Some clinically important diseases characterized by necrosis

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CHARACTERSTICS OF THE ACUTE PANCREATITIS FORMS

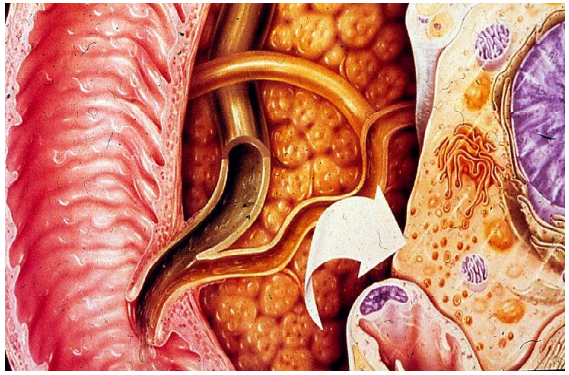
	Frequency	Lethality (%)
Interstitial (edematic)	80 - 90 %	0,3 %
Hemorrhagic (necrotizing)	10 - 15 %	50 - 90 %

ETIOLOGICAL FACTORS IN THE DEVELOPMENT OF ACUTE PANCREATITIS

40 - 50 %: cholelithiasis

30 - 40 %: alcoholism

10 - 30 %: idiopathic



<u>Other causes:</u>

Trauma (surgery!)

Hypercalcemia

Hyperlipoproteinemia

Vater-papilla obstruction

Infections (viruses)

Iatrogenic (ERCP, lithotripsy)

Familial

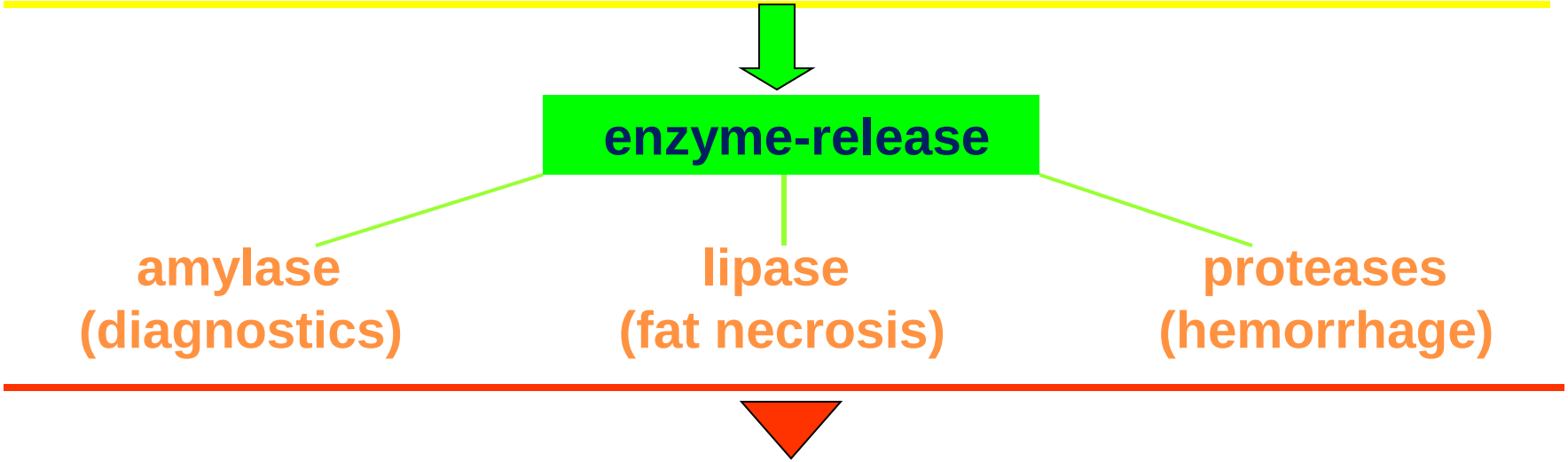
PATHOMECHANISM OF THE ACUTE PANCREATITIS

ACINAR DAMAGE

- alcohol
- viruses
- trauma
- hypercalcemia
- hyperlipidemia
- medicinal drugs

DUCTAL OBSTRUCTION

- cholelithiasis
- cystic fibrosis
- tumors
- Oddi-sphincter edema
- biliary reflux



