

HM/CH-1/L-10

MORPHOLOGY OF CELL INJURY

MORPHOLOGIC FORMS OF CELL INJURY

MECHANISMS

1. Reversible cell injury
2. Deranged cell metabolism
3. Irreversible cell injury
4. Programmed cell death
5. Residual effects
6. After-effects

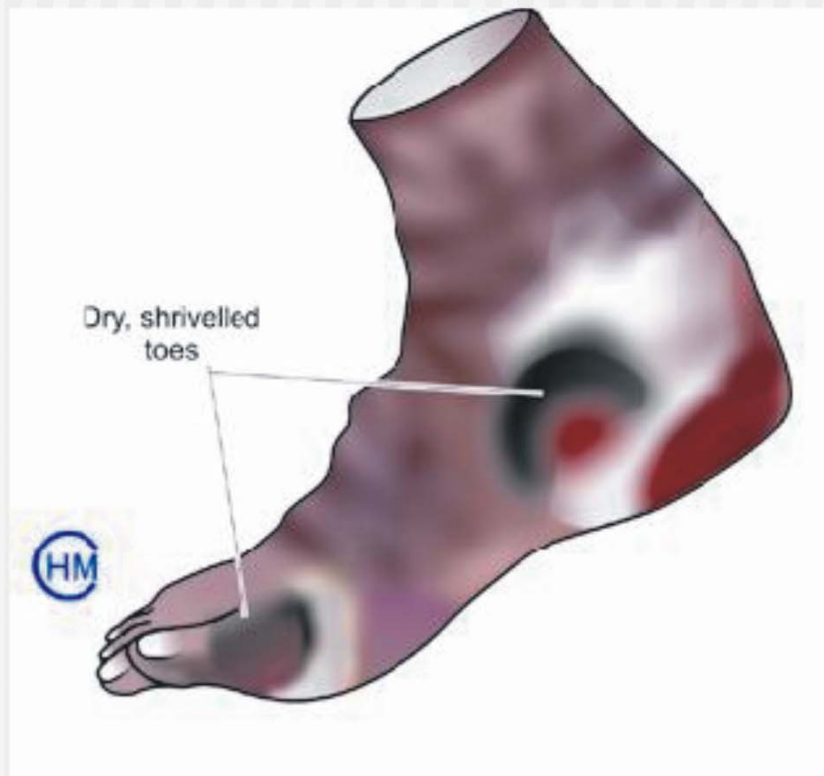
NOMENCLATURE

1. Retrogressive changes(degenerations)
2. Intracellular accumulations
3. Cell death-necrosis
4. Apoptosis
5. Subcellular alterations
6. Gangrene, pathologic calcification

