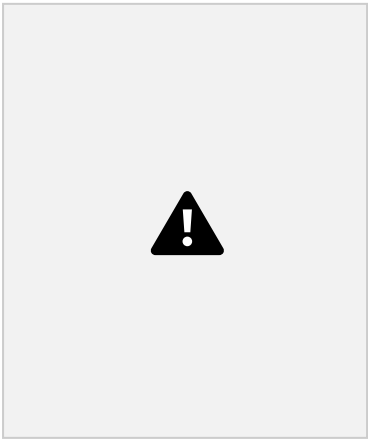
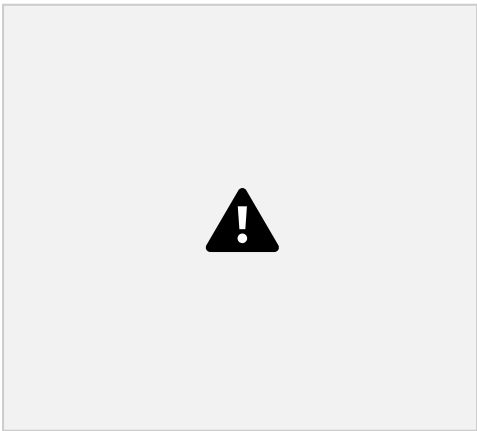


ionic/osmotic imbalance cell swelling.



INTRACELLULAR DEGENERATIVE CHANGES (CELL SWELLING, BALLOONING DEGENERATION, DEGENERATION INVOLVING FATS, INTRACELLULAR INCLUSIONS)

- Cell swelling/cloudy swelling
- Most common response to injury
- Observed in the epithelium and endothelium
- Autolysis (self-digestion of the cell bc of death; tissue, cells undergo changes w/o diseases, cannot conclude the cause of death) results in similar microscopic lesion

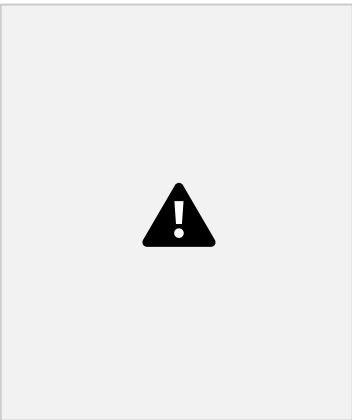


Intracellular degenerative changes

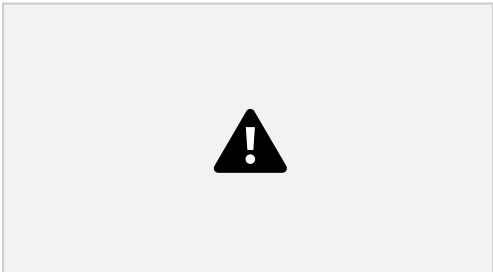
- Hydropic degeneration /ballooning degeneration more advanced; for microscopic lesion only
- Associated with epithelial lesions and clues to diagnosis of specific disease ex. Canine Distemper inclusion bodies in urinary tract epithelium, carbohydrate rumen overload in the cattle

VP61 GENERAL PATHOLOGY NOTES (jong

pogi)



(hydropic degeneration in Canine Distemper inclusion bodies)



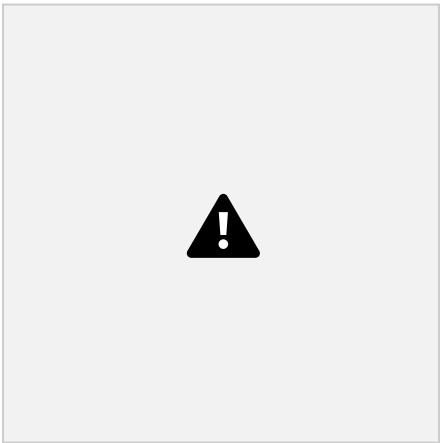
Eosin = pinkish color

Eosinophilic inclusion bodies

Degeneration involving fat

1. Fatty degeneration

- abnormal accumulation of fat in the parenchymal cells - Liver is the best known organ but also occur in renal tubular epithelium and myocardial cells
- Well known in diabetes (more on cats and dogs; not naman sa ruminants. Bc of Diet) and ketosis (body does not have enough carbohydrates to burn for energy)
- Utilization/availability of glucose is impaired, lipids are mobilized as an energy source; liver functional overloaded