ACUTE PANCREATITIS

POSSIBLE CONSEQUENCES OF THE ACUTE PANCREATITIS

Local:

- recovery
- fat necrosis
- pseudocyst

⬇

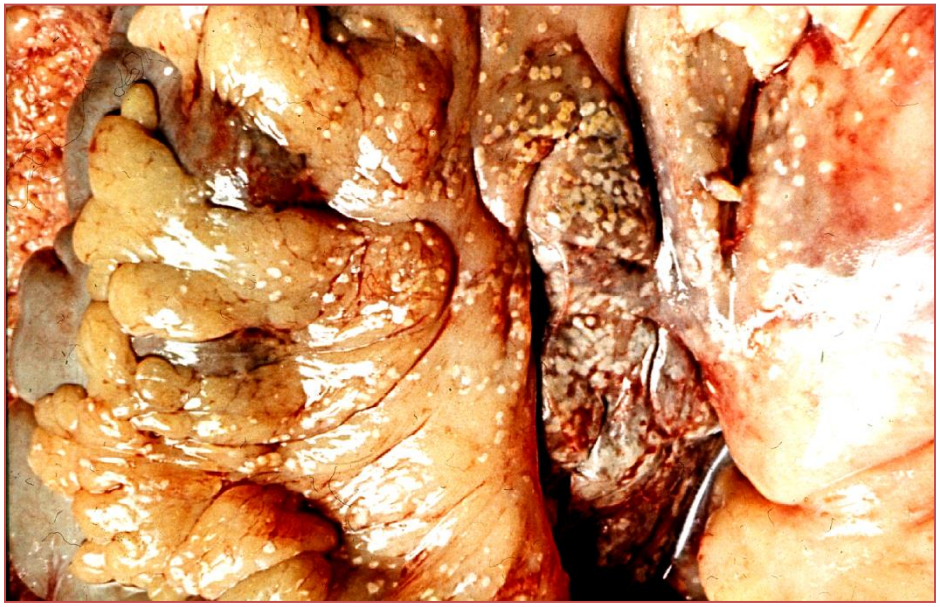
- abscess, ascites
- fistule
- pancreatic apoplexia

