

Cell Injury

p. patho

Slide 1: Reversible and Irreversible Cell Injury

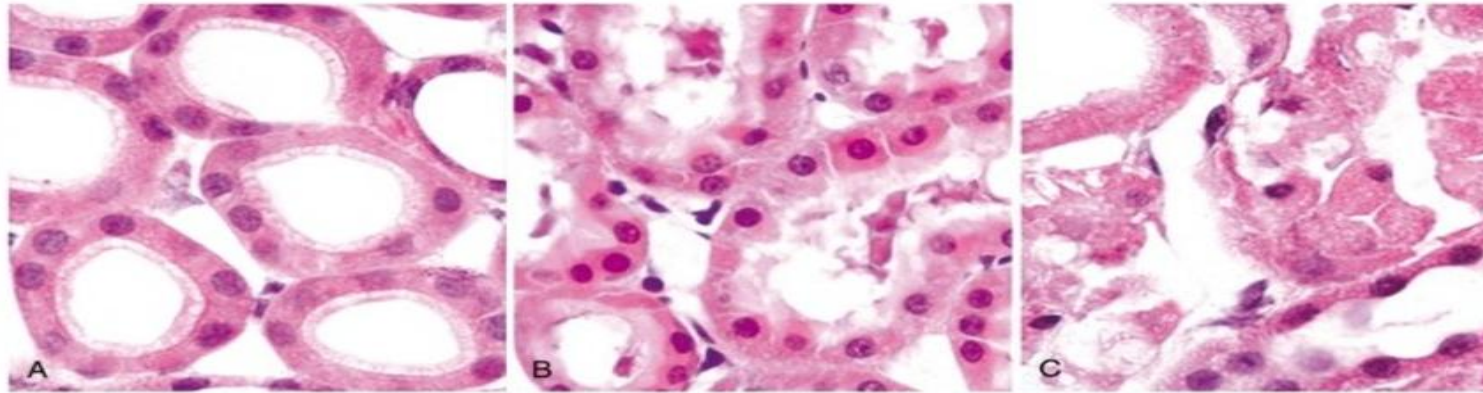


Fig. 2.8 Morphologic changes in reversible cell injury and necrosis. (A) Normal, healthy kidney tubules with cuboidal epithelial cells. (B) Early (reversible) ischemic injury: swollen surface blebs, increased eosinophilia of

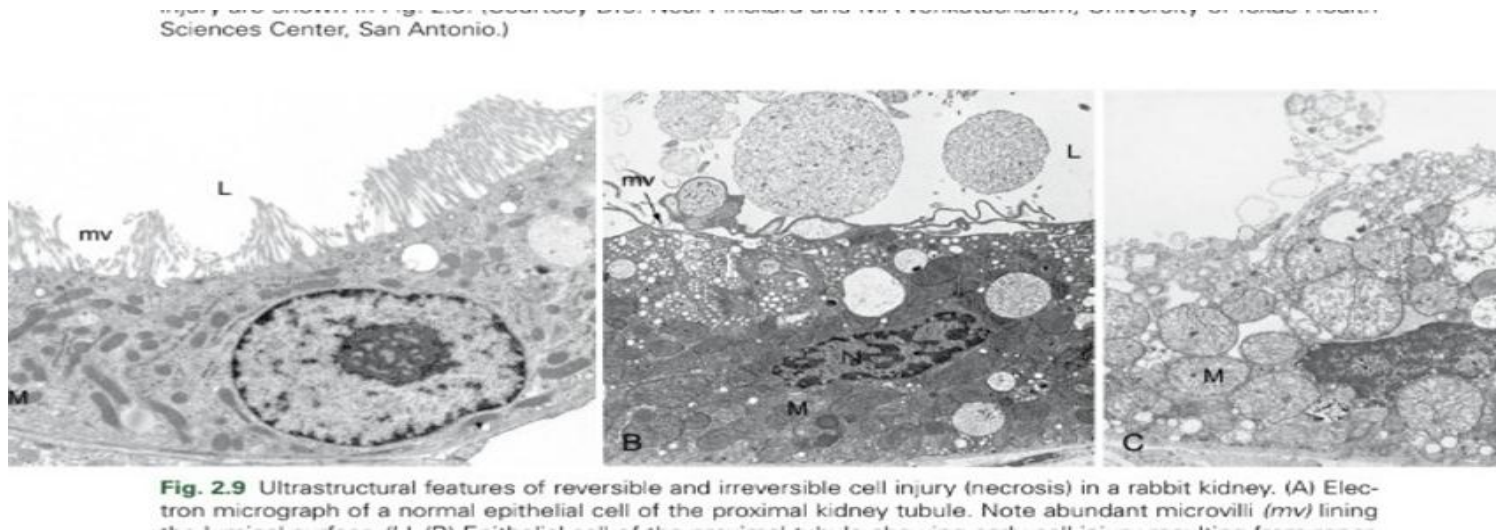
.Normal tubules: intact epithelial cells, nuclei, and cytoplasm.