Accumulation of triglycerides in fatty liver. Defects in any of the steps of uptake, catabolism, or secretion can result in lipid le mechanisms leading to accumulation.



- Microscopic lesion: large, clear, discrete droplets with foamy appearance
- Gross appearance: enlarged, uniformly yellow corresponding to the extent of fat accumulation, greasy texture on cut surface; dogs- groups of lobules affected called fatty cysts

- Differential diagnosis: hydropic degeneration or glycogen stored and postmortem change
- Special stains for kidney and myocardium; cats have large amount of fat in renal tubules therefore pale

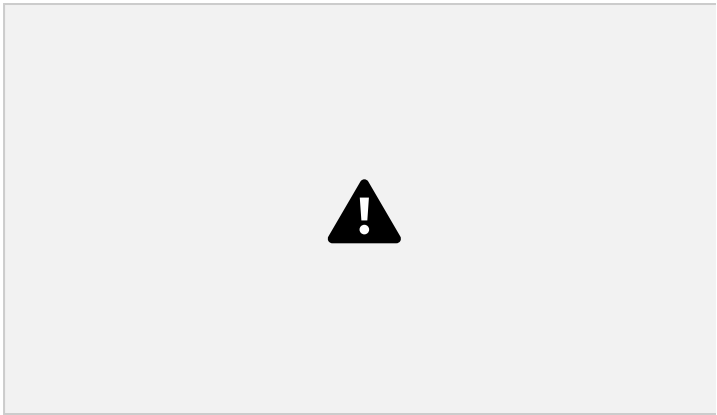
2. Fatty infiltration/fatty replacement

- Accumulation of adipose cells in tissue which are not normally present (2 types of adipose cells: Unilocular *most common* or white fat and the multilocular or the brown fat) - Replacing atrophied tissue
- Occurs in myocardial and skeletal muscle less common pancreas
 - Steatosis : pale muscle due to fatty infiltration in heavy muscles of leg, loin and shoulder in cattle and pigs - Panniculus adiposus where the fat should be; but if meron sa ibang organs dapat wala kay magkaroon ng fatty infiltration

Intracellular inclusion

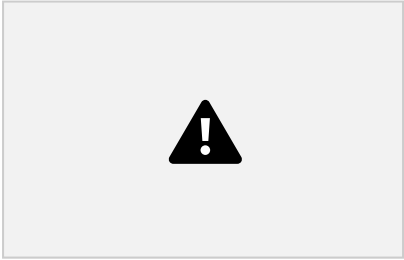
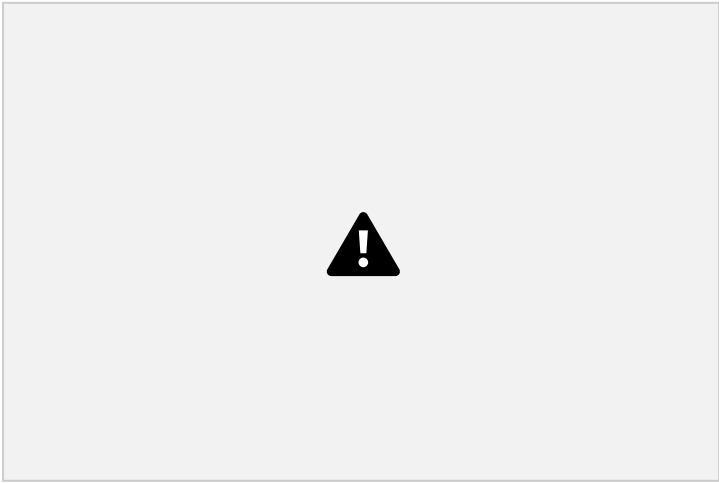
- Hyaline droplets/hyaline degeneration