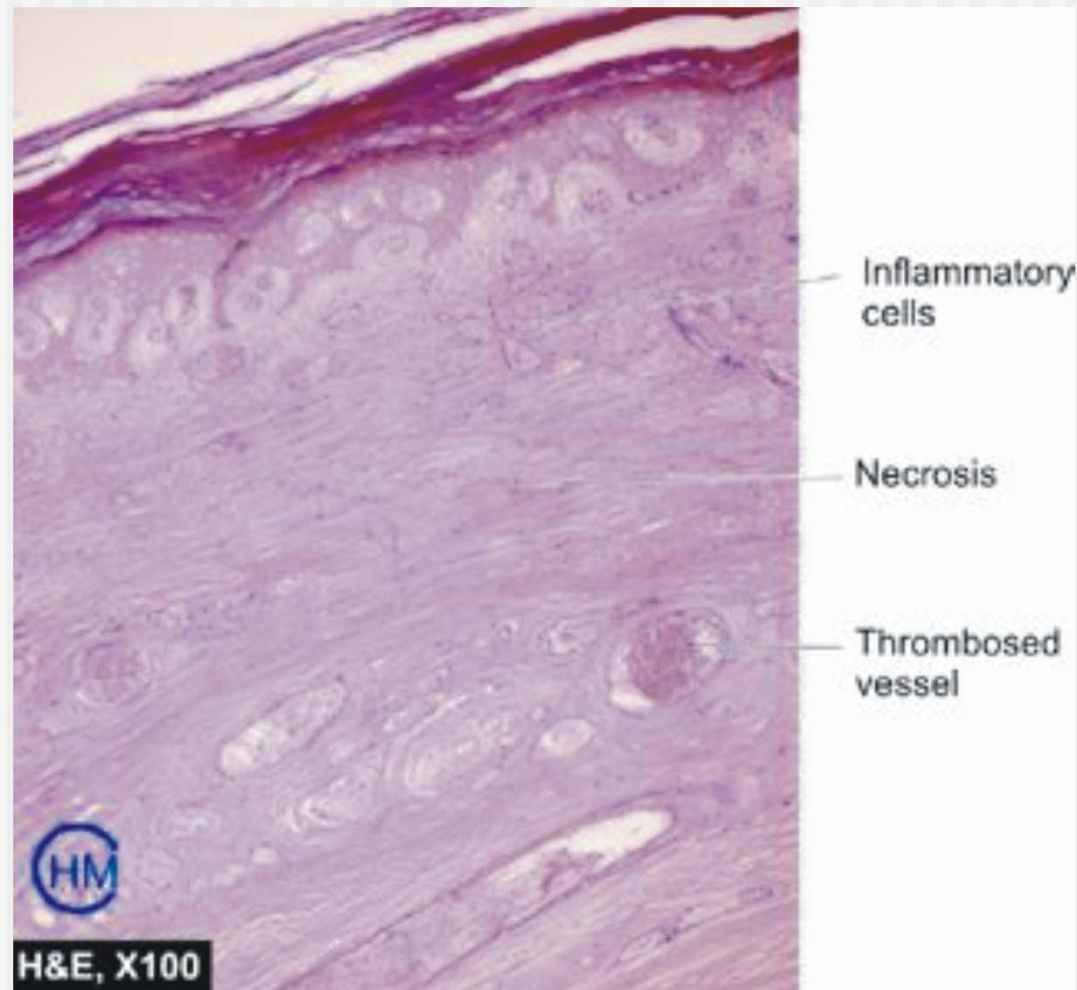
GANGRENE

- "A form of necrosis with superadded putrefaction"
- Gangrenous (necrotising) inflammation
- FORMS OF GANGRENE:
 - Dry gangrene
 - Wet gangrene
 - Gas gangrene