.Reversible injury: cell swelling, increased eosinophilia, and occasional surface blebs.

.Irreversible injury: loss of nuclei, membrane damage, and leakage of cellular contents.

..Key point: Reversible injury may recover; irreversible injury progresses to cell death.

.Slide 2: Ultrastructural Changes in Cell Injury



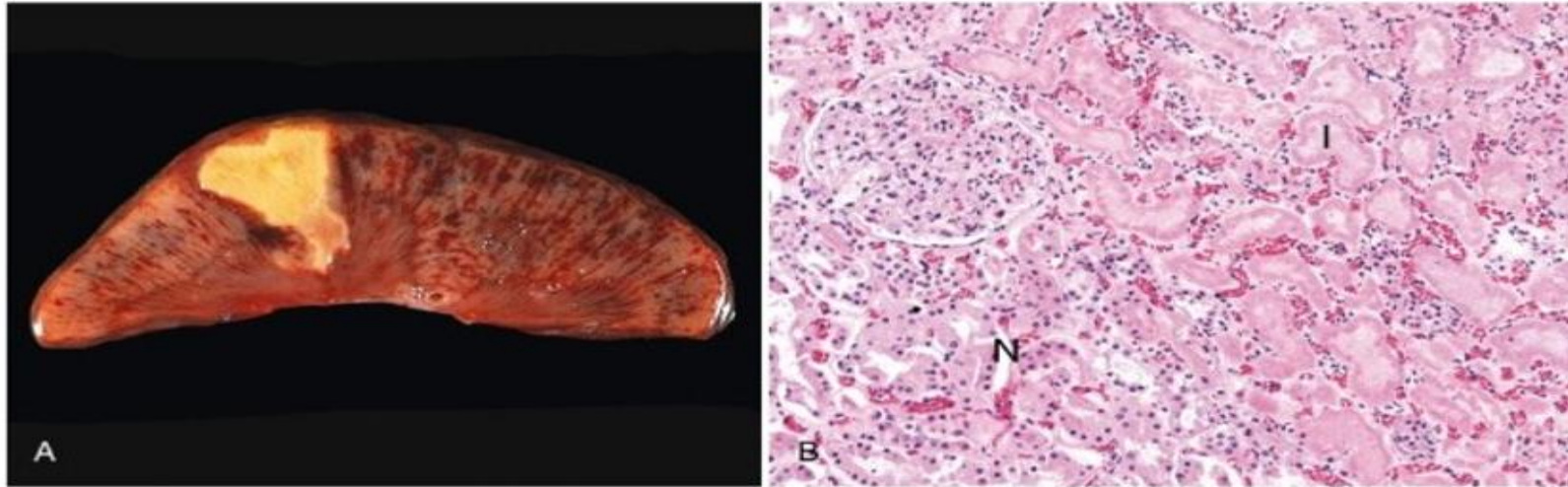
.**Normal cell:** intact microvilli, mitochondria, and cell membrane.

.**Reversible injury:** loss of microvilli and swelling of cells and mitochondria.

.**Irreversible injury:** severe mitochondrial damage, membrane disruption, and dense deposits.

.**Key point:** Severe membrane and mitochondrial damage suggests irreversible injury.