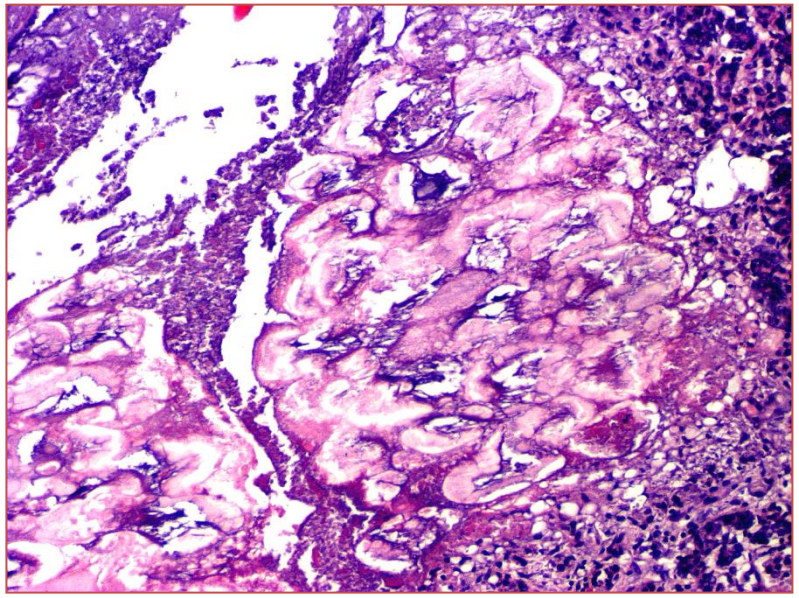
⬇

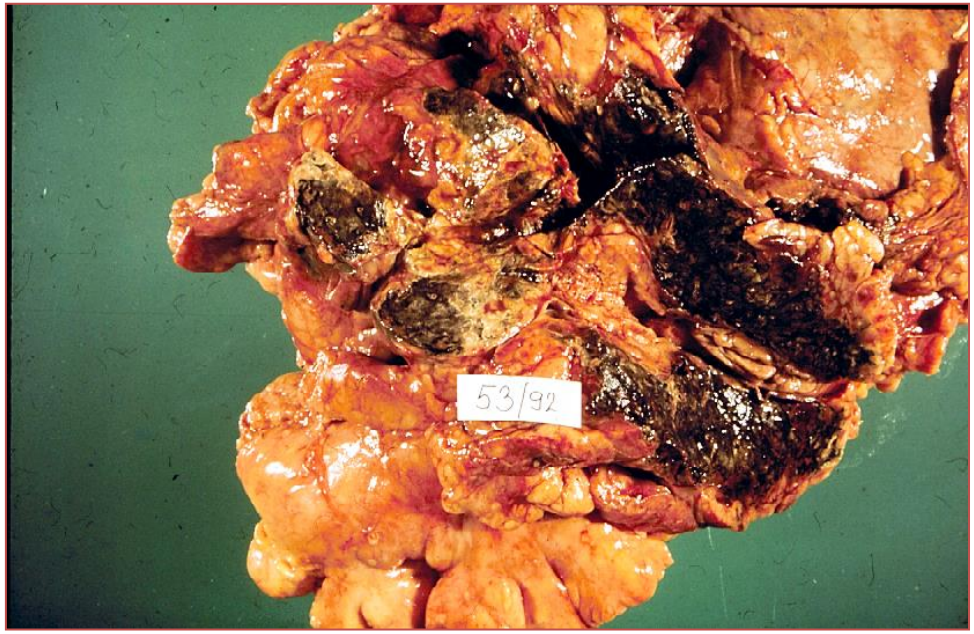
- retroperit. hemorrhage
- digestion of the surrounding organs

Systemic:

- paralytic ileus
- peritoneal (endotoxin) shock
- peritonitis
- acute renal insufficiency
- DIC
- ARDS, MOF
- diabetes mellitus



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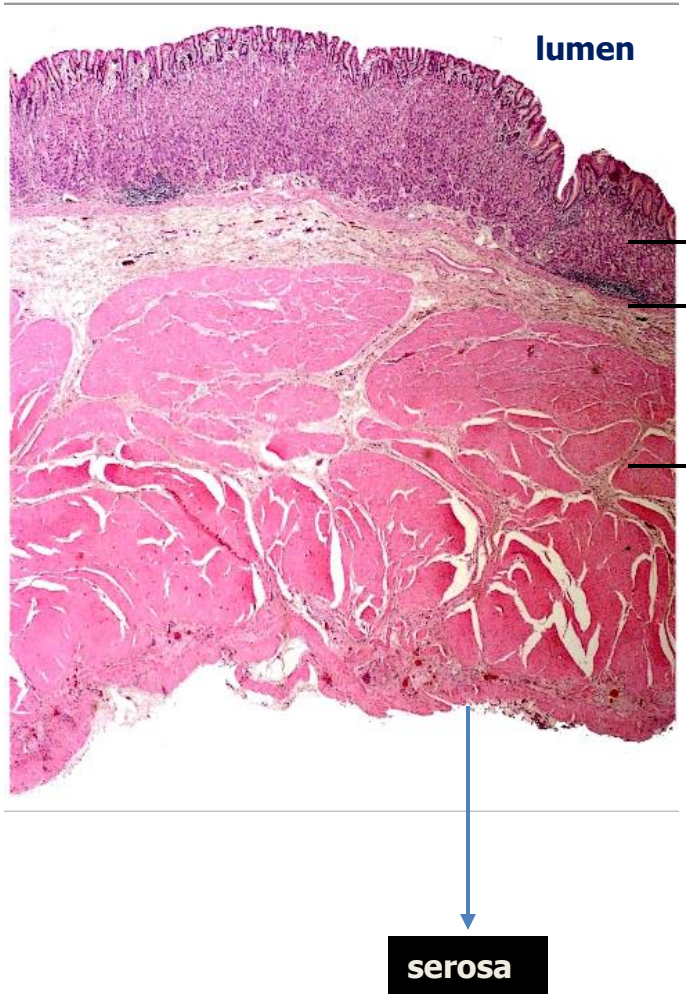
Erosion, acute ulcer, chronic ulcer

