striae usually persist
- Diastasis recti

## Blood & fluid

- Return to prepregnant level (2 weeks after delivery)
  - blood volume
  - stroke volume, heart rate, cardiac output
  - plasma fibrinogen
- Body weight reduced to nearly prepregnant weight (6 months postpartum)

## Complications of Obesity in Pregnancy

## Antenatal complications

- Spontaneous abortion
- Congenital anomalies (neural tube defect)
- Gestational hypertension/preeclampsia
- Gestational diabetes
- Preterm delivery
- Intrauterine fetal demise
- Nonalcoholic fatty liver disease
- Sleep apnea

## Peripartum complications

- Instrumental & cesarean deliveries
- Anesthesia complications
- Hemorrhage
- Infection/wound breakdown
- Deep vein thrombophlebitis
- Longer hospitalization

## Fetus/neonate complications

- Fetal macrosomia
- Increased neonatal ICU admission
- Fetal obesity
- Childhood obesity

## Gestational Diabetes

## Definition & prevalence

- Carbohydrate intolerance that begins or is first recognized during pregnancy
- 1.4-2.8% (low risk group), 3.3-6.1% (high risk group)
- 90% of all pregnancies complicated by diabetes

### \*\*\*Pregestational (overt) diabetes

--increased risk of vasculopathy, abortion & fetal congenital abnormalities

## White's classification of diabetes complicating pregnancy

| <u>Class</u> | <u>Onset</u> | <u>Fasting plasma glucose(mg/dL)</u> | <u>2-hr postprandial glucose(mg/dL)</u> | <u>Therapy</u> |
|--------------|--------------|--------------------------------------|-----------------------------------------|----------------|
| A1           | gestational  | <105                                 | <120                                    | diet           |
| A2           | gestational  | >105                                 | >120                                    | insulin        |

| <u>Class</u> | <u>Age of onset(yr)</u> | <u>Duration(yr)</u> | <u>Vascular disease</u>   | <u>Therapy</u> |
|--------------|-------------------------|---------------------|---------------------------|----------------|
| B            | over 20                 | <10                 | none                      | insulin        |
| C            | 10-19                   | 10-19               | none                      | insulin        |
| D            | before 10               | >20                 | benign retinopathy        | insulin        |
| F            | any                     | any                 | nephropathy               | insulin        |
| R            | any                     | any                 | proliferative retinopathy | insulin        |
| H            | any                     | any                 | coronary artery disease   | insulin        |
| T            | any                     | any                 | renal transplantation     | insulin        |

## Carbohydrate metabolism in pregnancy

- Normal pregnancy is a diabetogenic state:
  - mild fasting hypoglycemia
  - postprandial hyperglycemia
  - hyperinsulinemia
  - fasting hypertriglyceridemia, hypercholesterolemia, high free fatty acid
- Pregnancy-induced peripheral resistance to insulin
  - ensure sustained or maintained postprandial supply of glucose to fetus

## Maternal complications

- Hypertensive disorders
- Polyhydramnios, premature rupture of membrane
- Infection
- Cesarean section, third- or fourth-degree laceration
- Postpartum hemorrhage
- GDM in future pregnancy: 33-50%
- Overt DM(type II) in next 20 years: 50%

## Fetal complications

- Fetal macrosomia
- Preterm birth
- Intrauterine fetal death
- Shoulder dystocia, birth trauma ( brachial plexus injury & clavicular fracture )
- Respiratory distress syndrome
- Hypoglycemia, hypocalcemia, hypomagnesiemia, polycythemia, hyperbilirubinemia
- Obesity, glucose intolerance & DM in late adolescence & early adulthood

## Screening of GDM

- Universal screening
- 50-g, 1-hour oral glucose challenge test (OGCT)
- 24-28 weeks of gestation
- Pure glucose load of 50g in 150ml of fluid
- Regardless to time elapsed since the last meal
- Serum threshold: 130-140mg/dL
  - \* proposed by ADA & ACOG(2001)

## Diagnosis of GDM

-- 100-g, 3-hour oral glucose tolerance test (OGTT)

| <u>Status</u> | <u>Plasma level (mg/dL)</u><br><u>(ADA*)</u> | <u>Plasma level (mg/dL)</u><br><u>(NDDG**)</u> |
|---------------|----------------------------------------------|------------------------------------------------|
| Fasting       | 95                                           | 105                                            |
| One hour      | 180                                          | 190                                            |
| Two hours     | 155                                          | 165                                            |
| Three hours   | 140                                          | 145                                            |

-- 2 criteria must be met or exceeded for diagnosis

-- normal GTT + high risk, follow-up GTT at 32-34 wks to diagnose late-onset GDM

\* : American Diabetes Association, Carpenter & Coustan; 4th International workshop-conference on GDM

\*\* : National Diabetes Data Group

## Blood glucose monitoring

- Blood glucose reflectance meter
- Threshold
  - fasting : < 95mg/dL
  - 2-hour postprandial : <120 mg/dL
- High fasting value
  - related to intrauterine fetal death
- High postprandial value
  - related to fetal macrosomia

