# PATHOPHYSIOLOGY AND RELATIONSHIP TO CLINICAL PICTURE IN PREECLAMPSIA

Mehnas viji

Roll no 55

- The pathophysiological changes are the consequence of vasospasm,endothelial dysfunction and ischaemia.
- The system commonly involved are renal,hepatic and haematological systems.

# KIDNEY

- The main pathology in kidney is glomerular and tubular dysfunction and glomerular endotheliosis ie fibrin deposition.
- There will be proteinuria due to increased capillary permeability.
- Hyperurecimia due to renal tubular dysfunction and can be due to placental ischaemia.

- Glomerular dysfunction causes a reducction in the glomerular filtration rate and creatinine clearance, which in severe case causes in increase in blood urea and creatinine.
- Renal failure is usually found in association with HELPP syndrome ,abruptness sepsis.

# LIVER

- Periportal thrombosis and fibrin deposition, haemorrhages and necrosis are seen in the liver.
- Increase in the liver enzymes (SGOT and SGPT). Transaminase levels above 70 IU/L are considered significant and levels above 150 IU/L are associated with increased morbidity to the mother
- Liver changes are responsible for nausea and vomiting.

- Small haemorrhages coalesce to form a subcapsular haematoma, which cause stretching of Glisson's capsule and epigastric pain.
- This is a very serious sign and seen in impending eclampsia.
- The changes in the liver are responsible for HELLP syndrome.
- An extremely rare but catastrophic complication is liver rupture.

# CARDIOVASCULAR SYSTEM

- There are three major changes in the cardiovascular system: (i) the increased cardiac afterload caused by hypertension:
- (ii) the diminished cardiac preload due to the diminished hypervolaemia of pregnancy in preeclampsia, which can be increased iatrogenically by intravenous crystalloids or colloids;
- (iii) endothelial cell activation with increased capillary permeability which causes extravasation of fluid a from the intravascular space into the extracellular space and lungslungs leafing to pulmonary edema.

- Hemoconcentration from the generalised vasoconstriction following endothelial activation and leakage of plasma into the interstitial space.
- The haematocrit is increased because of the contracted blood volume and haemoconcentration in preeclampsia.
- low haematocrit need not always indicate improvement as it could be due to hemolysis.

# BLOOD AND COAGULATION

- Endothelial dysfunction will lead to activation of platelets and the coagulation system by tissue factor on the endothelium. This causes widespread subclinical to frank DIC, resulting in consumption of clotting factors and platelets and may manifest as thrombocytopaenia.
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- Severe preeclampsia is also frequently accompanied by microangiopathic haemolysis caused by endothelial cell damage with platelet adherence and fibrin deposition. This can be demonstrated by the presence of schistocytes, Burr cells and fragmented red cells in peripheral blood and also by elevated lactate dehydrogenase levels. Platelet count <1,00,000 can be associated with coagulation abnormality

# PLACENTA

- The placental changes are central to preeclampsia.
- The typical vascular lesion is termed acute atherosis of the decidual arteries. Decidual segments of the uteroplacental arteries is characterised by fibrinoid necrosis, macrophages and mononuclear cell infiltration.
- It will lead to fetal growth restriction, oligohydramnios, placental abruption and ultimately fetal demise.