Stomach



Imbalance between aggressive and mucosal protective forces

Aggressive factors

HCl, pepsin

Gastrin

Histamine

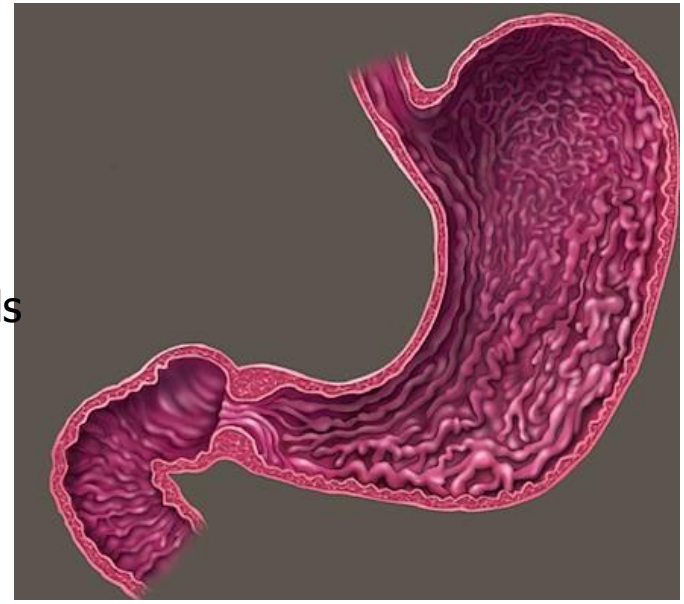
Glycocorticosteroids

Aspirin, NSAIDs

Helicobacter pylori

Alcohol

Smoking



Protective mechanizms

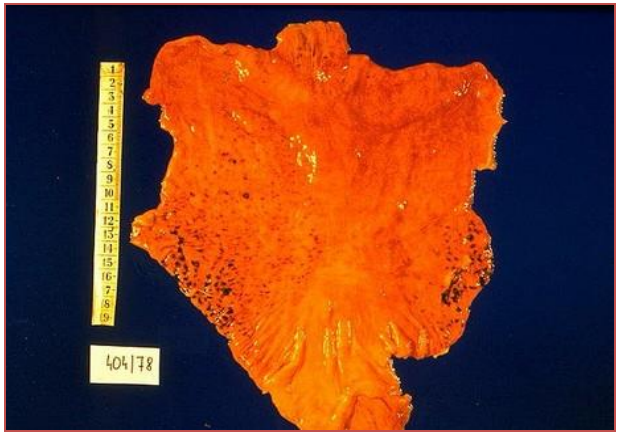
Surfcace mucus secretion

Mucosal blood supply

Bicarconate secretion