## Medical nutritional therapy(1)

- Goals:
  - maintain appropriate nutrition & weight gain
  - achieve euglycemia
  - avoid ketosis
- Individualized nutrition program
- 70% become euglycemic after MNT

## Medical nutritional therapy(2)

- Calorie
  - 30 kcal/kg\*/d (non-obese) \*pre-pregnancy weight
  - 25 kcal/kg/d (BMI > 30)
- Carbohydrate: 40-50% (complex, high-fiber carbohydrate)
- Protein : 20%
- Fat : 30-40% ( 90% unsaturated fat )
- Breakfast : 10-15%
- Lunch : 20-30%
- Dinner : 30-40%
- 3 snacks : 5-10% respectively

## Medical nutritional therapy(3)

- Avoid concentrated sweet
- Avoid convenience food
- Eat small frequent meals
- Eat a very small breakfast
- Choose high-fiber foods
- Lower fat intake

## Maternal ketosis

- Caused by
  - low carbohydrate intake
  - low calorie intake
  - skipped meals/snacks >10 hours
- Related to
  - lower children's psychomotor development & intelligence
  - fetal loss

## Exercise

- Aim
  - reduce body weight
  - improve glucose metabolism
  - improve cardio-respiratory fitness
- Ways
  - upper body muscle
  - three 20-minutes aerobic exercise weekly, brisk walking
- Caution
  - palpate the uterus during & after exercise

## Insulin therapy

- 30% of GDM after MNT need insulin
- Indications
  - fasting glucose > 95mg/dL OR
  - 1-hr postprandial > 140mg/dL OR
  - 2-hr postprandial > 120 mg/dL
- 0.8-1.2U/kg/day
- Short or rapid-acting insulins
  - reduce glucose elevations associated with eating
- Longer-acting insulins
  - restrain hepatic glucose production between meals & in fasting state
- Reduce macrosomia from 36% to 10%

## Education to patient

- Self-administration of insulin
- Self monitoring of blood glucose
- Dietary adjustment
- Recognition, avoidance & treatment of hypoglycemia
  - <35mg/dL by glucometer
  - a glass of milk is preferable
  - glucagon on hand for severe hypoglycemia

## Breastfeeding

- Encourage breastfeeding
- Additional 500kcal are given as 100g of carbohydrate & 20g of protein
- Becomes hypoglycemia immediately after nursing & first week of delivery

## Postpartum screening

- 6-12 weeks after delivery

|          | <u>FPG*(mg/dL)</u> | <u>75-g,2-h PG^(mg/dL)</u> |
|----------|--------------------|----------------------------|
| Normal   | <110               | <140                       |
| Impaired | 110-125            | 140-199                    |
| Diabetes | >126               | >200                       |

\*: fasting plasma glucose, ^: plasma glucose

- 75-g,2-h OGTT normal: reassess at 3 years interval & before next pregnancy
- Impaired glucose tolerance: reassess annually

## Postpartum counseling(1)

- Increased risk of developing overt DM, 50% vs control, 7%
- Recurrent GDM (33-50%)
- Diet, exercise, weight reduction & maintenance
- With weight reduction, risk of overt DM is 25%
- Avoid medication that worsen insulin resistance, e.g. glucocorticoid & nicotinic acid

## Postpartum counseling(2)

- Contraception with low dose estrogen-progesterone oral contraceptives for women without vasculopathy and smoking
- Maintain euglycemia 3 months before next pregnancy
- HbA<sub>1c</sub> with 2 SD of the mean for the reference laboratory
- Takes folic acid 0.4mg/d

## Gestational Hypertension & Preeclampsia

## Gestational Hypertension & Preeclampsia

- Incidence: 6-8% of all pregnancies
- 70% of women with hypertension during pregnancy
- Gestational hypertension
  - BP  $\geq$  140/90mmHg for first time during pregnancy
  - BP returns to normal < 12 weeks' postpartum
- Preeclampsia
  - BP  $\geq$  140/90mmHg after 20 weeks' gestation
  - proteinuria  $\geq$  300mg/24 hr or  $\geq$ 1+ dipstick