# BRAIN

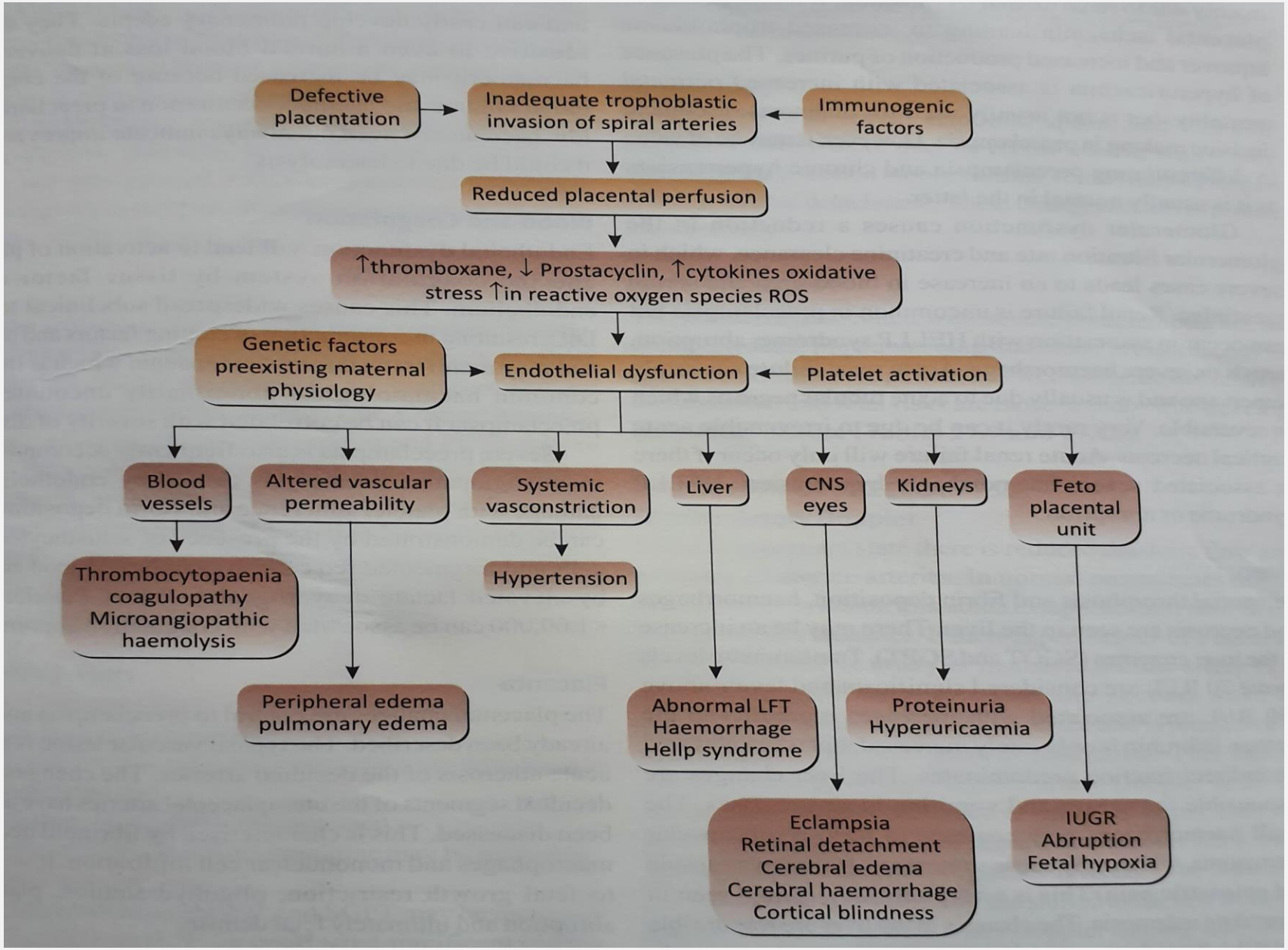
.When the differences between the cerebral intraarterial pressure and the cerebral venous pressure rises higher than 160 mm Hg, autoregulation is lost and cerebral blood flow becomes dependant on mean arterial pressure.

When the cerebral pressure increases the cerebral vessels dilate and cerebral vascular resistance decreases. This causes huge increase in cerebral blood flow and causes hypertersive encephalopathy with headache, restlessness, altered sensorium  and develop cerebral hemorrhage.

- Other cerebral signs of preeclampsia are hyperreflexia and clonus.
- Small cerebral haemorrhages, thrombosis and fibrinoid necrosis can occur in eclampsia and are secondary to endothelial dysfunction.
- Cerebral edema is also usual in eclampsia. If there is widespread cerebral edema, the patients are usually comatose. With CT imaging hypertensive lesions are seen termed as PRES or posterior reversible encephalopathy syndrome.