- Slide 3: Coagulative Necrosis



.Tissue architecture is preserved for a time.

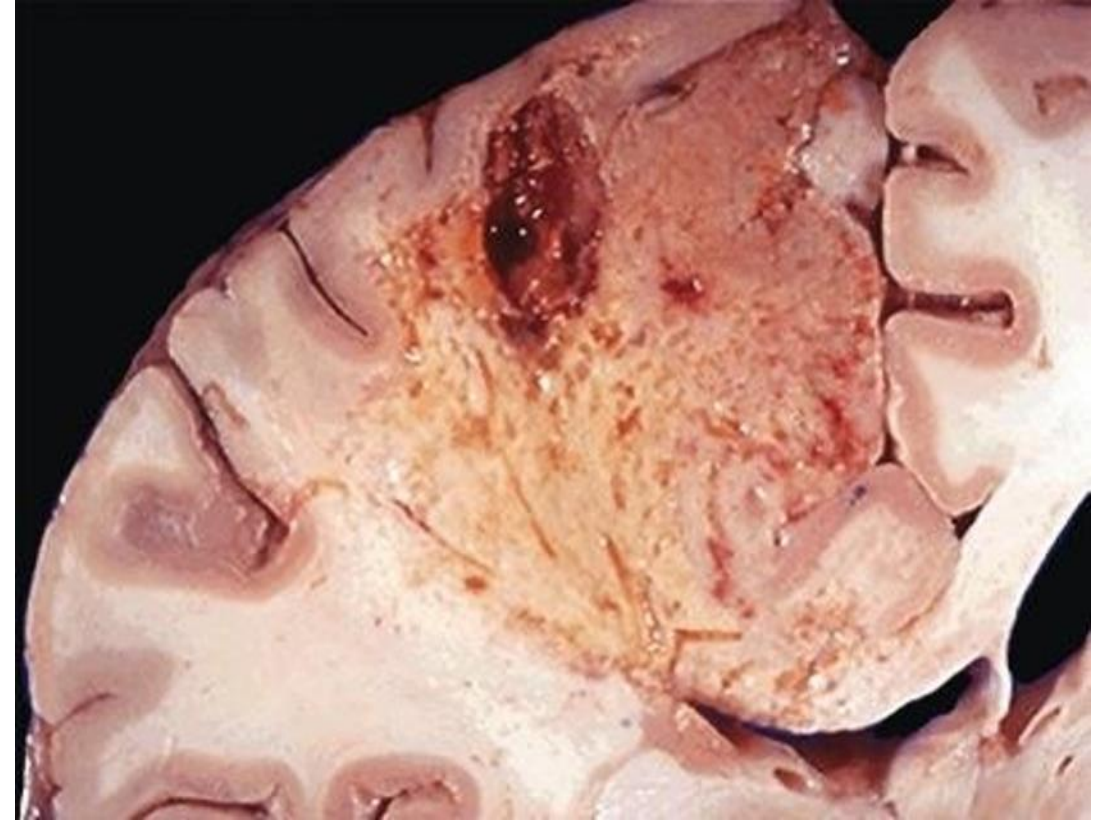
.Nuclei disappear and cytoplasm becomes more eosinophilic.

.Inflammatory cells may appear at the edge of the necrotic area.

Key point: Example: kidney infarction.

- Slide 4: Liquefactive Necrosis

- .Enzymes digest the dead tissue.
- .Normal tissue architecture is lost.
- .The affected area becomes soft or liquid.
- .**Key point:** Examples: brain infarction and bacterial abscesses



- Slide 5: Caseous Necrosis

..Often has a white, cheese-like gross appearance.

Normal tissue architecture is lost.