Microscopic: eosinophilic structures in the cytoplasm in renal



Ovine, submandibular lymph node, with a disease called “caseous lymphadenitis” caused by infection with bacteria *Corynebacterium pseudotuberculosis*; note the type of necrosis in this exudate is caseous (not quite liquefactive, but more broken than coagulation necrosis, IE it would be a thick pasty texture, if you could cut/feel it); onion like

pneumonia due to *Rhodococcus equi*; not caseous exudate (arrows)

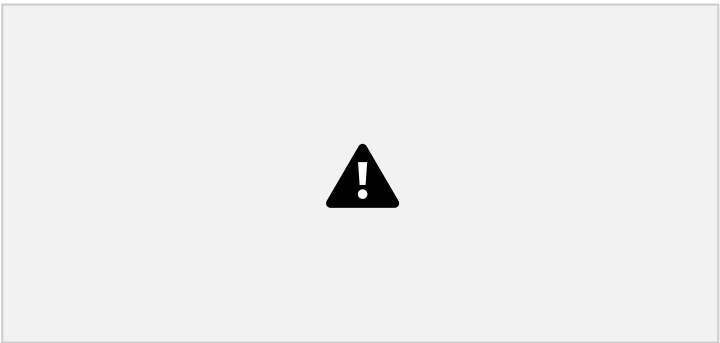


equine, lung tissue, pyrogranulomatous

VP61 GENERAL PATHOLOGY NOTES (jong pogi)

GANGRENOUS NECROSIS

- Definition = necrosis (usually ischemic) of extremities eg.



Digits, ear tips

- **Dry gangrene** = coagulation necrosis of an extremity
- **Wet gangrene** = when the coagulative necrosis of dry gangrene is modified by *liquefactive action of saprophytic/putrefactive bacteria*
- Gangrene (lesion) is caused by





POST-MORTEM CHANGES AND PATHOLOGICAL PIGMENTS

Postmortem scavenging – postmortem removal of organs of carcass by carrion eating animals (scavengers)

Predation – a predator is an animal that hunts and kills prey for food in an act called predation

Post-mortem autolysis

- Autolysis
- self-digestion by the tissue enzymes after death
- liver, pancreas, and kidney change relatively quickly
- Loss and increase of weight in organs
- Fixed ASAP: *retina is the most sensitive*, seminiferous tubules, intestine

VP61 GENERAL PATHOLOGY NOTES (jong pogi)

Rigor Mortis

- *contraction of muscles* after death
- usually within 106 hours after death & lasts 1-2 days (glycogen stores, ambient temp)
- after death, circulation of blood ceases → muscle cells resort to anaerobic glycolysis→ glycogen stored run out & ATP depleted (required for muscle relaxation) → Ca^{++} floods into muscle cells causing myofilaments to “lock-up” mag tigas(all muscles affected, flexors/extensors, causing rigidity of joints)→ rigor gradually dissipates with autolysis of structural and functional muscle proteins

Algor Mortis

- gradual cooling of cadaver to ambient temperature (in humans 1.5 F per hr)

Livor Mortis (postmortem lividity)

- hypostatic congestion, ie. *Gravitational pooling of blood* to the dependent regions (“down side”) of the body



Postmortem clotting

- occurs in *heart and vessels*
 - *rbc’s may separate from plasma*; esp in animals with high fibrinogen eg horse=“chicken fat clot” (as seen in image)



Hemoglobin Imbibition

- HgB released by rbc breakdown (after death) → *staining tissues*.
- Especially lining of heart & blood vessels; also common in tissues of aborted fetuses

