SAWA UNIVERSITY

PATHOLOGY 1ST.COURSE

NORMAL CELL&NECROSIS

Sawa University — جامعـــــة ســـــــاوة الاهلية

College of health and medical techniques — كليـــــة التقنيـــات الصحيـة والطبيـة

Department of Radiology Tech. — قسم تقنيات الاشعة والسونار

third Stage — المرحلـــــــــــة : الثالثه

محاضرة رقم 1

الجانب النظري

Lecture No1.

Theoretical

تدريسي المادة : المحاضر م .م فاضل عوفي عواد

Normal vs Necrosis (Cell Death)

General Pathology — Slide Examination

Normal Cell Morphology

- Intact plasma membrane
- Clear nuclear envelope, nucleolus visible
- Even chromatin distribution
- Cytoplasm: normal staining, no vacuoles
- Preserved tissue architecture

Necrotic Cell Morphology

- Loss of plasma membrane integrity
- Eosinophilic (pink) cytoplasm
- Organelle swelling & disruption
- Nuclear changes (pyknosis, karyorrhexis, karyolysis)
- Inflammatory response present

Nuclear Changes in Necrosis

- Pyknosis — chromatin condensation, shrunken nucleus
- Karyorrhexis — nuclear fragmentation
- Karyolysis — dissolution of chromatin, faded nucleus

Cytoplasmic & Structural Changes

- Increased eosinophilia
- Vacuolization, loss of fine structure
- Loss of tissue architecture (except in coagulative necrosis)
- Membrane rupture & leakage of contents

Inflammation & Tissue Response

- Leakage of intracellular contents triggers inflammation
- Neutrophils and macrophages infiltrate
- Cellular debris and edema present
- Fibrosis or calcification may follow

Normal vs Necrosis — Key Differences

Normal Cells:

- Intact membranes, nuclei, cytoplasm
- Preserved tissue architecture
- No inflammation

Necrotic Cells:

- Membrane rupture, eosinophilic cytoplasm
- Nuclear pyknosis, karyorrhexis, karyolysis
- Loss of architecture, inflammation present

Summary

- Normal cells show preserved structure and no inflammation
- Necrotic cells show membrane rupture, cytoplasmic & nuclear changes
- Necrosis always elicits inflammation
- Recognition of features is key in pathology slide examination

Here’s a comparison (with slide / microscopic features) between normal (viable) tissue / cells and necrosis / pathological cell death.