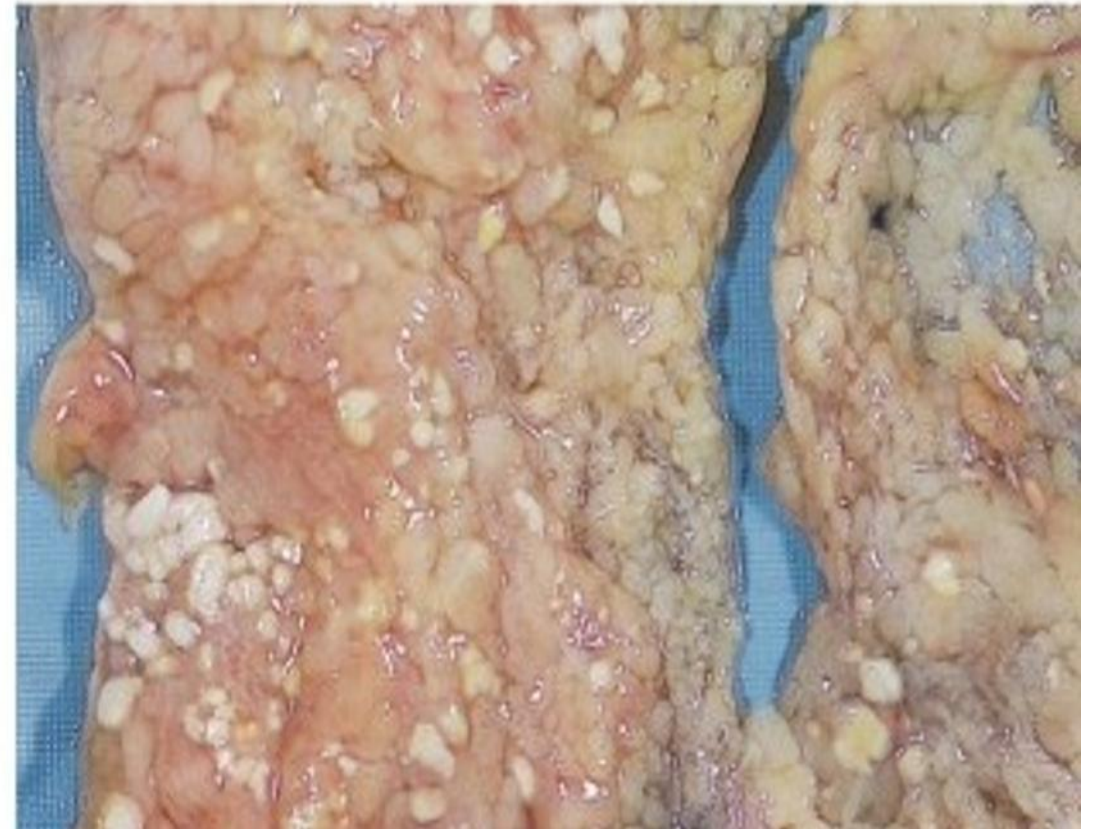
.Microscopy shows granular necrotic debris, often surrounded by granulomatous inflammation.

Key point: Example: tuberculosis.



s in the infarct (//) showing preserved cellular outlines
as nuclei of inflammatory cells in between necrotic

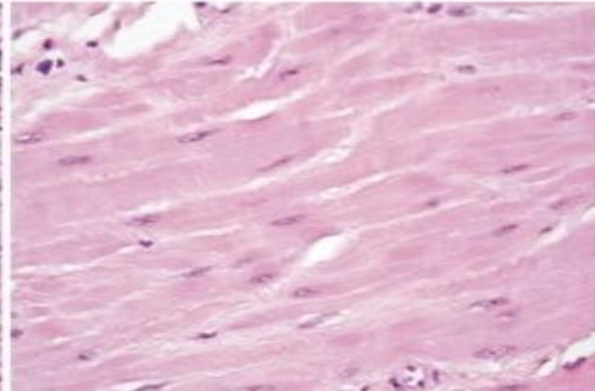
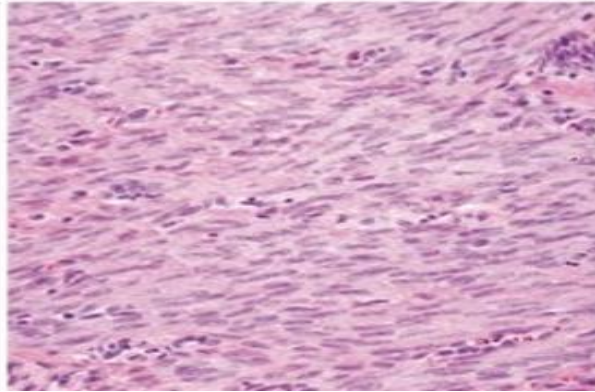
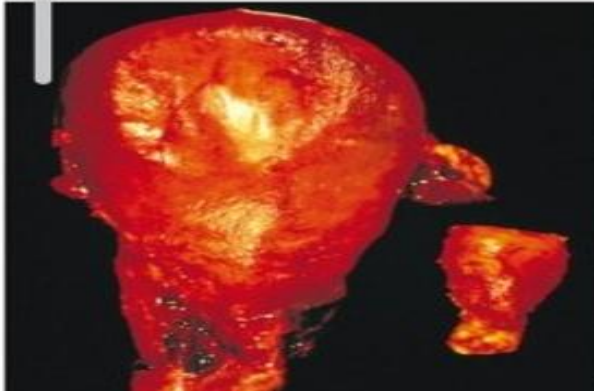
- **slide 6; fat necrosis**
- Fat cells are destroyed.
- Chalky white deposits may form.
- Calcium combines with fatty acids to form soaps (saponification).
- **Key point:** Example: acute pancreatitis.



