**Degeneration Slide 1 Canine Liver**
**Go to** https://slice.edu.au/s/ae575364

**What are the main histological features of lipidosis in cells?**

a) Nucleus displaced to the periphery
b) Discrete intracytoplasmic vacuolation (clearing), often a single large lipid vacuole but can be multiple smaller vacuoles

**What is the gross appearance of a liver with hepatic steatosis/lipidosis?**

- Enlarged with rounded borders
- Pale, cream-yellow-orange in colour
- Soft or friable texture
- Greasy
- May float in water

**What are the main causes of hepatic lipidosis?**

- Entry of excess fatty acids into the liver
- Inadequate supply of proteins or cofactors to permit synthesis ofapoproteins
- Sublethal hypoxia
- Sublethal toxic injury

**Describe the process (pathogenesis) of lipid accumulation in the liver.**

- The fatty acids enter the liver (e.g. chylomicrons derived from the diet, very low-density lipoproteins (VLDL) in circulation, low density lipoproteins (LDL) mobilised from body fat depots etc.) and are oxidised as an energy source in the mitochondria of hepatocytes. Others are used by the hepatocytes to synthesise cholesterol esters or phospholipids or are oxidised to form ketone bodies

- Most incoming fatty acids are esterified by the hepatocytes to form **triglycerides**

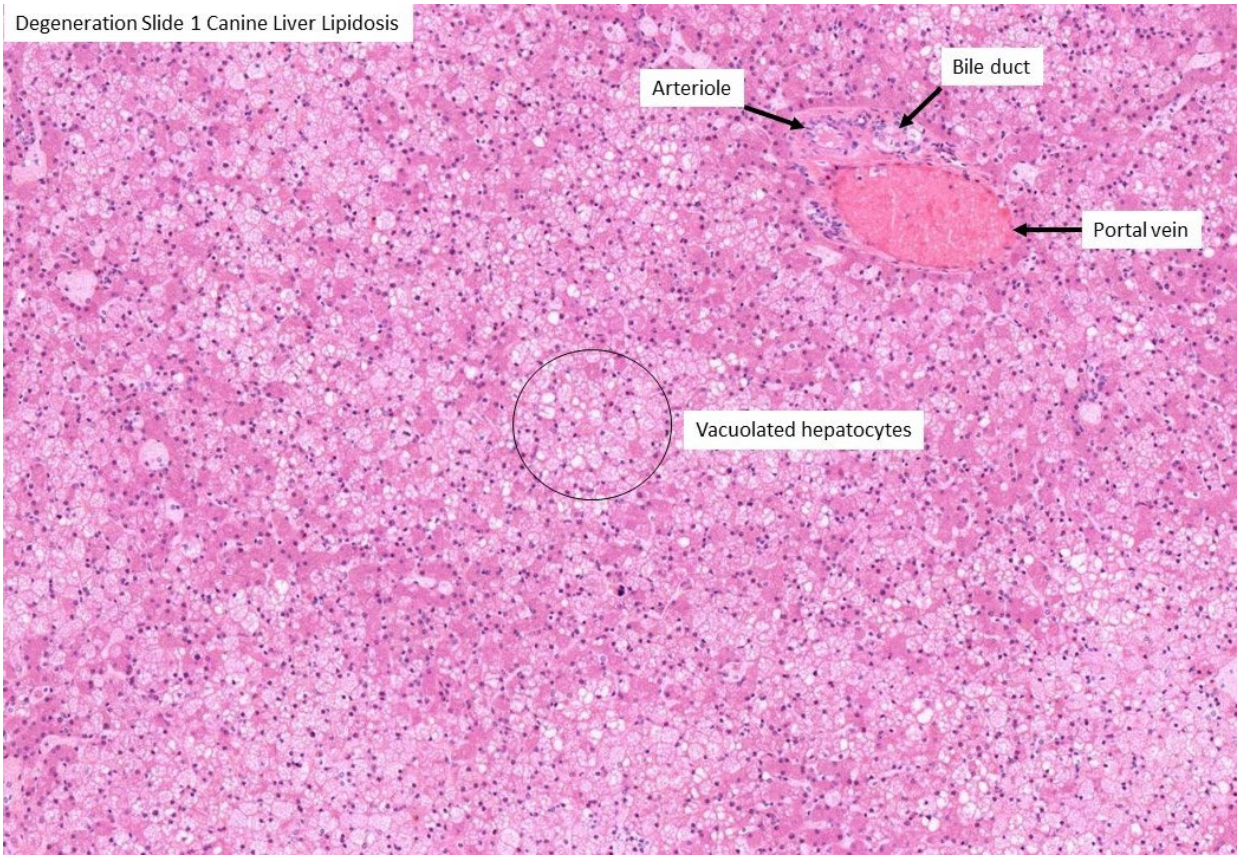
- The triglycerides are then packaged by the hepatocytes with **apoproteins** to form **VLDL** → exported into the circulation as a readily available energy source for other tissues.