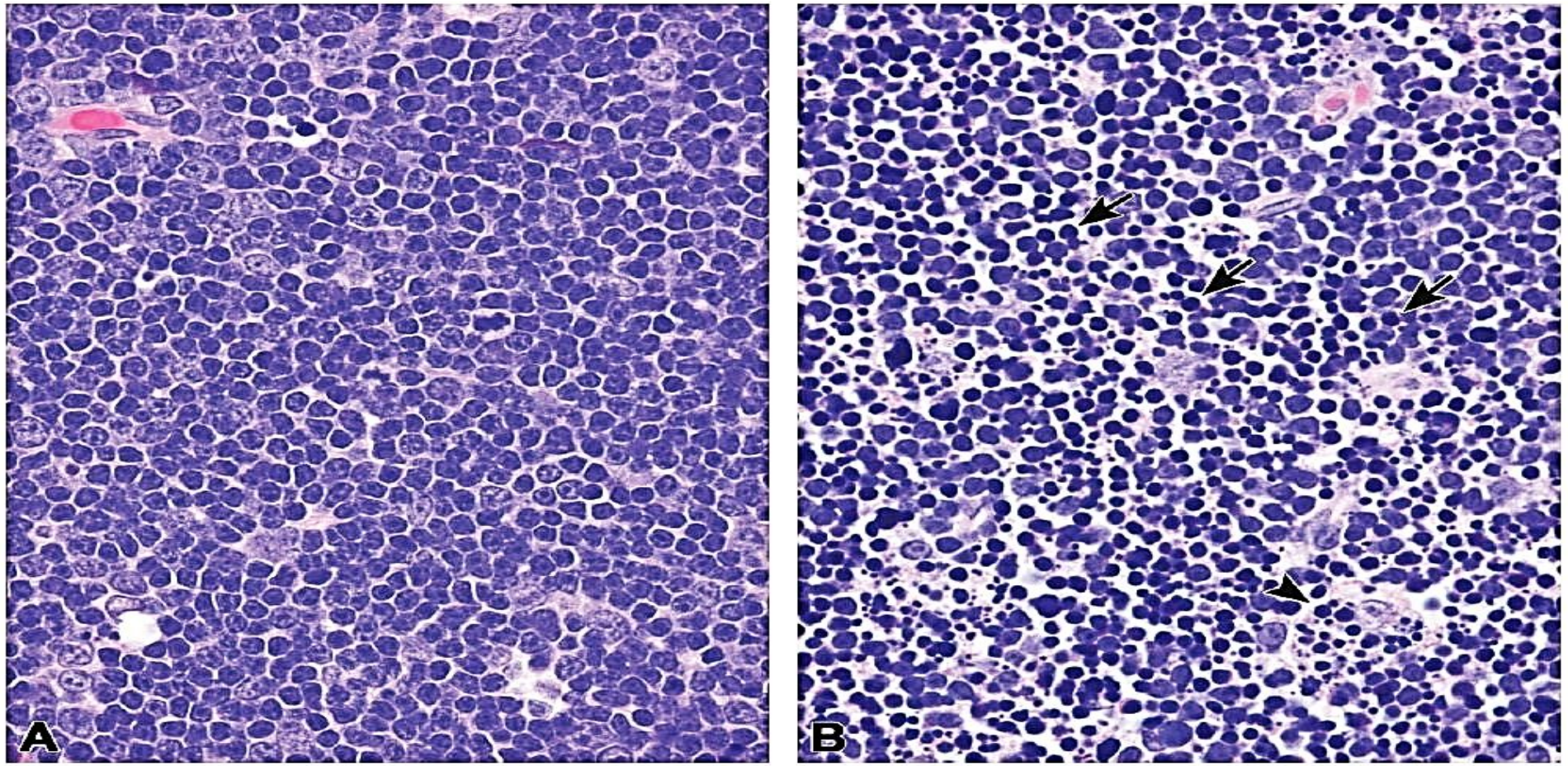
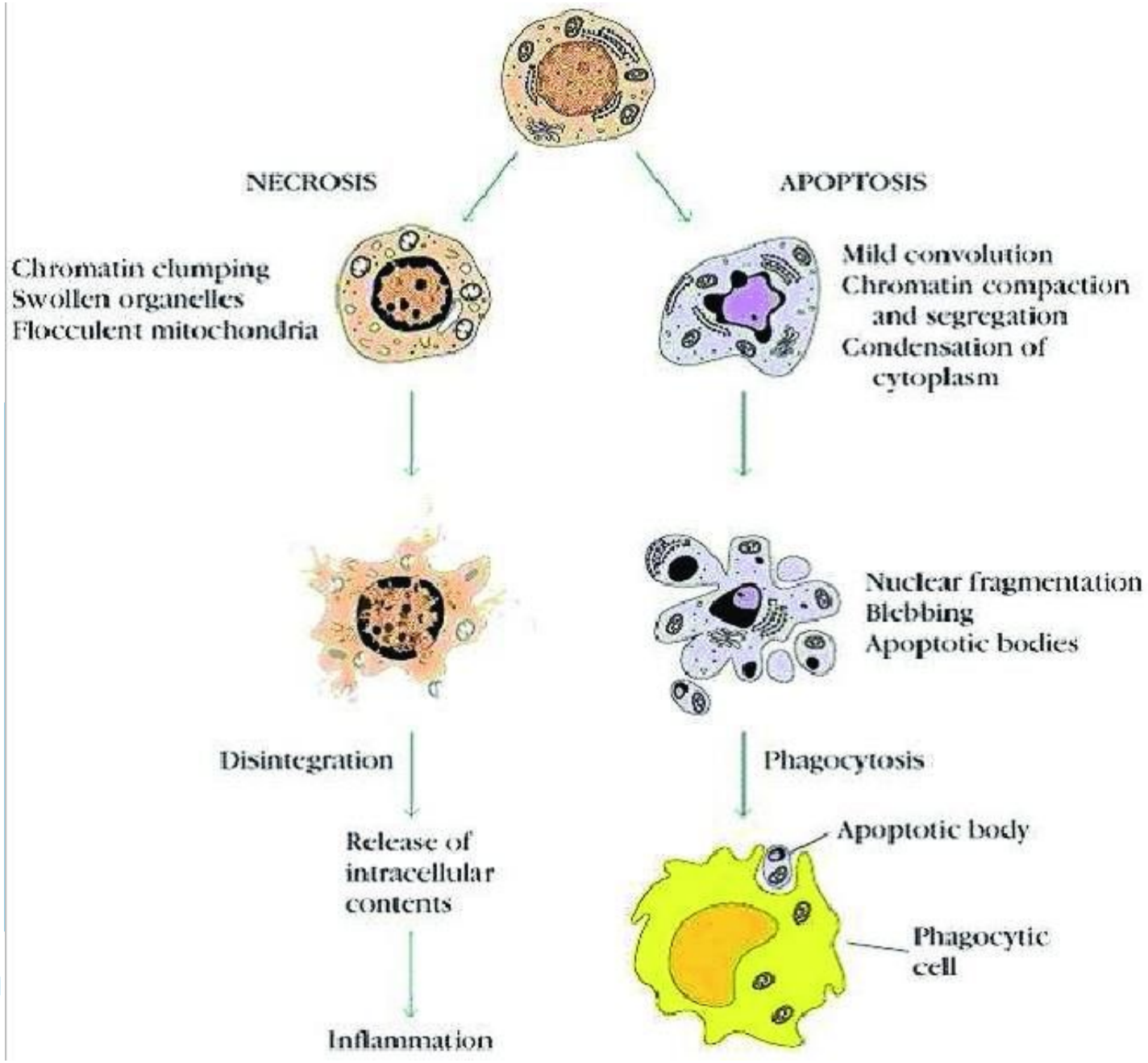