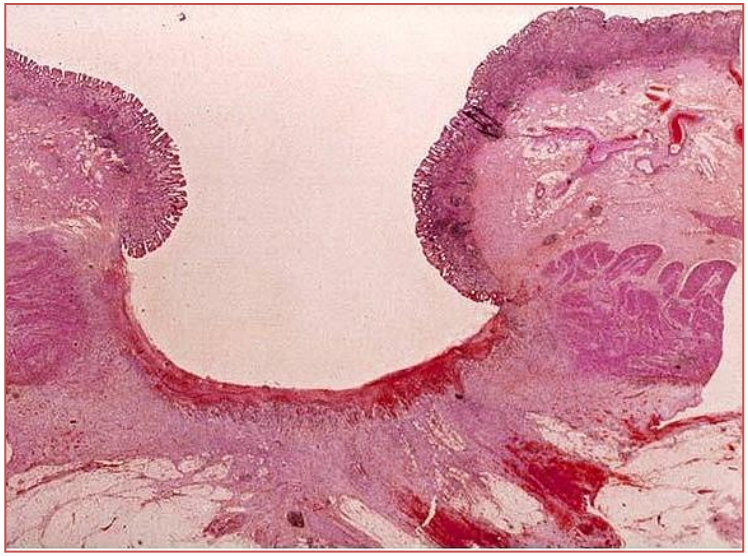
Prostaglandin-synthesis

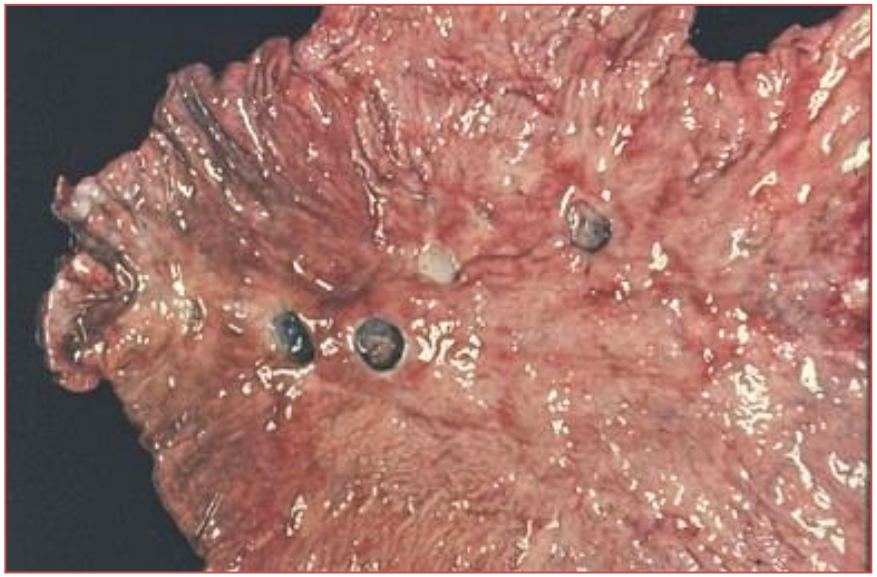
Good regenerative capacity

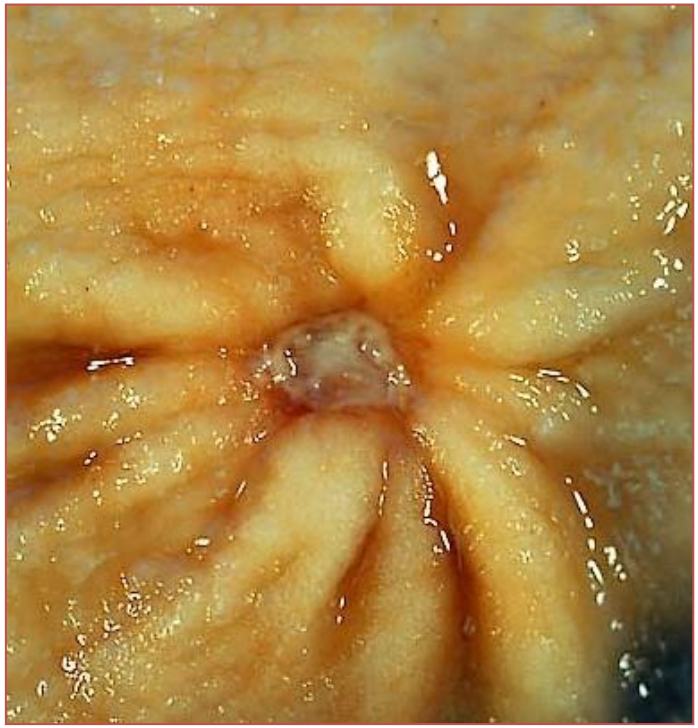


a.) stress-situations (shock, brain oedema, burns)
b.) Zollinger-Ellison syndromy
c.) Helicobacter pylori
d.) NSAID
e.) CMV (immunsuppressed patients)

danger: bleeding (hematemesis – melena)