- Hepatic lipidosis may develop if any of the steps in normal hepatic lipid metabolism is compromised: synthesis of apoproteins and the packaging of apoproteins and triglycerides into VLDL for export, as these steps require considerable energy consumption by hepatocytes

- Esterification of fatty acids to triglycerides is less energy-dependent and may continue, even in an injured hepatocyte

**What other organs or systems are commonly affected by lipid degeneration? Give examples.**

-Liver, Kidney, Myocardium (heart)


Degeneration Slide 1 Canine Liver Lipidosis
Arteriole
Bile duct
Portal vein
Vacuolated hepatocytes

**Degeneration Slide 3 Feline Liver**
**Go to** https://slice.edu.au/s/d23af689

**Name the different types of amyloidosis and their causes:**

**a)** AL (light chain): derived from antibody (immunoglobulin) light chains (especially λ light chains) produced by plasma cells

**b)** AA (SAA): is often referred to as secondary or reactive amyloidosis because increased hepatic synthesis and release of SAA occurs as a response to active inflammation and/or tissue damage anywhere in the body

**c)** IAPP (islet amyloid): is derived from the hormone, islet amyloid polypeptide (IAPP), which is normally co-secreted with insulin by β islet cells and antagonises the action of insulin by stimulating breakdown of muscle glycogen (increased bloodglucose)

**d)** Aβ: in association with cerebral plaques with Alzhemier's disease

**e)** APrP: Amyloid derived from misfolded proteins: in some of the transmissible spongiform encephalopathies (TSE), amyloid deposits composed of misfolded proteins may develop in the brain. in these disorders, the amyloid is thought to be due to aberrant post-translational misfolding (β-pleating) of a normal α-helical host cell membrane sialoglycoprotein (PrPc), caused by exposure to a prion (a proteinaceous infectious particle) (PrPSc)

**Find the following structures:**

- **Portal triads**
- **Bile ducts**
- **Central vein**
- **Hepatic arterial branch**
- **Portal vein**
- **Lymphatics**

**Zoom in and look for the areas of pallor. Describe the location of the amyloid accumulation and what structures are affected.**

- Location: amyloid appears as amorphous, homogeneous, eosinophilic (pink) extracellular material in the perisinusoidal space (space of Disse), between the hepatocytes and the vascular sinusoids (capillaries).
- Structures affected: hepatocytes (atrophied), sinusoids (vasculature/ blood circulation).

**What are the main effects of amyloidosis?**

- mild to moderate forms of amyloidosis may be asymptomatic

- amyloid deposits can cause physical compression of adjacent cells and compromised vascular perfusion leading to atrophy or cell degeneration (with decreased cell function) or cell death

- severe hepatic amyloidosis can cause risk of spontaneous liver rupture leading to potentially fatal haemoperitoneum (haemorrhage into the peritoneal cavity)

- animals with renal amyloidosis often succumb to renal failure and those with renal glomerular amyloidosis may suffer from hypoproteinaemia due to loss of circulating proteins through the damaged glomeruli into urine.