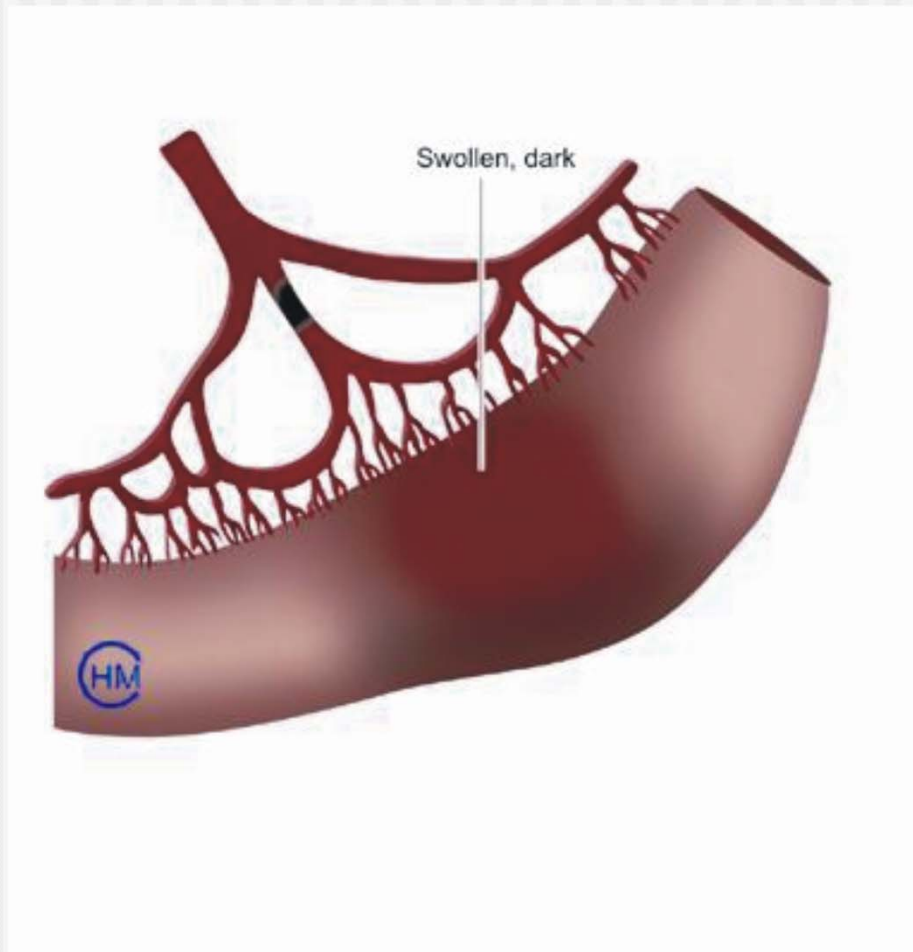
Dry gangrene



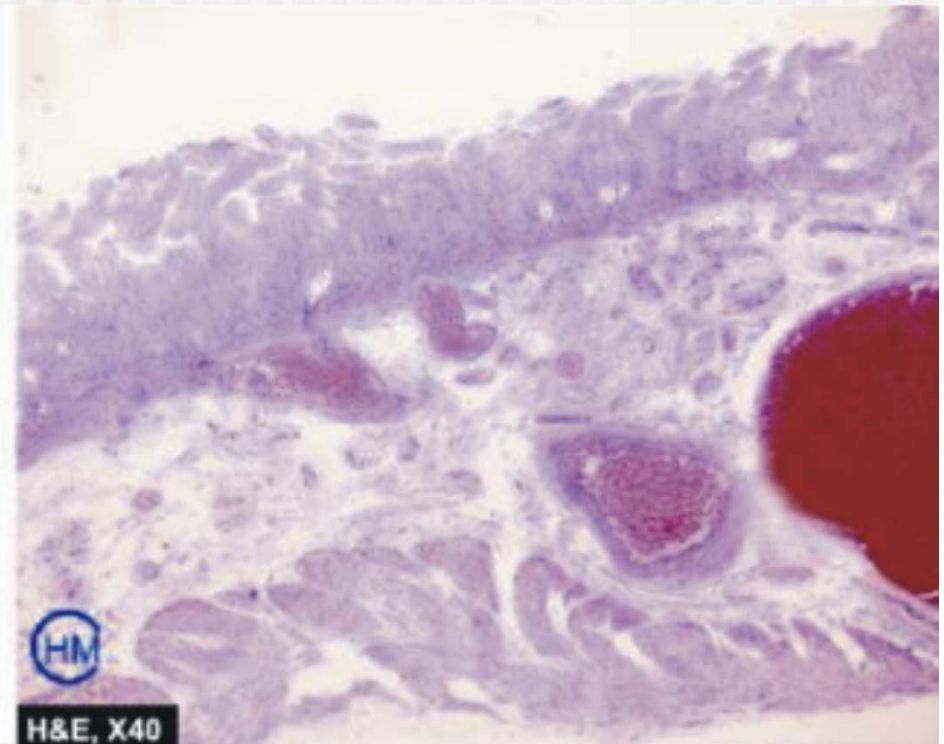
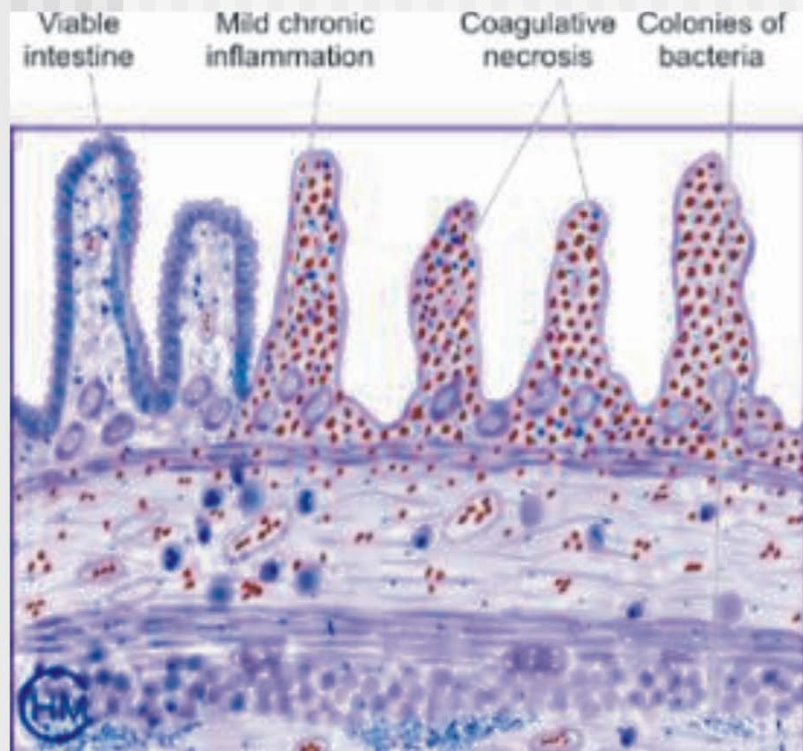
Dry gangrene: M/E