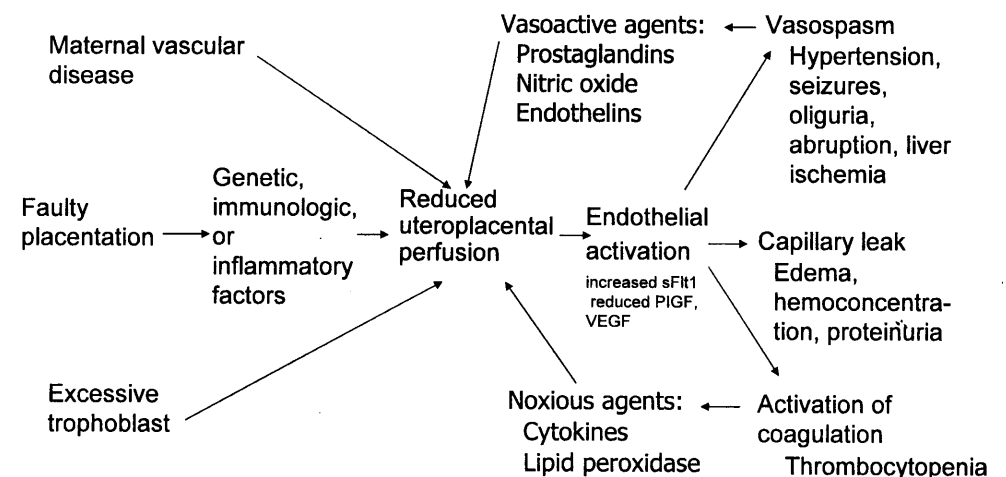
## Other hypertensive disorders in pregnancy

- Eclampsia
  - New-onset grand mal seizure that cannot be attributed to other causes in a woman with preeclampsia
- Chronic hypertension
  - BP  $\geq$  140/90mmHg before pregnancy or diagnosed before 20 weeks gestation not attributable to gestational trophoblastic disease or
  - Hypertension first diagnosed after 20 weeks' gestation and persistent after 12 weeks' postpartum
- Preeclampsia superimposed on chronic hypertension

## Risk factors

- Nulliparity
- Multifetal gestation
- Obesity
- Family history of preeclampsia-eclampsia
- Preeclampsia in a previous pregnancy
- Abnormal uterine Doppler studies at 18-24 wk
- Pregestational diabetes mellitus
- Presence of thrombophilias
- Hypertension or renal disease

## Pathophysiology of hypertensive disorders in pregnancy



## Vascular & hematologic changes

- Hemoconcentration
- Vasospasm
- Capillary leak & decreased colloid oncotic pressure
- Thrombocytopenia & hemolysis
- Disseminated intravascular coagulopathy

## Pulmonary & hepatic changes

- Pulmonary edema & cyanosis
- Elevated liver enzymes (GOT & GPT)
- Hyperbilirubinemia
- Periportal hemorrhage of liver
- Subcapsular hematoma
- Hepatic rupture

## HELLP syndrome

- 20% of severe preeclampsia
- **Hemolysis**: bilirubin  $\geq 1.2\text{mg/dL}$ , LDH  $\geq 600\text{ IU/L}$ , abnormal peripheral blood smear
- **Elevated Liver enzymes**: GOT  $\geq 2\text{X}$  normal
- **Low Platelet**:  $< 100,000/\mu\text{L}$
- Associated with placental abruption, renal failure, subcapsular hepatic hematoma, recurrent preeclampsia, preterm delivery, and even fetal or maternal death

## Renal changes

- Oliguria ( $< 500\text{mL}$  in 24 hours)
- Secondary to vasospasm, hemoconcentration & decreased renal blood flow
- Acute tubular necrosis
- Acute renal failure

## Neurologic & cerebral manifestations

- Eclampsia
- Intracranial hemorrhage
- Infarction
- Vasogenic edema
- Transtentorial herniation
- Temporary blindness
- Headache, blurred vision, scotomata, hyperreflexia

## Fetal changes

- Secondary to impaired uteroplacental blood flow or placental infarction
- Fetal growth restriction
- Oligohydramnios
- Placental abruption
- Nonreassuring fetal status

## Prediction & prevention

- No reliable and cost-effective screening test
- No effective prevention

## Maternal surveillance

- **Goals:** early detection of severe preeclampsia and end-organ dysfunctions
- **Symptoms:** altered mentation, severe headache, nausea or vomiting, visual changes, shortness of breath, RUQ or epigastric pain & decreased urine output
- **Signs:** vital signs, visual acuity, visual field, basilar rales, abdominal tenderness, edema, deep tendon reflex
- **Laboratory tests**
  - hematocrit, platelet, liver transaminases, serum creatinine, 24-hour urine protein, lactate dehydrogenase, bilirubin, peripheral smear, coagulation profile( PT, APTT, fibrinogen, D-dimer), electrolytes, ABG, CXR(PA), brain CT

## Fetal surveillance

- Fetal movement counts
- Nonstress test (NST)
- Biophysical profile (BPP)
- Ultrasound

## Management objectives

- Termination of pregnancy with the least possible trauma to mother & fetus
- Birth of an infant who subsequently thrives
- Complete restoration of health to the mother

## Management strategies

- Recognize the disease (severity & end-organ involvement)
- Prevent eclampsia
- Control hypertension
- Determine timing & route of delivery

## ***DELIVERY***

### **Ultimate Cure of Preeclampsia**

# Postpartum management

- Monitor vital signs, fluid intake & urine output
- MgSO<sub>4</sub> for at least 24 hours
- Antihypertensives if BP > 155/105 mmHg
- BP usually normalize by 6 weeks postpartum; becomes chronic hypertension if BP still high beyond 12 weeks postpartum
- Counseling: risk of hypertensive complications in future pregnancy (20%)