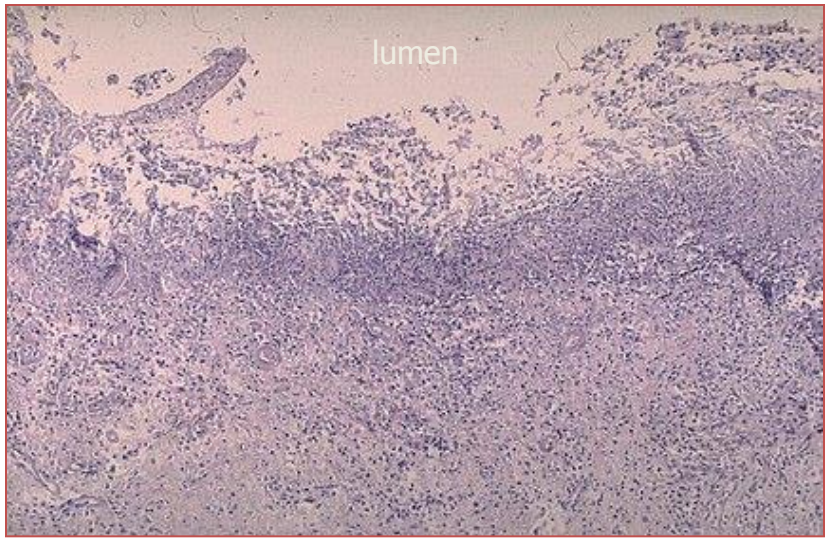




Chronic peptic ulcer

Complications:

1. **bleeding**
2. **perforation**
3. **penetration**
4. **stenosis**
5. **malignant transformation (carcinoma)**



epithelial slough, necrotic debris

fibrinoid necrosis

granulation tissue

scarring

Bisphosphonate-associated osteonecrosis of jaw (BONJ)

Bisphosphonates:

Bone-lysis inhibiting drugs (osteoclast-apoptosis induction + obliteration of capillaries)

Half-life: 1-10 years!

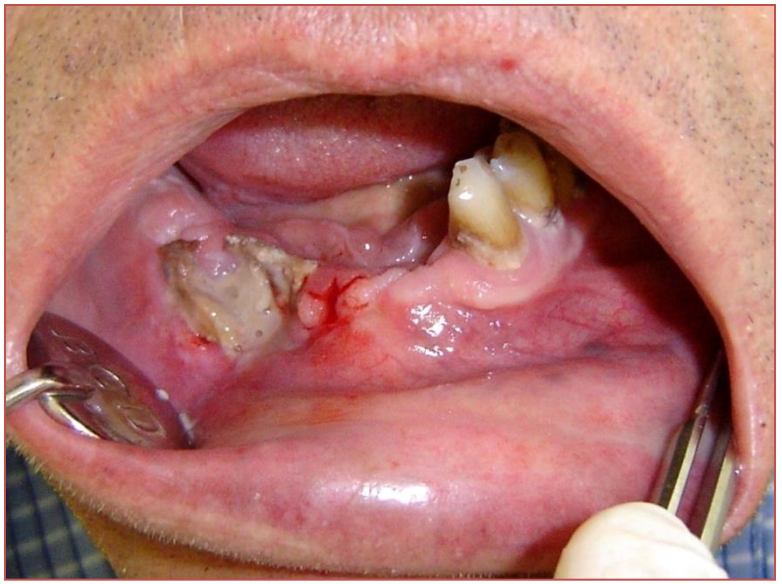
Hungary: 70 000 pts are treated (osteoporosis, lytic bone metastasis of malignant tumors /life-long!/

Jaw-necrosis: 1 –5 % (increaasing incidence)

Especially the jaw is affected – circumscribed aseptic bone necrosis

Following tooth extraction, dental procedures

Does not heal well!