# EYES

- Localised retinal vasospasm is the most common finding.
- Haemorrhages and papilledema may be seen rarely in severe hypertension.
- Visual disturbances are common and are usually due to edema of the occipital lobe. Cortical blindness can rarely occur due to occipital edema and PRES may be present.
- Blindness can also occur due to involvement of lateral geniculate nuclei and retina. In the retina, the problem may be ischaemia, infarction or retinal detachment. The prognosis is usually good and vision returns normal following delivery.


Defective placentation
Inadequate trophoblastic invasion of spiral arteries
Immunogenic factors
Reduced placental perfusion
↑thromboxane, ↓ Prostacyclin, ↑cytokines oxidative stress ↑in reactive oxygen species ROS
Genetic factors preexisting maternal physiology
Endothelial dysfunction
Platelet activation
Blood vessels
Altered vascular permeability
Systemic vasconstriction
Liver
CNS eyes
Kidneys
Feto placental unit
Thrombocytopaenia coagulopathy Microangiopathic haemolysis
Hypertension
Peripheral edema pulmonary edema
Abnormal LFT Haemorrhage Hellp syndrome
Proteinuria Hyperuricaemia
Eclampsia Retinal detachment Cerebral edema Cerebral haemorrhage Cortical blindness
IUGR Abruption Fetal hypoxia

# LABORATORY TEST

- **Renal Function Tests**
- 24-hour urinary protein
- Urine for pus cells and casts (casts are indicative of renal disease)
- Serum uric acid may be increased
- Blood urea and serum creatinine may be increased

2. **Liver Function Tests**

Transaminases (SGOT and SGPT may be increased. Levels > 70 IU/L are considered significant and lev els > 150 IU/L are associated with increased morbidityto the mother.)

Bilirubin may be increased

3. **Hematological Tests**

Haematocrit may be increased (A low haematocrit need not always indicate improvement as it could be due to haemolysis)

Haemolysis on peripheral smear (Burr cells, frag- mented red cells and schistocytes)

Lactic dehydrogenase (LDH may be increased>600 u/L)

Serum haptoglobin may be increased.

Thrombocytopenia

Low fibrinogen

APTT and prothrombin is prolonged

# FUNDOSCOPY

- Fundoscopy will reveal arteriolar spasm and vascular damage. Initially there may be retinal edema, which may then progress to cotton wool exudates, retinal haemorrhages and even retinal detachment. These changes are all reversible after delivery.