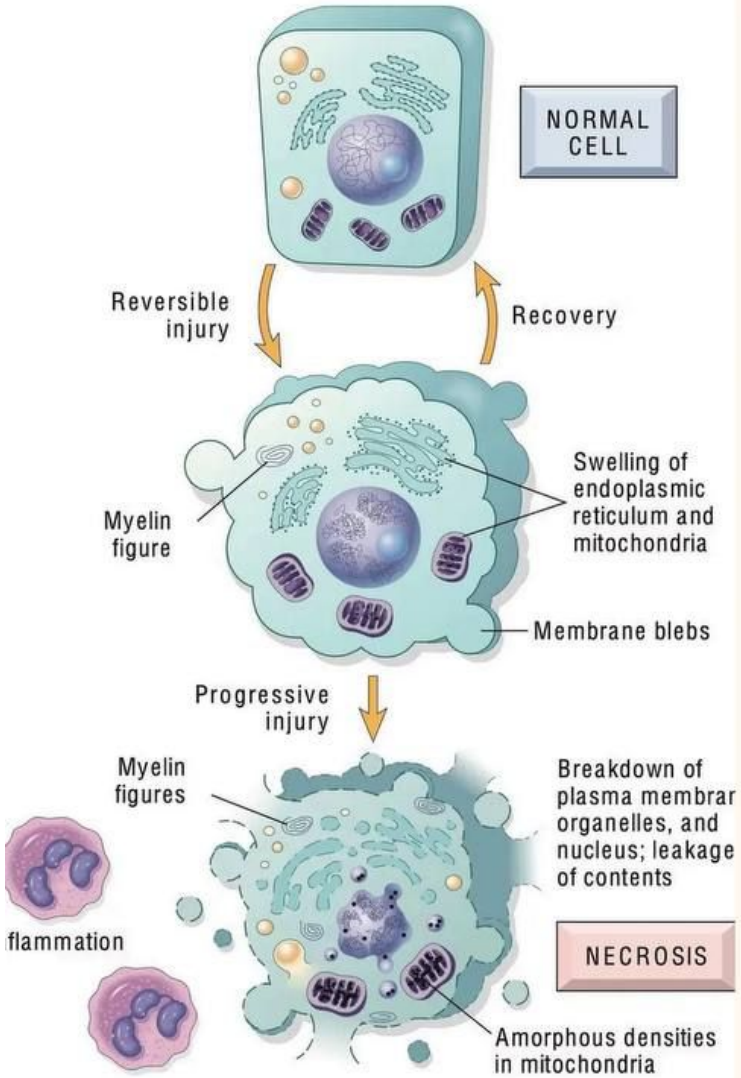


