NECROSIS



NECROSIS VS APOPTOSIS

- Necrosis is the death of cells or organs within living organism.
- In contrast, apoptosis is a naturally occurring programmed and targeted cause of cellular death.
- While apoptosis often provides beneficial effects to the organism, necrosis is almost always detrimental and can be fatal.





Apoptosis	Necrosis
Affects single cells	Affects groups of neighboring cel
No inflammatory response	Significant inflammatory response
Cell shrinkage	Cell swelling
Membrane blebbing but integrity maintained	Loss of membrane integrity
Increased mitochondria membrane permeability, release of proapoptotic proteins and formation of apoptotic bodies	Organelle swelling and lysosomal leakage
Chromatin condensation and non-random DNA fragmentation	Random degradation of DNA
Apoptotic bodies ingested by neighboring cells	Lysed cells ingested by macrophage

https://drrajivdesaimd.com/wp-content/uploads/2013/12/apoptosis-vs-necrosis-1.png

NECROSIS

Causes of necrosis

- Necrosis is caused by :
- Chemical substances such as mineral acids, caustic alkalines and phenol.
- Physical influences such as heat, cold, electricity
- Mechanical forces
- Nutrition (failure of blood to reach the organs because of thrombosis, embolism, volvulus, invagination, tumor, abscess, etc.)
- ✓ **Neural causes** (peripheral nerve destruction)
- Infectious causes (bacterial: necrobasillosis, tuberculosis, pseudotuberculosis. viral: rinderpest, foot and mouth disease. mycotic: aspergillosis, blastomycosis

NUCLEAR CHANGES IN NECROSIS

- Four types of nuclear changes may occur:
- Pyknosis : condensation of nuclear chromatin into a dark round, homogeneous mass smaller than in a normal nucleus.
- Karyorrhexis : breaking up of the nucleus into numerous pieces.
- Karyolysis : dissolution of nuclear chromatin, leaving a ghost form of the nucleus
- And finally : complete absence of the nucleus.



Figure 1-16 Cytoarchitecture of Cellular Necrosis. A, Schematic representation of nuclear and cytoplasmic changes in the stages of necrosis. rER, rough endoplasmic reticulum. B, Pyknosis and karyolysis, renal cortex, chloroform toxicosis, mouse. Some tubular epithelial cells have undergone hydropic degeneration; others are necrotic with pyknosis (arrow) or karyolysis (arrowhead). H&E stain. C, Karyorrhexis, lymphocytes, spleen, dog. Necrotic lymphocytes have fragmented nuclei (arrow) because of parvovirus infection. H&E stain. (A courtesy Dr. M.A. Miller, College of Veterinary Medicine, Purdue University; and Dr. J.F. Zachary, College of Veterinary Medicine, University of Illinois; B and C courtesy Dr. L.H. Arp.)

TYPES OF NECROSIS

Coagulative necrosis

Caseous necrosis

Liquefactive necrosis

Gangrene

COAGULATIVE NECROSIS

- The denaturation of cytoplasmic proteins, which at the histologic level imparts an opaque and intense cytoplasmic eosinophilia to necrotic cells.
- Coagulative necrosis is a typical early response to hypoxia, ischemia, or toxic injury.
- Occurs in **infarcts** of the kidney, spleen and myocardium.
- **Grossly**, the area of necrotic tissue is yellowish grey or pale yellow in color and is depressed below the surface of the surrounding tissue.
- Microscopically, the architectural outline of the tissue is preserved but the cellular detail is lost.

COAGULATIVE NECROSIS



Coagulative Necrosis, Infarct, Kidney, Ox.

CASEOUS NECROSIS

- Caseous, from the Latin word for cheese, refers to the curdled or cheese like gross appearance of this form of necrosis.
- Characterized by the loss of both architectural and cellular detail.
- Macroscopically, caseation may appear as crumbled, granular, or laminated yellow-white exudate in the center of a granuloma or a chronic abscess.
- Histologically, the lysis of leukocytes and parenchymal cells converts the necrotic tissue into a granular to amorphous eosinophilic substance with basophilic nuclear debris.
- This type of necrosis is characteristic of the lesions of **tuberculosis**.

CASEOUS NECROSIS



Tuberculosis (Caseous Necrosis), Lymph Node, Transverse Section, Ox.

