

GENERAL PATHOLOGY

L-10 IRREVERSIBLE INJURY

CELL DEATH / NECROSIS

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1

NECROSIS / ONCOSIS / ONCOTIC NECROSIS

- ❑ Death of cells in Living Organisms
- ❑ Morphological Changes – **6-8 hours** after
- ❑ **Autolysis** – self digestion
- ❑ **Heterolysis** – Digestion by neighboring cells
- ❑ Cell death by ischemia is more severe than hypoxia alone

2

NECROSIS

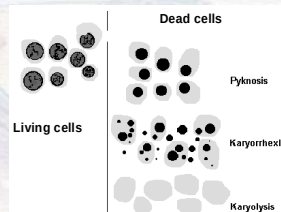
MICROSCOPIC CHANGES

❑ Cytoplasm

- Eosinophilic -- arginine and lysine
- Hyalinized Cytoplasm (glycogenolysis)
- Granular (Eosinophilia of Organelles)
- Moth-eaten (vacuolated)

❑ Nucleus

- Pyknosis
 - ❑ More Dark (nucleic acid set free)
 - ❑ Lack nucleolus
- Karyorrhexis
- Karyolysis (endonucleases/DNAse)



3

MICROSCOPIC CHANGES

- ❑ Loss of cell outline
- ❑ Loss of differential staining
- ❑ Absence of cells

ULTRASTRUCTURAL CHANGES

- ❑ discontinuities in plasma and organelle membranes
- ❑ marked dilatation of mitochondria with the appearance of large amorphous densities
- ❑ Intra-cytoplasmic
 - myelin figures
 - amorphous debris
 - aggregates of fluffy material probably representing denatured proteins

4

GROSS CHANGES

- Lighter colour (coagulation of proteins, reduction of blood supply)
- May be swollen area or depressed
- May be softer to touch
- Loss of Strength
- Red Zone of Congestion (2-3 days old)
- Coagulated, Creamy liquid or Dry crumbly

5

Types of Necrosis

□ Necrosis (irreversible)

- Coagulative
- Liquifactive
- Caseative

- Fat Necrosis
- Zenker's Necrosis
- Fibrinoid Necrosis
- Gangrene

6

Types of necrosis

■ Coagulative necrosis

- Original cell Shape and tissue architecture is protected
- Denaturation of proteins but incomplete digestion
- Cytoplasm is eosinophilic
- Outline of cells can be seen, as there is a loss of nuclei
- Cause can be sudden and severe ischemia in most tissues
- Often occurs in heart, kidney, adrenal tissue
- Over time macrophages will phagocytose the dead tissue and the area will be repaired.

7

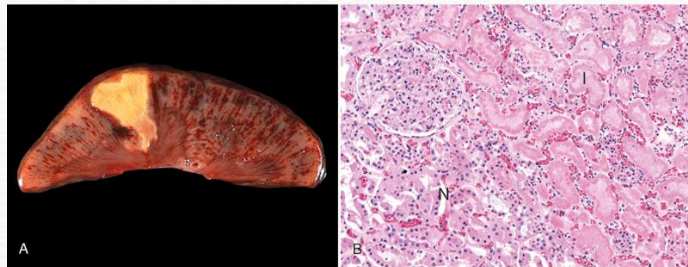
Coagulative necrosis

- Protein denaturation causes albumin to become firm.

CAUSES

- Hypoxia
- Chemicals (Mercuric chloride)
- Necrobacillosis (bacterial toxins)
- Burn injury

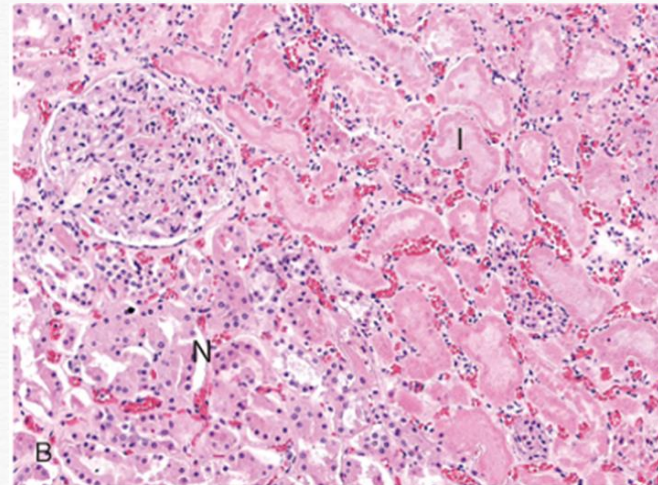
8



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A, A wedge-shaped kidney infarct (yellow) with preservation of the outlines.
B, Microscopic view of the edge of the infarct, with normal kidney (N) and necrotic cells in the infarct (I)

9



10

▪ Caseative necrosis

- ❑ necrotic tissue with appearance of cottage cheese; soft, fluffy, grey-white cheesy appearance (gross);
- ❑ Loss of tissue architecture and cellular details
- ❑ form of coagulative necrosis with limited liquefaction
- ❑ Amorphous granular debris surrounded by granulomatous inflammation; seen in TB