Figure 10. Experimental induction of apoptosis in the thymus cortex of a male 3-month-old Sprague-Dawley rat dosed with 1 mg/kg dexamethasone and necropsied 12 hr later (B) and compared to the thymus cortex from a concurrent control rat (A). The majority of cortical thymocytes in the treated rat (B) have undergone apoptosis. Most are in late stages of apoptosis with small, rounded hyperchromatic apoptotic bodies (arrows). Some have been engulfed by tingible body macrophages (arrowhead).


NECROSIS
APOPTOSIS
Chromatin clumping
Swollen organelles
Flocculent mitochondria
Mild convolution
Chromatin compaction
and segregation
Condensation of
cytoplasm
Nuclear fragmentation
Blebbing
Apoptotic bodies
Disintegration
Phagocytosis
Release of
intracellular
contents
Apoptotic body
Phagocytic
cell
Inflammation

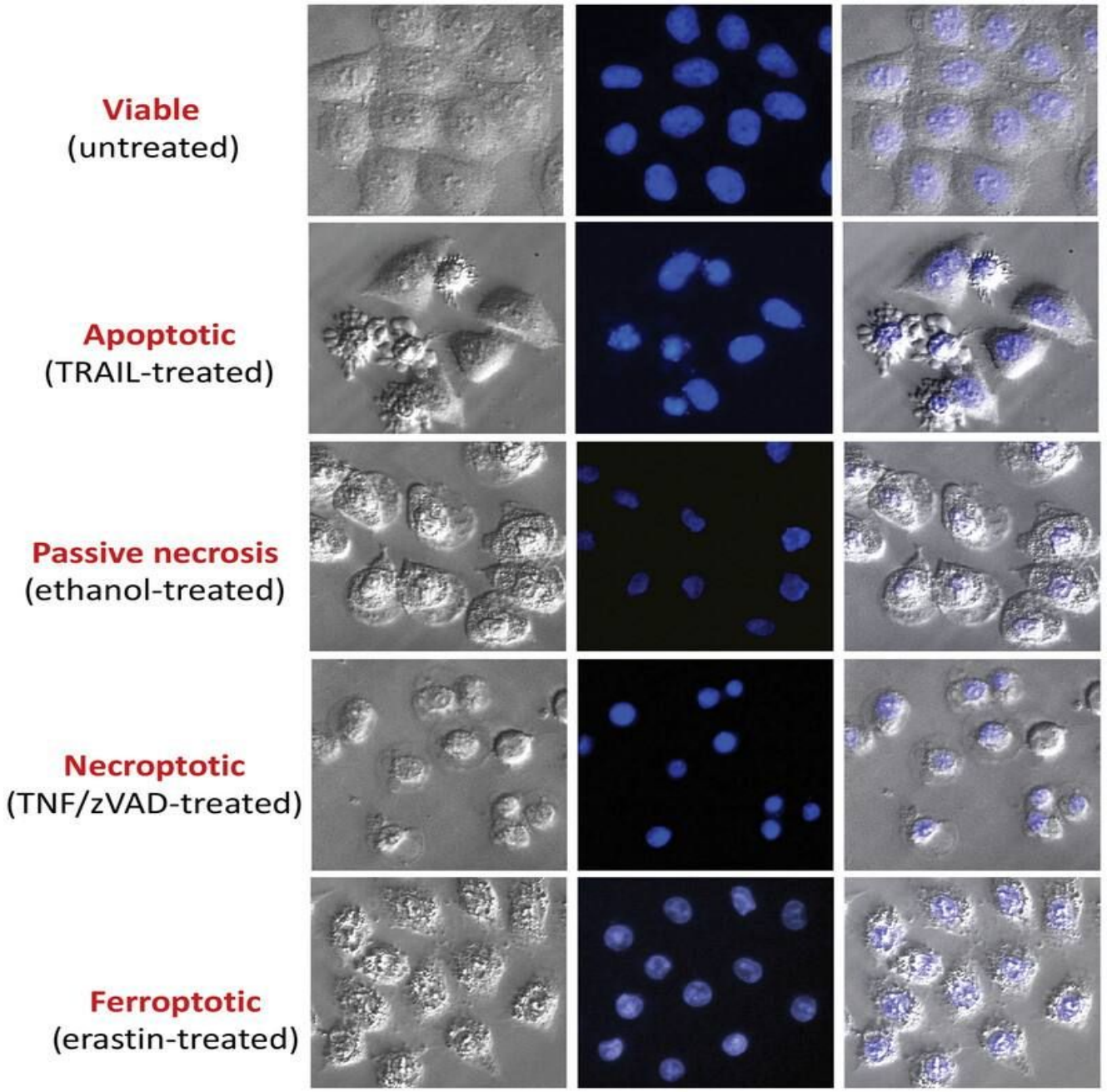


Introduction to Necrosis

Necrosis is the death of cells or tissues in the body due to injury, disease, or lack of blood supply. It is a serious condition that can have severe consequences if not properly managed.

SA **by Surendra Patel**

Made with Gamma


Viable
(untreated)
Apoptotic
(TRAIL-treated)
Passive necrosis
(ethanol-treated)
Necroptotic
(TNF/zVAD-treated)
Ferroptotic
(erastin-treated)

TYPES OF NECROSIS

General Pathology

COAGULATIVE NECROSIS

- • Most common type
- • Due to ischemia/infarction (except brain)
- • Gross: firm, pale
- • Microscopy: cell outlines preserved, nuclei disappear
- • Example: Myocardial infarction

LIQUEFACTIVE NECROSIS

- • Enzymatic digestion dominates
- • Seen in brain infarcts, abscesses
- • Gross: liquid, pus-like
- • Example: Cerebral infarction

CASEOUS NECROSIS

- • 'Cheese-like' appearance
