Necrosis Types

PLAY PICMONIC

Coagulative

Clog

The coagulative necrosis type can be found in most tissues with ischemia/infarct except the brain. Injury of the cells causes denaturation of the enzymes, and the architecture of the cells is preserved. However, the nuclei disappear with increased cytoplasmic binding to the eosin stain.

Liquefactive

Liquor

The liquefactive necrosis type can be found in brain infarcts and bacterial abscesses. This necrosis is characterized by the digestion of the tissue by lysosomal enzymes produced by neutrophils. It is a type of necrosis that transforms the softening necrotic tissue into a pastelike mush, a liquid, viscous mass, or watery debris. Early histologic findings will show macrophages and cellular debris, and late findings will show cystic spaces and cavitation in CNS.

Brain Infarcts

Brain Infarction-fart

Brain infarcts seen in stroke patients are characterized by a focal blockage of blood supply to the brain. Necrosis can occur, showing loss of neurons and neuroglial cells.

Caseous

Cheesy

The caseous necrosis type can be found in tuberculosis, systemic fungal infections (e.g., Histoplasma capsulatum), and Nocardia. Caseous necrosis is characterized by a cheese-like appearance that occurs from the necrosis tissue's friable white appearance. Macrophages digest cells, but not all cells, causing granular debris. Granuloma will be seen as a collection of debris and fragmented cells surrounded by macrophages and lymphocytes. This type of necrosis is a combination of coagulative and liquefactive necrosis.

Tuberculosis

TB-TV

Tuberculosis is the most common cause of caseous necrosis and presents with granuloma in the lungs.

Fat

Fat-guy

The fat necrosis type can be found in acute pancreatitis and trauma. This necrosis is characterized by a chalky-white appearance due to the deposition of calcium from a process called saponification. Histologic findings will show necrotic adipose tissue without peripheral nuclei, and saponification appears dark blue on the H&E stain.

Acute Pancreatitis

Acute-angle Pancreas-on-fire

Saponification in acute pancreatitis occurs due to the binding of calcium and lipase. Lipase is produced from damaged pancreatic cells. Acute pancreatitis is categorized as enzymatic fat necrosis.

Trauma

Trauma-spike

Saponification in trauma occurs due to the binding of calcium and fatty acids. Fatty acids are released from damaged cells, such as injury to breast tissue. Trauma-induced necrosis is categorized as non-enzymatic fat necrosis.

Fibrinoid

Fabric

The fibrinoid necrosis type can be found in PAN, hypertensive emergency, and preeclampsia. It occurs due to necrotic damage to the blood vessel walls causing plasma protein leakage in combination with immune complex deposition (type III hypersensitivity reaction). These will result in bright pink staining from the eosinophilic layer of the wall microscopically.

Immune Vascular Reactions

Immune-moon Vessels

Polyarteritis nodosa (PAN) shows an immune-vascular reaction with fibrinoid necrosis. It is a hallmark lesion of this disorder.

Non-Immune Vascular Reactions

Nun-Immune-moon Vessels

Hypertensive emergency and preeclampsia can cause non-immune vascular reaction fibrinoid necrosis.

Gangrenous

Gang-of-green

The gangrenous necrosis type can be found in the lower extremities and GIT after chronic ischemia. There are two types of gangrenous necrosis: dry, caused by ischemia, and wet, caused by superinfection. Dry gangrene is characterized by coagulative necrosis that resembles mummified tissue. Wet gangrene is characterized by liquefaction necrosis superimposed on coagulation.

Lower Extremities

Lower Extremities

Blood supply lost in the lower extremities causes gangrenous necrosis. An example of this is an extensive arterial thrombosis on sepsis-induced DIC.