(Dr. Redl Pál' s material)

ACUTE TUBULAR NECROSIS

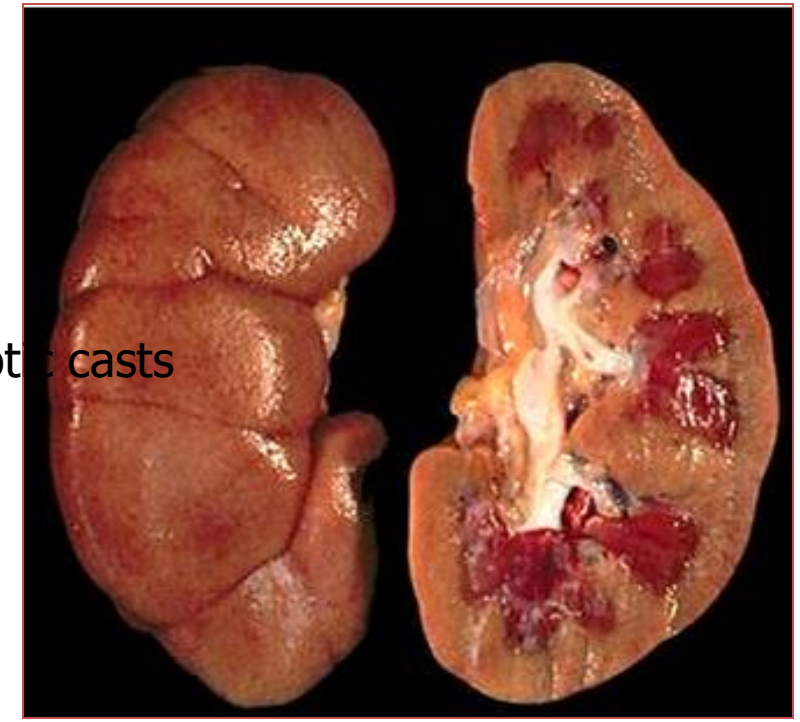
CAUSES:

a.) Ischemia (prolonged decrease of renal perfusion)

massive blood loss

prolonged hypotension / hypovolemic shock

b.) Toxic damage



COURSE:

- Necrosis of the tubular epithelium
- Blockage of the tubular lumens by necrotic casts
- Decreased GFR
- Acute renal insufficiency, anuria
- Regeneration

RENAL CORTICAL NECROSIS

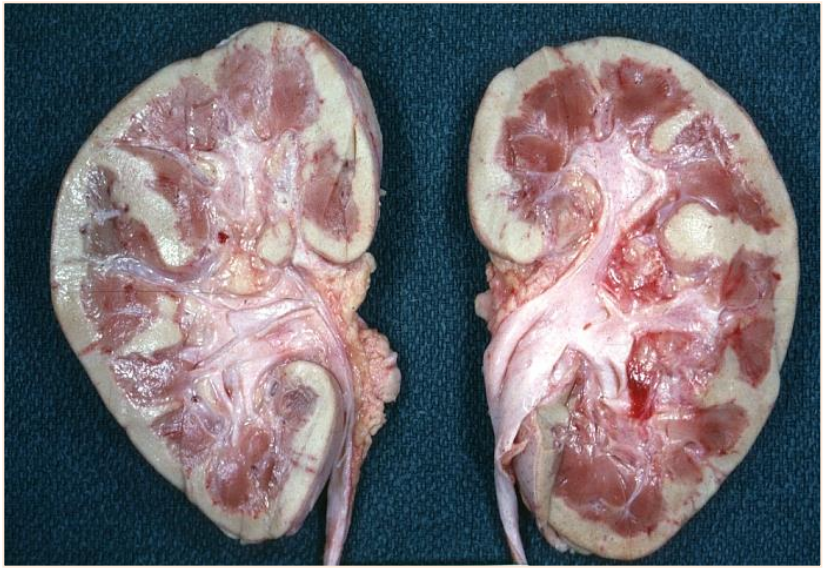
Bilateral necrosis of all cortical structures