- • Seen in tuberculosis, fungal infections
- • Gross: soft, crumbly
- • Microscopy: granular debris + granulomatous inflammation

FAT NECROSIS

- • Enzymatic: pancreatitis → lipase action, saponification
- • Traumatic: after trauma to fat (breast, subcutaneous tissue)
- • Gross: chalky white deposits

FIBRINOID NECROSIS

- • Immune-mediated vascular damage
- • Seen in vasculitis, malignant hypertension
- • Microscopy: bright pink fibrin-like deposits in vessel walls

GANGRENOUS NECROSIS

- • Clinical term (not a distinct pattern)
- • Dry gangrene: coagulative necrosis (ischemia)
- • Wet gangrene: bacterial infection superimposed
- • Gas gangrene: Clostridium infection with gas bubbles

Necrosis Type	Gross Image	Histology Slide	Key Visual Features to Label
Coagulative	pale infarcted tissue (e.g. kidney, heart)	H&E slide showing “ghost” cells, loss of nuclei	preserved architecture, eosinophilic cytoplasm, absent nuclei
Liquefactive	abscess / pus or soft/cystic area	tissue with many neutrophils, cell debris, “liquid” spaces	loss of structure, neutrophils, macrophages
Caseous	cheese-like necrotic material (e.g. lung)	granuloma with central necrosis, Langhans giant cells	central eosinophilic debris, surrounding macrophages/lymphocytes
Fat necrosis	chalky white fat (e.g. pancreas, breast)	adipocytes with basophilic calcium deposits (saponification)	“soap” deposits, disrupted fat cells
Fibrinoid	vessel wall with bright pink (fibrin-like) deposits	vessel sections with immune complexes + fibrin	“fibrinoid” deposits, damaged vessel wall
Gangrenous	blackened / dry (ischemic) or wet gangrene	mixed features depending on infection	tissue darkening, liquefaction + bacterial invasion

Thank You