# MATERNAL COMPLICATION

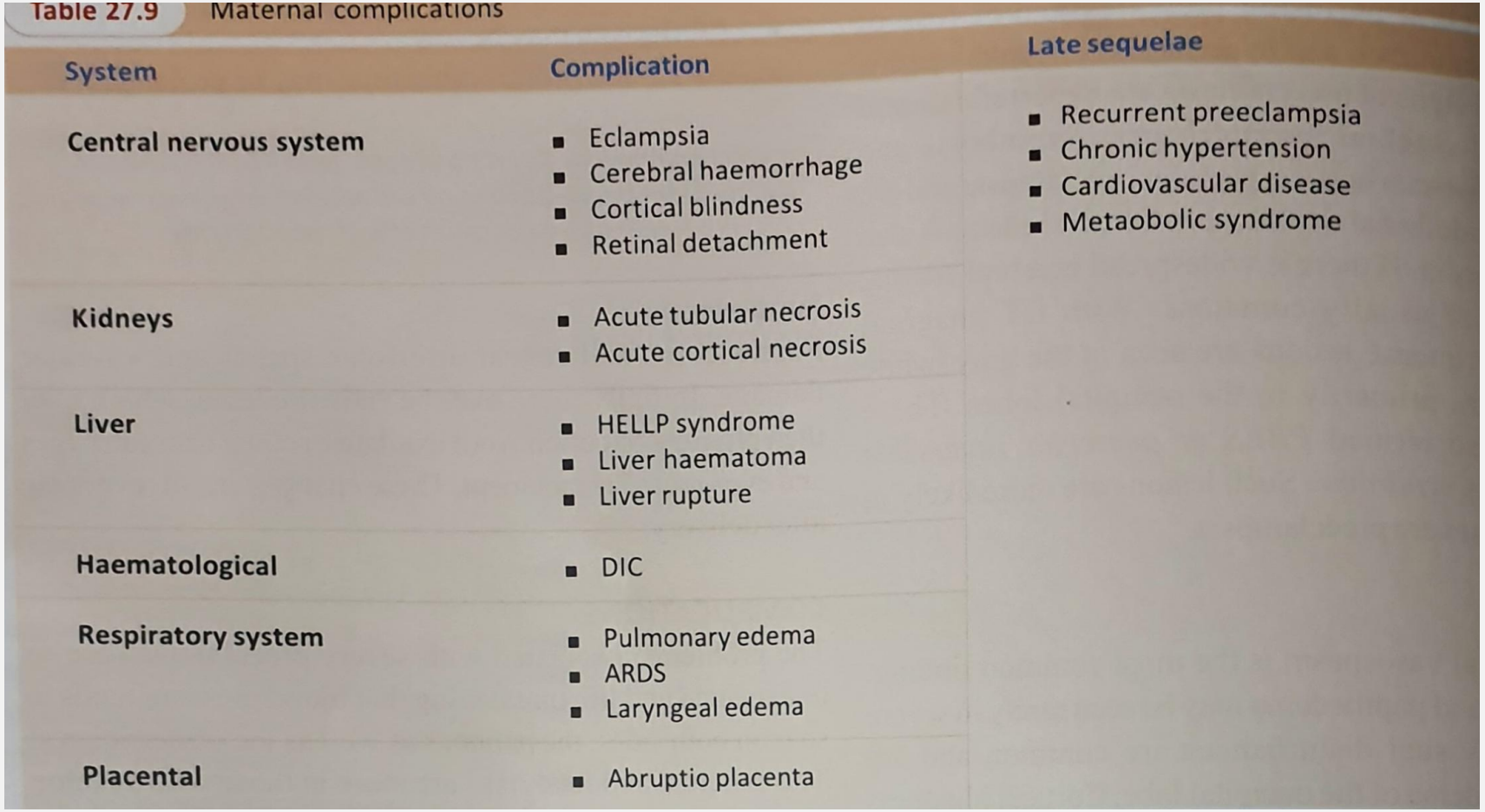


Table 27.9 Maternal complications

| System | Complication | Late sequelae |
|---|---|---|
| **Central nervous system** | ▪ Eclampsia<br>▪ Cerebral haemorrhage<br>▪ Cortical blindness<br>▪ Retinal detachment | ▪ Recurrent preeclampsia<br>▪ Chronic hypertension<br>▪ Cardiovascular disease<br>▪ Metaobolic syndrome |
| **Kidneys** | ▪ Acute tubular necrosis<br>▪ Acute cortical necrosis | |
| **Liver** | ▪ HELLP syndrome<br>▪ Liver haematoma<br>▪ Liver rupture | |
| **Haematological** | ▪ DIC | |
| **Respiratory system** | ▪ Pulmonary edema<br>▪ ARDS<br>▪ Laryngeal edema | |
| **Placental** | ▪ Abruptio placenta | |

# FETAL COMPLICATIONS

| IMMEDIATE | LATE |
| --- | --- |
| Prematurity<br>Fetal growth restrictioin<br>Iud<br>Intrapartum asphyxia<br>Hypoxic ischemic encephalopathy | Long term cardiovascular morbidity |

# THANK YOU

# REFERENCE


- Textbook of obstetrics –sheila balakrishnan 3rd edition