Wet gangrene



Wet gangrene



Gas gangrene

- Gas-forming clostridia
(Gram +ve anaerobic bacteria)
- PATHOLOGIC CHANGES:
 - GA
 - ME

Web Table 3.5: Contrasting Features of Dry and Wet Gangrene.

Feature	Dry Gangrene	Wet Gangrene
1. Site	Commonly limbs	More common in bowel
2. Mechanisms	Arterial occlusion	More commonly venous obstruction, less often arterial occlusion
3. Macroscopy	Organ dry, shrunken and black	Part moist, soft, swollen, rotten and dark
4. Putrefaction	Limited due to very little blood supply	Marked due to stuffing of organ with blood
5. Line of demarcation	Present at the junction between healthy and gangrenous part	No clear line of demarcation
6. Bacteria	Bacteria fail to survive	Numerous present
7. Prognosis	Generally better due to little septicaemia	Generally poor due to profound toxemia

PATHOLOGIC CALCIFICATION

- Dystrophic calcification:
 - dead/degenerated tissues
 - normal Ca metabolism
 - normal serum Ca
- Metastatic calcification:
 - normal tissues
 - deranged Ca metabolism
 - raised serum Ca

Dystrophic calcification: etiology

■ Calcification in dead tissues:

Caseous necrosis, liquefactive necrosis, fat necrosis, Gamna-Gandy bodies, infarcts, thrombi, haematomas, dead parasites, breast cancer, toxoplasmosis

■ Calcification in degenerated tissues:

Dense old scars, atheromas, Monckeberg's sclerosis, stroma of tumours, cysts, calcinosis cutis, senile degenerative changes