**DVM1: Cells to Systems 2023**
**Cell/Tissue Degeneration and Necrosis**
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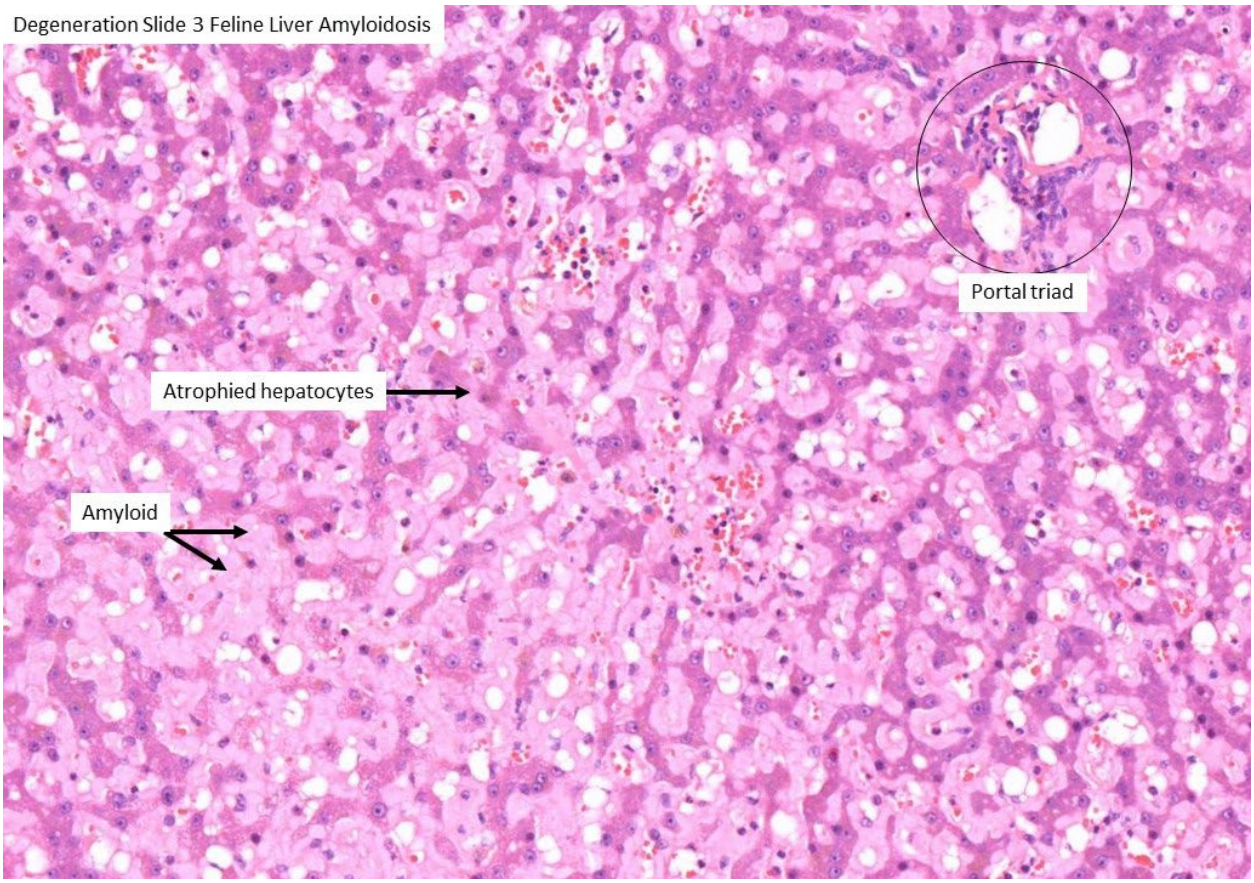

**Degeneration and Necrosis Slide 4 Rat Liver**
**Go to** https://slice.edu.au/s/b3e0271f

**Look for evidence of degeneration and necrosis. Describe the histological differences between the degenerate cells and necrotic cells.**

- Degenerate cells: swollen pale hepatocytes, filled with water (free in the cytoplasm) or lipid (vacuolated), nucleus might be displaced to the periphery
- Necrotic cells: shrunken or normal size hepatocytes, dark-staining cytoplasm (hypereosinophilic), nucleus might be darkly stained and condensed (pyknotic), fading (lytic), fragmented (karyorrhectic)

**Name the types of nuclear changes seen in a necrotic cell.**

a) Pyknotic: condensed nucleus, dark-staining
b) Karyolytic: fading nucleus
c) Karyorrhectic: fragmented nucleus

**What are the differences between apoptosis and oncotic necrosis?**

| **Feature** | **Necrosis** | **Apoptosis** |
|---|---|---|
| Cell size | Enlarged (swelling) | Reduced (shrinkage) |
| Nucleus | Pyknosis →karyorrhexis →karyolysis | Fragmentation into nucleosome-size fragments |
| Plasma membrane | Disrupted | Intact; altered structure, especially orientation of lipids |
| Cellular contents | Enzymatic digestion; may leak out of cell | Intact; may be released in apoptotic bodies |
| Adjacent inflammation | Frequent | No |
| Physiologic or pathologic role | Invariably pathologic (culmination of irreversible cell injury) | Often physiologic, means of eliminating unwanted cells; may be pathologic after some forms of cell injury, especially DNA damage |