- Slide 7: Hypertrophy

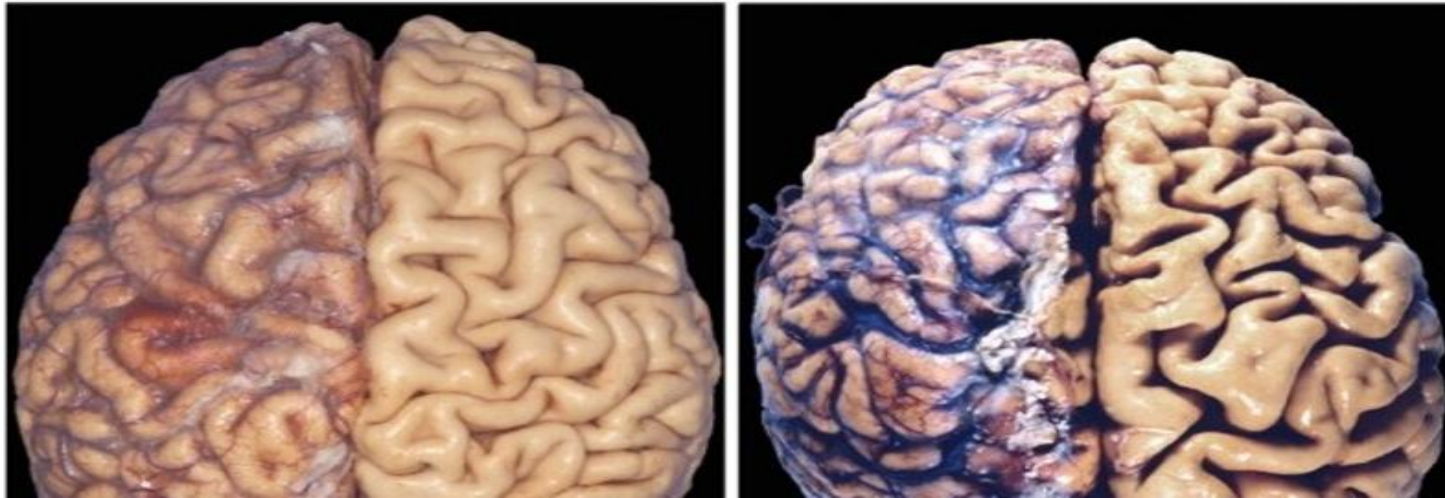
the synthesis and assembly of additional intracellular structural components. Cells capable of division may respond to stress by undergoing

dial hypertrophy because beyond a certain point it becomes maladaptive. Hypertrophy results from the action of growth factors and direct



- .Increase in cell size.
- .The organ becomes larger.
- .There is no increase in cell number.
- .**Key point:** Example: uterine smooth-muscle hypertrophy during pregnancy.