Pathogenesis

Denatured proteins +
phosphates + calcium =
calcium phosphates

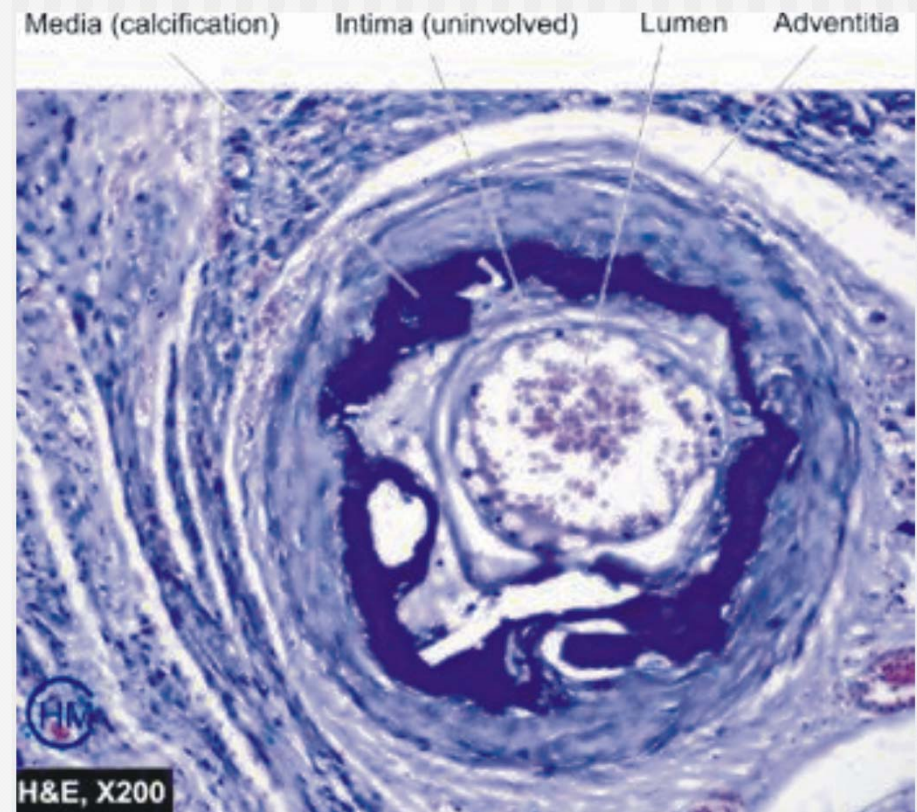
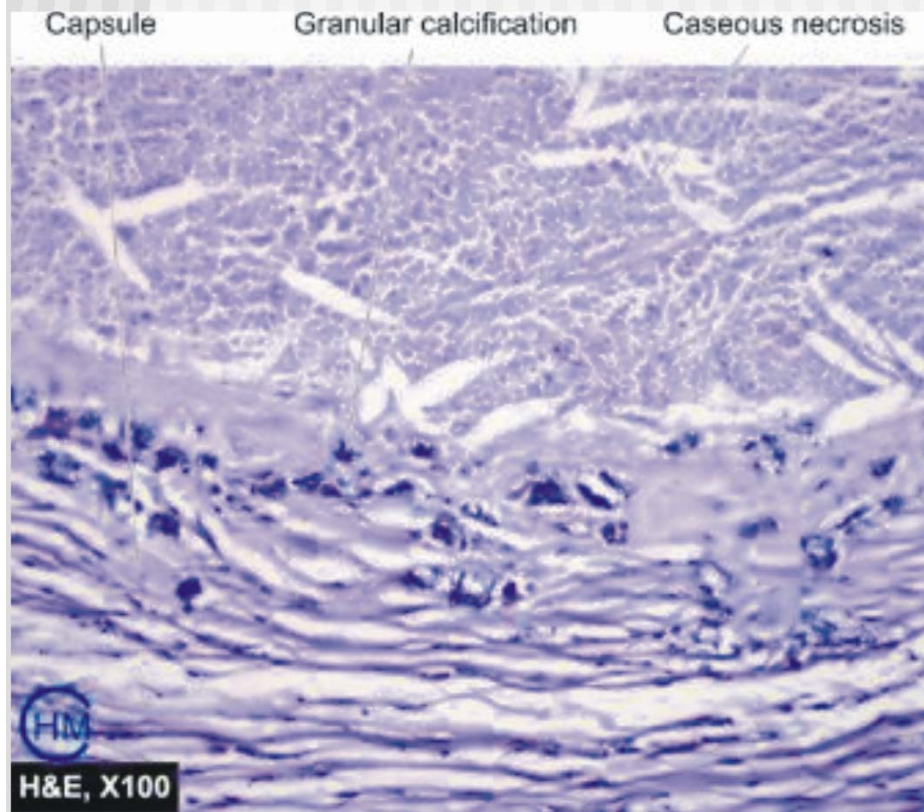
- Initiation:

intracellular in mitochondria,
extracellular in vesicles

- Propagation:

mineral crystals

Morphology



Metastatic calcification: etiology

- Excessive mobilisation from bone:
Hyperparathyroidism, bony destructive lesions, prolonged immobilisation
- Excessive absorption from gut:
Hypervitaminosis D, Milk-alkali syndrome, hypercalcaemia of infancy