CASEOUS NECROSIS









LIQUEFACTIVE NECROSIS

- Liquefactive necrosis is typical of organs in which the tissues have a lot of lipid.
- Its often seen in brain and spinal cord and the term for the macroscopic (gross) appearance of necrosis in the brain and spinal cord is malacia.
- In liquefactive necrosis, cells are lysed, and the necrotic tissue is converted to a fluid phase.
- Grossly, the dead tissue is in a liquid form and sometimes has a creamy or pasty consistency.
- Microscopically, empty spaces are seen since the liquefied material are lost during the processing of the tissue. A pinkish staining protein material may remain or may not as a precipitate from the liquid.

LIQUEFACTIVE NECROSIS



Liquefactive Necros Brain, Goat.



Liquefactive necrosis. An infarct in the brain showing dissolution of the tissue.

GANGRENE

- Gangrene denotes a type of necrosis that tends to develop at the distal aspect of extremities, such as the limbs, tail, or pinnae, or in dependent portions of organs, such as the mammary glands or lung lobes.
- Gangrene can be designated as **wet** or **dry**.
- Wet gangrene occurs if the necrotic tissue is infected by certain bacteria.
- Hydrogen sulfide is produced
- As a result of hydrogen sulfide binding with blood pigments, the tissues become greenish-black in color.
- E.g. Septic ulcers of the uterus, gangrenous pneumonia

GANGRENE

- If those bacteria are gas forming (e.g., Clostridium spp.), then wet gangrene becomes gas gangrene.
- E.g. Blackleg and malignant edema.
- **Dry gangrene** is the result of decreased vascular perfusion and/or loss of blood supply.
- ✓ It is a form of infarction resulting in coagulative necrosis that imparts a dry, leathery texture to the necrotic tissue, providing that it remains <u>free of putrefactive bacteria</u>.
- ✓ Necrosis in case of tearing of the umbilical cord after birth.
- Dry dying fetus in utero.



RESULTS OF NECROSIS

- Dissolution and resorption of necrotic tissue.
- Abscess formation
- Sloughing and desquamation of the necrotic tissue
- Encapsulation without liquefaction ; a containing wall of leukocytes and connective tissue is formed around the mass and the area become encapsulated.
- Organisation of necrotic tissue: occurs when necrotic tissue is invaded with capillaries, connective tissue and leucocytes. The necrotic tissue is digested by leukocytes then the areas are infiltrated with connective tissue leaving a scar.
- Calcification of necrotic material
- Gangrene
- Atrophy
- Regeneration

<u>INF & RCTUS</u>

Infarction is a local area of ischemic necrosis in a tissue caused by occlusion of the arterial supply or venous drainage.

It is usually formed in organs with insufficient veins, without collateral connections.

As a result of embolism, thrombosis or spasm, a complete blockage of the artery does not lead to the region where the endothelium feeds, and ischemia (local anemia) develops.

The coagulation necrosis on the side of the occluded vein is called infarct.



In the formation of infarctus

The organ is resistant to oxygen (for example, the kidneys are unstable, the infarctus is seen)

Whether blood circulation, vascularisation is sufficient,

collateral vascularisations are not adequately fed and fed on other vessels (eg infarctus does not occur due to excessive vascular anastomoses in skeletal muscles, skin, uterus, thyroid tissue).





ACCORDING TO MORPHOLOGY Anemic infarction

Artery blockage in organs such as the heart, kidney, spleen, brain is the result. It appears pale from the beginning.

Hemorrhagic infarction

Occurs when the venous system is obstructed. This is most commonly seen in the lungs. It also occurs in the testes and ovaries. It is reddish due to blood stagnation.

ACCORDING TO TIME

New (acute), old (chronic)





Infartos hemorrágicos

MORPHOLOGICAL APPEARANCE

Coagulation NECROSIS is occured

Lungs, heart-like organs do not have a specific shape. It's like a map and its boundaries are irregular.

It is wedge-shaped (like triangular). in the kidneys and the spleen.

Infarcts tend to be wedge-shaped; the base of the wedge is at periphery, with the occluded vessel at the apex. The margins of the infarct may be irregular, a reflection of the vascular supply adjacent, nonaffected tissue.

However, almost six of the capsules are not significantly affected by being fed with blood vessels in the capsule.



Abomasitis thromboembolica, mucosal multiple hemorrhagic infactus; bacterial endocarditis



Infarctus

According to the organ affected by the infarct area; At the beginning the infarctus region is

hyperemic and slightly inflamed.

After a day or two, the necrosis part becomes pale, it gets a decent color, its borders become clear and there is a hyperemic belt around it.

In the following days, it becomes brown-yellow and soft.

This necrotic area is cleared and organized by macrophages. In this case; it is closed with granulation texture consisting of developing connective texture and capillar veins.

Then the veins are reduced, the collagen fibers are increased and the scatrix texture is formed. This is hard, greyish and collapsing.