Initiating factor: intense vasospasm

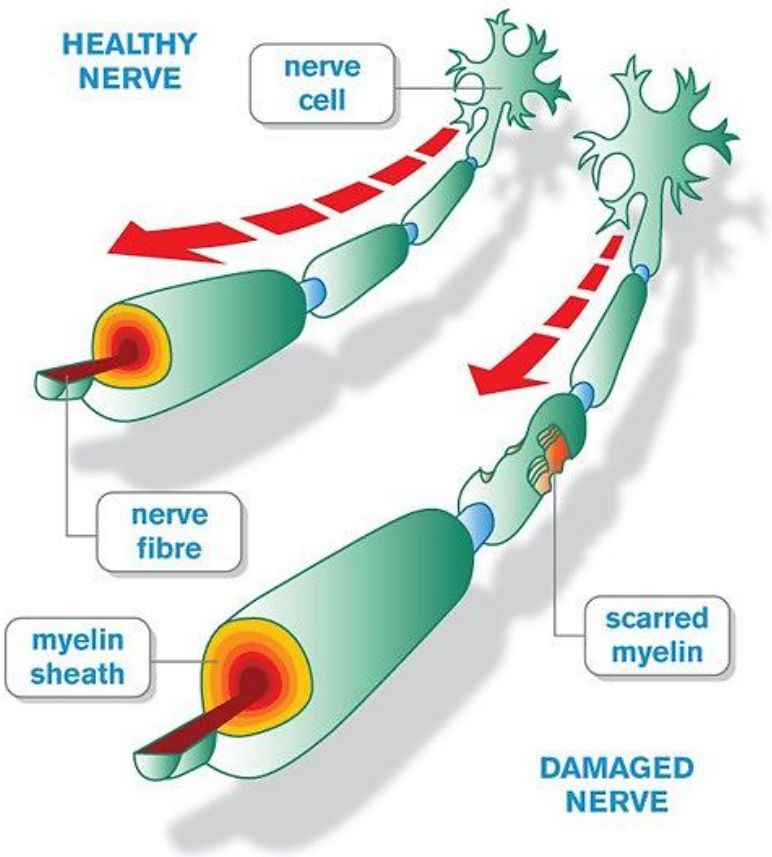
Etiology:

- pregnancy related causes (50%)
  - placental abruption
  - infected abortion
  - prolonged intrauterine fetal death
  - eclampsia
- sepsis
- shock
- poisonings

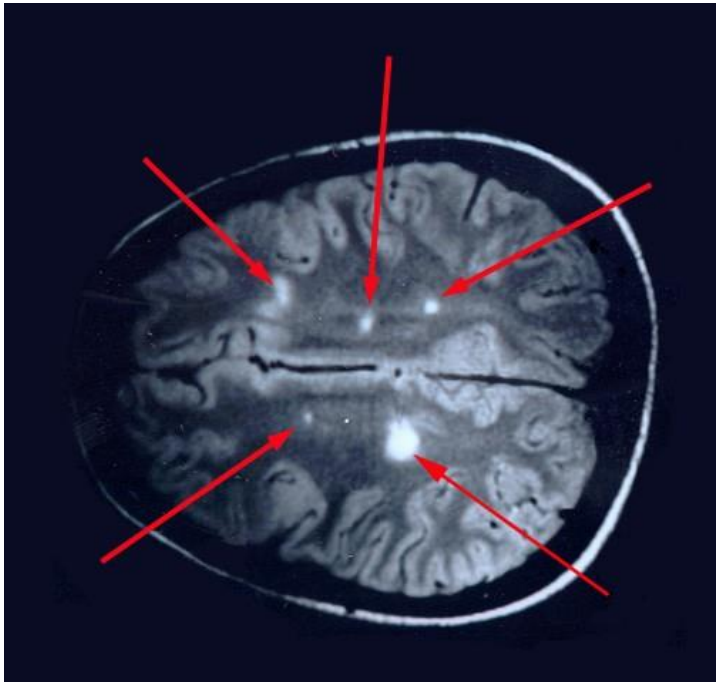
ACUTE RENAL FAILURE



DEMYELINATING DISEASES



Multiple sclerosis



plaques