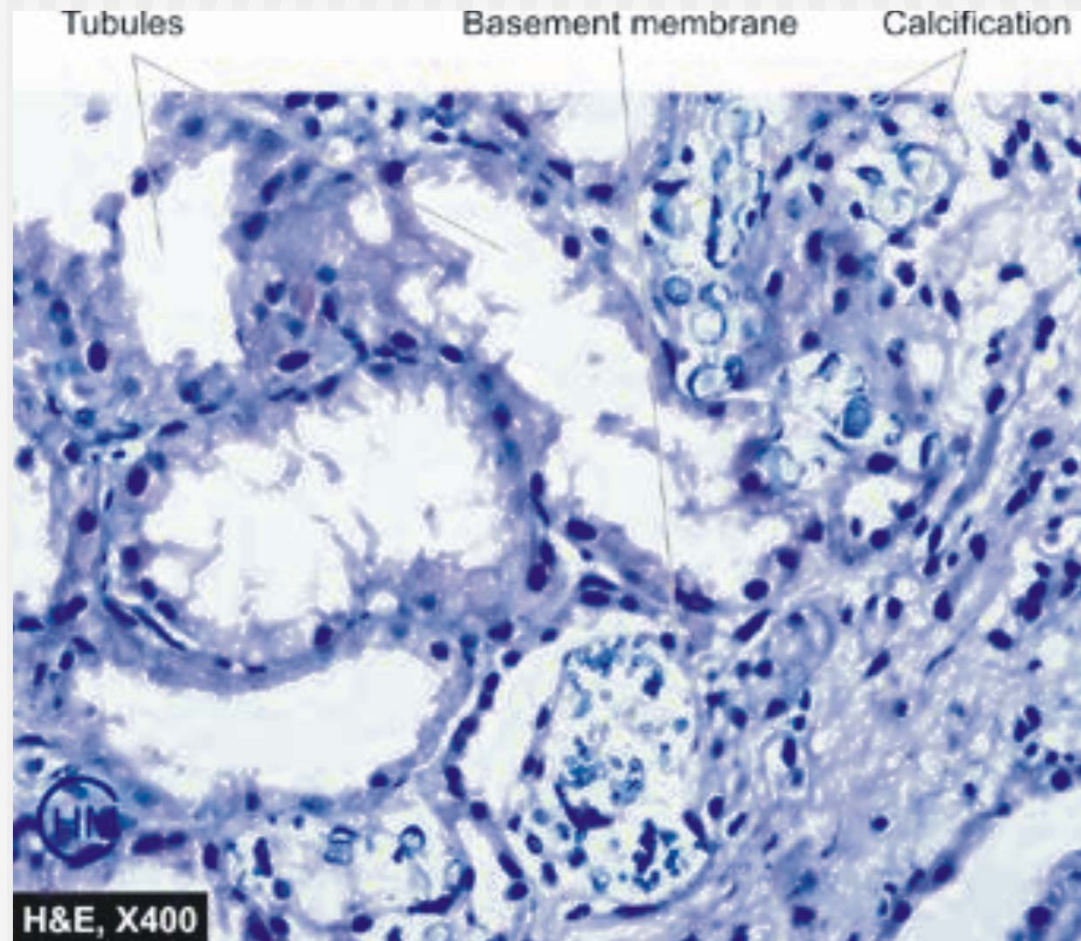
Pathogenesis

- Excessive binding of inorganic phosphate with calcium
- Precipitates of calcium phosphates at sites
- Reversible

Morphology

SITES:

- Kidneys,
- lungs,
- stomach,
- BVs,
- cornea





WEB TABLE 3.6: Differences between Dystrophic and Metastatic Calcification.



Feature	Dystrophic Calcification	Metastatic Calcification
1. <i>Definition</i>	Deposits of calcium salts in dead and degenerated tissues	Deposits of calcium salts in normal tissues
2. <i>Calcium metabolism</i>	Normal	Deranged
3. <i>Serum calcium level</i>	Normal	Hypercalcaemia
4. <i>Reversibility</i>	Generally irreversible	Reversible upon correction of metabolic disorder
5. <i>Causes</i>	Necrosis (caseous, liquefactive, fat), infarcts, thrombi, haematomas, dead parasites, old scars, atheromas, Mönckeberg's sclerosis, certain tumours, cysts, calcinosis cutis	Hyperparathyroidism (due to adenoma, hyperplasia, CRF), bony destructive lesions (e.g. myeloma, metastatic carcinoma), prolonged immobilisation, hypervitaminosis D, milk-alkali syndrome, hypercalcaemia of infancy
6. <i>Pathogenesis</i>	Increased binding of phosphates with necrotic and degenerative tissue, which in turn binds to calcium forming calcium phosphate precipitates	Increased precipitates of calcium phosphate due to hypercalcaemia at certain sites e.g. in lungs, stomach, blood vessels and cornea