11

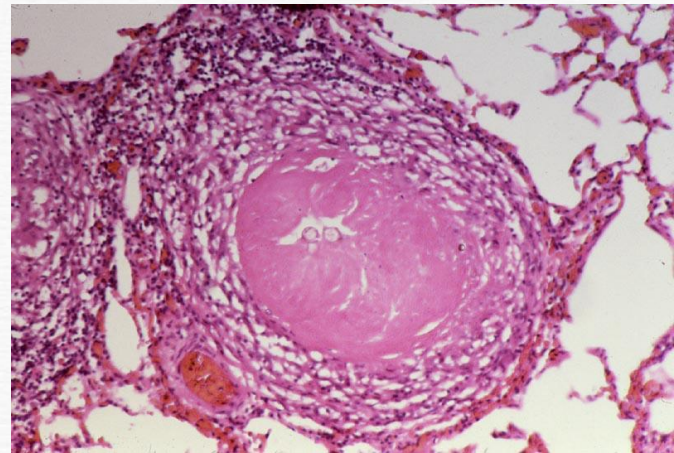
▪ Caseative necrosis

- ❑ Hydrolases do not completely digest tissue, looks like clumped cheese (caseous)
- ❑ Occurs in lung
- ❑ Cause- often infection with *Mycobacterium tuberculosis*

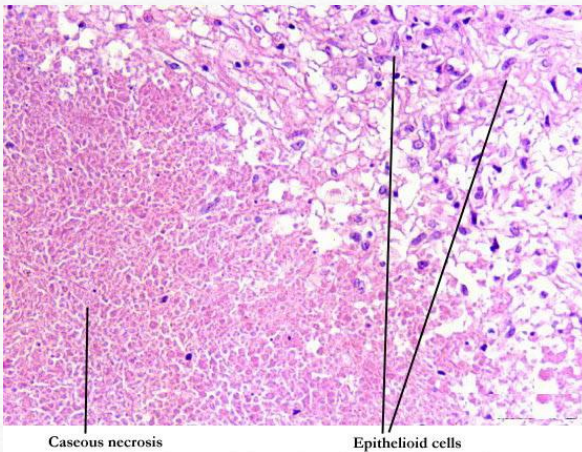
12



13



14



Caseous necrosis

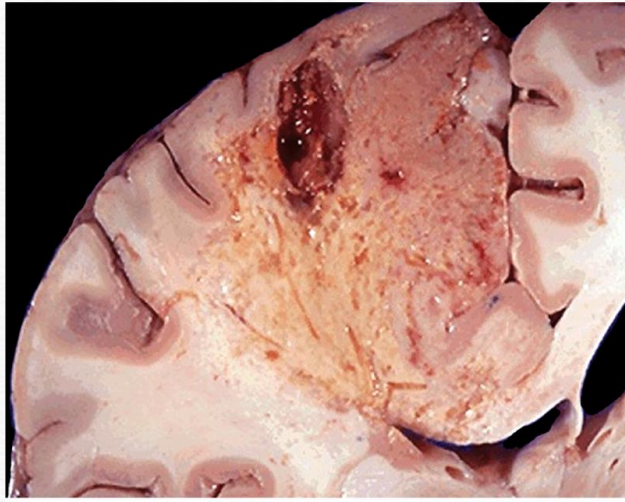
Epithelioid cells

15

▪ Liquefactive necrosis

- no cell architecture remains; **dissolution**, **liquid viscous mass**
- **Hydrolases** cause tissue to become soft, often cyst form
- prominent **neutrophilic** infiltrate
- common in bacterial lesions
- results from enzymatic degradation of tissue; (Autolysis + WBC)
- brain is classic example even in ischaemia
- necrotic area is soft and filled with fluid (brain infarct)
- Tissue or area is very soft, even liquid (abscess)
- Causes - bacterial infections such as staphylococci, streptococci and *E. coli*

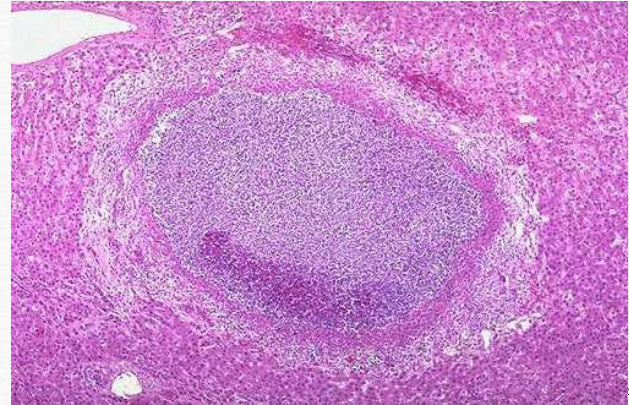
16



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17

Liver abscess: Liquifactive necrosis



18

QUESTIONS
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19