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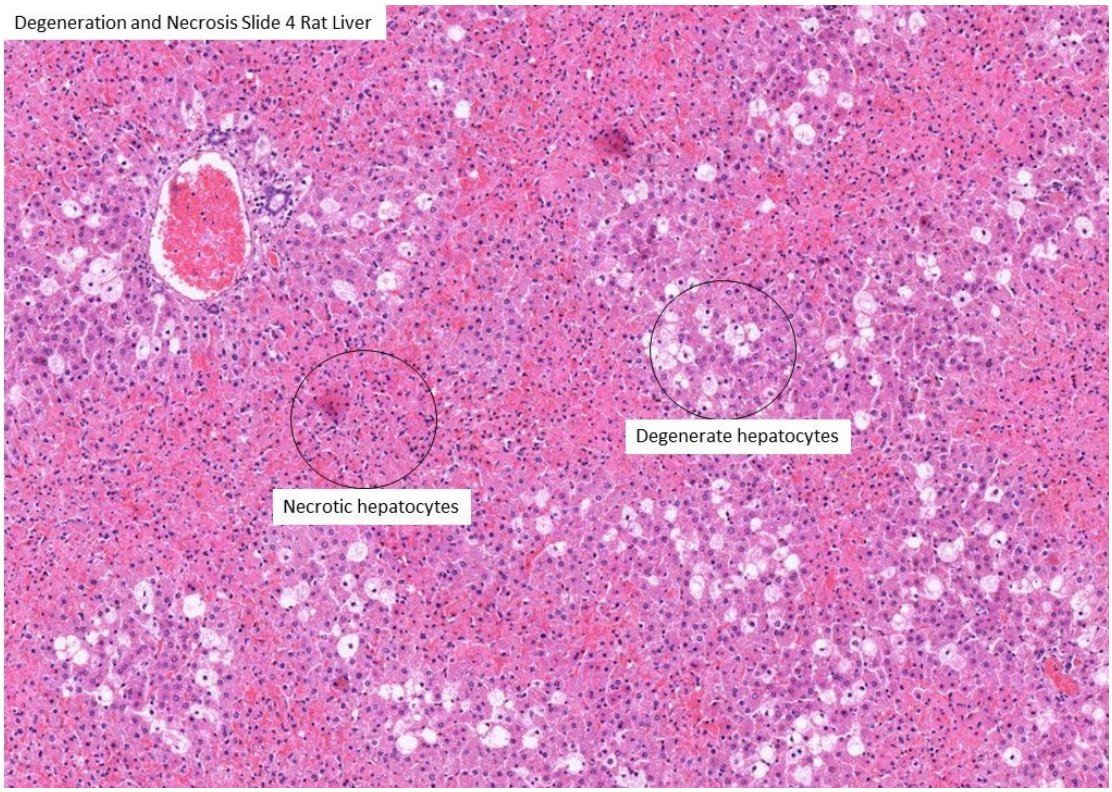


