Disorders of placentation, preeclampsia & eclampsia

OBJECTIVES
At the end of the lecture students will be able to understand the disorders of late pregnancy including:

- Twin placentas
- Abnormalities of placental implantation
- Placental infections
- Preeclampsia
- Eclampsia

**Twin placentas**

- Monochorionic twin pregnancy -- twin-twin transfusion syndrome
- Vascular anastomoses, connecting the circulations of the twins
- Abnormal sharing of fetal circulations through an arteriovenous shunt
- Marked disparity in fetal blood volumes may result in twin-twin transfusion syndrome and the death of one or both fetuses

**Abnormalities of placental implantation**

- Placenta previa:
  - Placenta implants in the lower uterine segment or cervix
  - Serious third-trimester bleeding
  - A complete placenta previa covers the internal cervical os
  - Requires delivery via cesarean section
— placental rupture and fatal maternal hemorrhage during vaginal delivery

• Placenta accreta
  — partial or complete absence of the decidua
  — adherence of the placental villous tissue directly to the myometrium
  — failure of placental separation
  — Postpartum bleeding, often may be life-threatening to the mother

PLACENTAL INFECTIONS
• Two pathways:
  • ascending infection through the birth canal
  • hematogenous (transplacental) infection.

• Ascending infections are by far the most common
  • virtually always bacterial
  • localized infection of the membranes
  • premature rupture of membranes and preterm delivery.
  • The amniotic fluid may be cloudy with purulent exudate
  • “vasculitis” of umbilical and fetal chorionic plate vessels.
• Bacterial infections may arise by the hematogenous spread of bacteria directly to the placenta.
• The villi will then show acute inflammatory cells (acute villitis)

Placental infections derived from ascending and blood-borne routes
Acute chorioamnionitis.
On gross examination the placenta contains greenish opaque membranes.
B. A photomicrograph illustrates a dense bandlike inflammatory exudate on the amniotic surface (arrow). C, Acute necrotizing intervillitis, from a fetal-maternal infection. The chorion-amnion contains a polymorphonuclear leukocytic infiltrate. Edema and congestion of the vessels.

Hematogenous infections
- TORCH: Toxoplasmosis and others
- Syphilis,
- Tuberculosis,
- Listeriosis,
- Rubella,
- Cytomegalovirus,
- Herpes simplex
- They give rise to inflammatory infiltrates in the chorionic villi,
- chronic inflammatory cells (chronic villitis)
- Often, the cause of chronic villitis is obscure and may involve immunological phenomena

Pre-eclampsia:
Probably a disorder of placentation.
The physiologic alterations in the uterine spiral arteries and the failure of their remodeling in preeclampsia

Poor-placentation theory of pre-E:
Syncytiotrophoblast invades myometrium but does not denervate spiral arteries of mother properly.
Hence, intervillous flow is sub-optimal.
Chorionic villi are ischemic and release mediators (VEGF, etc) which damage maternal endothelium.
Pre-eclampsia: ischemic chorionic villi release pre-E mediators into maternal blood

PREECLAMPSIA AND ECLAMPSIA

A systemic syndrome characterized by widespread maternal endothelial dysfunction presenting
(1) It occurs in about 3% to 5% of pregnant women
(2) Last trimester
(3) More commonly in primiparas

Traditional pre-eclampsia triad:

(1) Hypertension
(2) Proteinuria
(3) Edema

New understanding of traditional pre-eclampsia triad:

(1) Hypertension $\rightarrow$ arteriolar constriction (endothelial dysfunction).
(2) Proteinuria $\rightarrow$ leaky glomerulus (capillary) (endothelial dysfunction).
(3) Edema $\rightarrow$ leaky capillaries in skin, muscle, liver, brain, airway, nose. (endothelial dysfunction).

“4th component” of endothelial dysfunction in pre-eclampsia
(1) Muscular artery spasm $\rightarrow$ increased arterial wave reflection back to heart
(2) Increased “augmentation index” (Alx)
(3) Increased Alx $\rightarrow$ extra work for heart muscle
(4) LVH, CHF.

Modern concept of pre-eclampsia: symptoms are due to arterial, arteriolar and capillary endothelial damage.

(1) Q: Damage by what?
Deranged smooth muscle function, due to damaged endothelium overlying smooth muscle.

Leaky capillary endothelium (no smooth muscle).

Endothelial factors in pre-E:

In health, there is a balance between vasodilatory factors: NO, PGI2 (Prostacyclin) and vasoconstrictive factors: thromboxane, endothelin.

This normal balance is messed up in pre-E.
Endothelial damage causes problems in 3 sizes of blood vessels:

(1) Muscular arteries → increased wave reflection (heart work, augmentation index).
(2) Arterioles → increased SVR (severe vascular resistance)
(3) Capillaries → proteinuria and tissue edema (glomerulus, liver, skin, muscle, brain)

Morphology
1. The placenta reveals various microscopic changes, most of which reflect malperfusion, ischemia, and vascular injury.
2. Placental infarcts—small, peripheral ones that may occur in normal full-term placentas—are larger and more numerous in preeclampsia. Ischemic changes in the chorionic villi and trophoblast. Increased syncytial knots and the appearance of accelerated villous maturity.
   There is increased frequency of retroplacental hematomas due to bleeding and instability of uteroplacental vessels.

(3) Finding is in the decidual vessels, reflecting abnormal implantation. This can be in the form of
   thrombosis
   fibrinoid necrosis
   intraintimal lipid deposition (acute atherosis)

   The liver lesions, when present, take the form of
   1. irregular,
   2. focal,
   3. subcapsular,
   4. intraparenchymal hemorrhages
   On histologic examination there are fibrin thrombi in the portal capillaries and foci of hemorrhagic necrosis.

1. The kidney lesions are variable.
2. Glomerular lesions are diffuse,
3. They consist of
4. Marked swelling of endothelial cells
5. Deposition of fibrinogen-derived amorphous dense deposits on the endothelial side of the basement membrane, and mesangial cell hyperplasia.
6. Abundance of fibrin in glomeruli.
7. Fibrin thrombi are present in the glomeruli and capillaries of the cortex.
8. May produce complete destruction of the cortex in the pattern referred to as bilateral renal cortical necrosis.
9. The brain may have gross or microscopic foci of hemorrhage along with small-vessel thromboses.
10. Similar changes are often found in the heart and the anterior pituitary.

Edema– imagine same process in liver and brain.

Thank You