- Slide 8: Atrophy



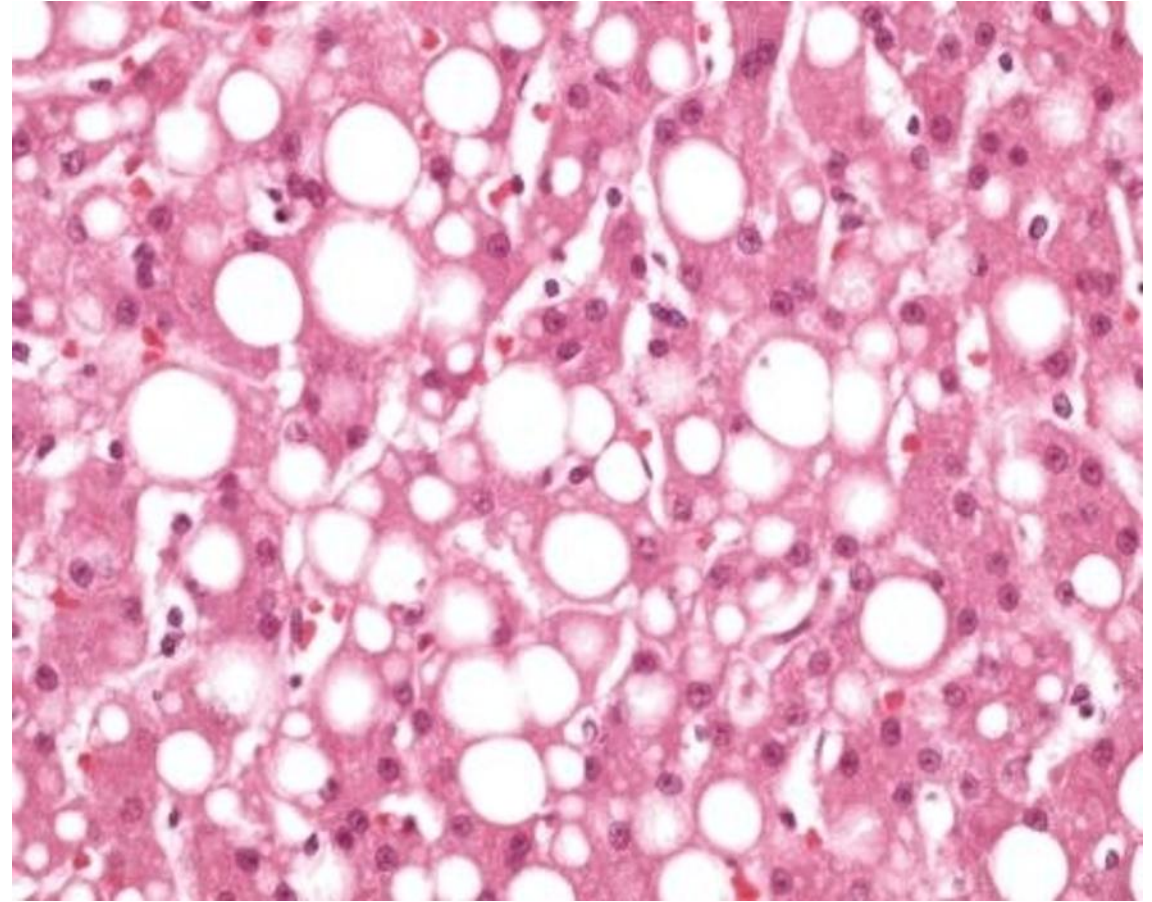
.Cells become smaller and tissue mass decreases.

.Cellular activity and metabolic demand are reduced.

.Key point: Example: brain atrophy associated with aging or reduced blood supply.

Slide 9; fatty change

- .Clear lipid vacuoles appear in the cytoplasm.
- .The nucleus may be pushed toward the cell edge.
- .Commonly affects hepatocytes.
- .Key point:** Example: fatty liver (hepatic steatosis)



- Slide 12: Protein Accumulation

.Pink eosinophilic droplets are visible in renal tubular epithelial cells.

.They represent reabsorbed proteins.

.Key point: May be associated with proteinuria