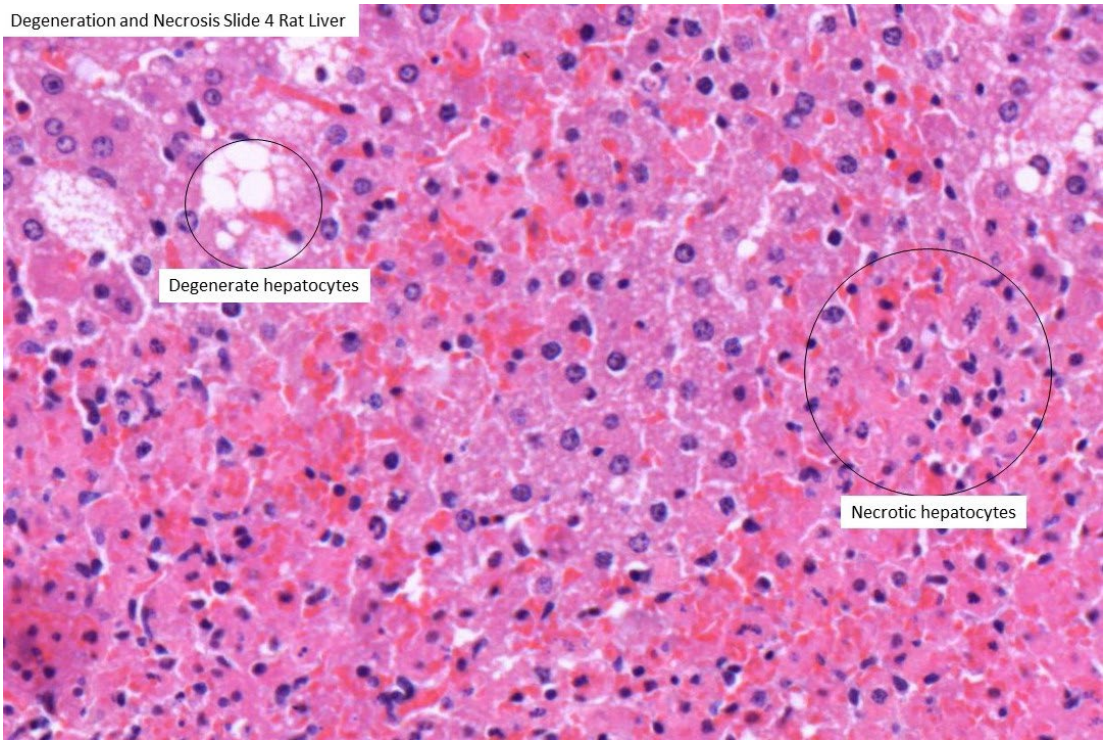

**Necrosis Slide 2 Ovine Testicle**
**Go to** https://slice.edu.au/s/0dfcedf5

**Find the following structures**

- **Testis**
- **Epididymis**
- **Tunica albuginea**
- **Tunica vaginalis**

**Find the area of coagulative necrosis. Describe the histological features of this type of necrosis.**

Cytoplasmic changes include:

Progressive severe cell swelling, increased cytoplasmic eosinophilia, cytoplasmic pallor with a moth-eaten fragmented appearance, detachment from basement membranes and surrounding cells (sloughing) or simply rupture (ghost outlines of cell debris)

Nucleus changes include:

Cell nuclei may undergo one of the following morphological changes:

- pyknosis = a shrunken, darkly staining (basophilic, hyperchromatic) nucleus
- karyorrhexis = rupture of the nuclear envelope with extrusion of dark nuclear fragments
- karyolysis = fading of the nucleus

**Give examples of tissues susceptible to coagulative necrosis.**

Heart, Kidneys, Adrenal glands, Liver, Spleen, etc

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**Necrosis Slide 3 Ovine Skeletal muscle**
**Go to** https://slice.edu.au/s/5d60c4b3

**Describe the changes to the tissue that you can recognize.**

- Necrotic cells: fragmentation of muscle fibres, brightly pink cytoplasm (hypereosinophilia), pyknosis, karyolysis, karyorrhectic nuclei
- Proteinaceous oedema
- Gas bubbles
- Inflammatory cells
- Distended lymphatics

**What causes this type of necrosis?**

This disease is produced by spore-forming anaerobic bacteria, Clostridium species (known as 'Blackleg'). Bacterial spores are eaten in contaminated feed or soil by the animal, then enter the bloodstream and lodge in various organs and tissues, including muscles. Here they lie dormant until stimulated to multiply, possibly by some slight injury to the animal. The injury reduces blood flow to the area, thereby reducing the supply of oxygen to the tissues. In the absence of oxygen, the spores germinate and multiply. As they grow, the bacteria produce toxins which destroy surrounding tissues. The toxins are absorbed into the animal's bloodstream which makes the animal acutely sick and causes rapid death.

**Name the types of gangrene and describe their difference.**

**a)** Dry gangrene: coagulative necrosis induced by ischaemia (i.e. infarction). The affected tissue eventually mummifies due to dehydration that causes shriveled, dry and brown to black tissue that eventual will slough

**b)** Wet gangrene: necrosis of tissue (usually of coagulative type) that is then colonised by bacteria causing liquefaction and putrefaction.

**c)** Gas gangrene: a type of coagulative necrosis where affected tissue becomes moist, soft, red-brown to black and is malodorous due to gas production by the bacteria

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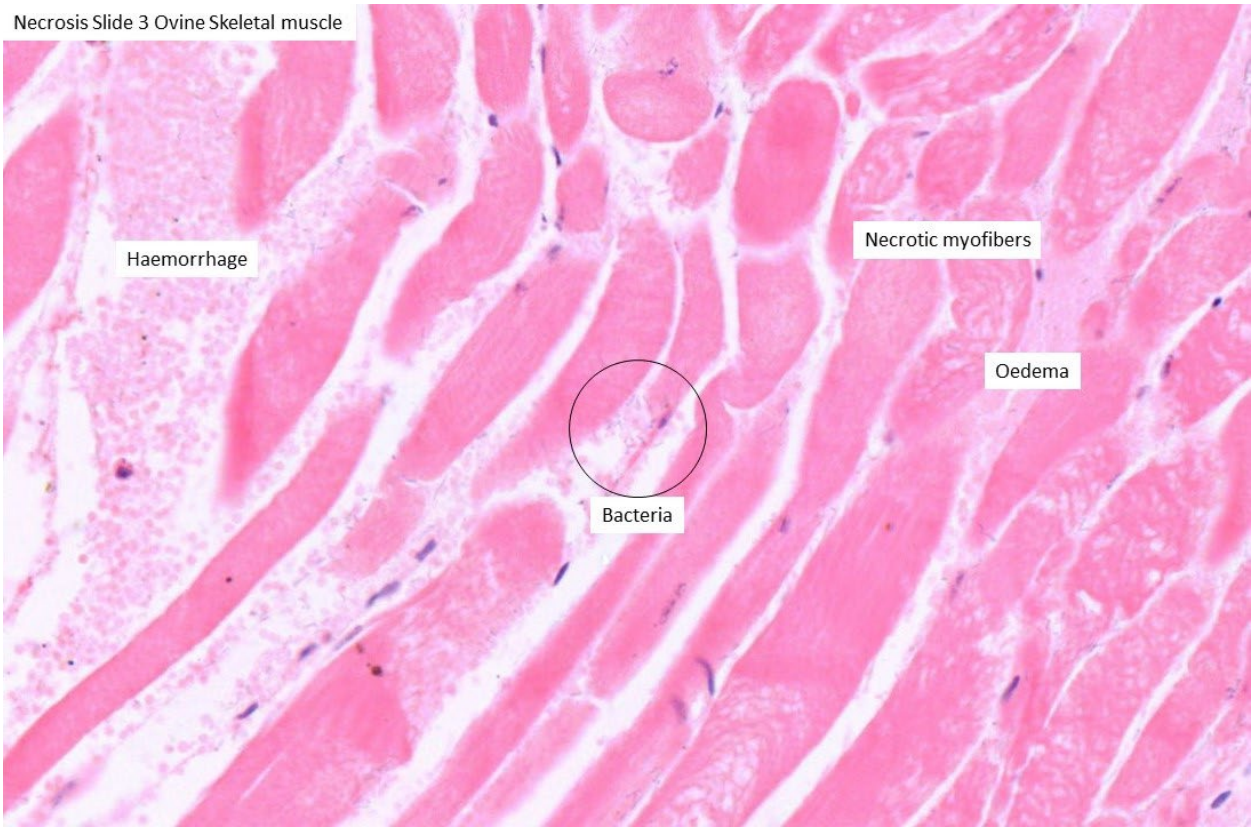

**Necrosis Slide 4 Bovine Adipose tissue**
**Go to** https://slice.edu.au/s/45daa6c5

**What is the normal morphology of adipocytes? Are any of these cells normal adipocytes?**

Normal adipocyte: A round-shaped cell that contain often a single large lipid vacuole surrounded by a layer of cytoplasm. The nucleus is flattened and located on the periphery.

None of the adipocytes present in the slide are normal, they are undergoing coagulative necrosis or are mummified. Nuclei are not identifiable, and the cellular margins are often irregular and poorly defined.

**Describe the histological changes.**

- Wispy to crystallized content or multiple lipid vacuoles of variable size
- Nucleus is not identifiable

**What other types of cells can you recognise in this slide?**

- Fibroblasts
- Multinucleated giant cells
- Macrophages
- Lymphocytes
- Plasma cells

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