Cell Injury: Necrosis and Apoptosis

Answers to Questions

Q1 What are the colourless vacuoles in the cytoplasm of the cells?

A1 The colourless vacuoles in the cytoplasm are the lipid droplets, which have accumulated due to tissue damage. These droplets dissolve during routine processing by organic solvents and appear as sharp-edged vacuoles in paraffin sections.

Q2 Why are they colourless?

A2 The vacuoles are empty spaces and can not take up any stain and therefore appear colourless.

Q3 What do you understand by the term eosinophilia applied to injured cells and how is it brought about?

A3 Eosinophilia is a description of the staining of the tissue by the pink dye eosin (literally liking/loving eosin). Injured cells are eosinophilic because the proteins of their cytoplasm are denatured and the denatured proteins bind eosin more avidly than native protein.

Q4 How can you tell the injury to the liver shown in Section 1.2 was inflicted whilst the rat was still alive (but anaesthetised)? After all, freezing the surface of dead liver might cause changes.

A4 The injury to liver in Section 1.2 must have been caused in life because inflammatory cells, mainly neutrophils are present. The recruitment of these leukocytes could not have occurred post mortem (after death).

Q5 Following tissue injury, what major types of tissue reaction might one expect?

A5 Following tissue injury the following events might be expected, usually in the following order:

1. Death of parenchymal cells by necrosis and/or apoptosis.
2. Formation of inflammatory exudate containing:
   - Fibrin, derived in the tissue from fibrinogen deposited from the blood. Hence fibrinous exudate.
   - Leukocytes, almost all, in the first instance, neutrophils.
3. Recruitment of mononuclear cells from the blood:
   - Monocytes mostly, which differentiate in the tissue into macrophages.
4. Organisation of the dead material involving:
   - Possible regeneration of parenchymal cells
   - Digestion of dead material by macrophages
Migration of endothelial cells and fibroblasts to form new blood vessels to supply nutrients and oxygen (so-called **angiogenesis**). These processes are orchestrated by chemotactic factors and growth factors released from macrophages.

Synthesis of collagen by fibroblasts.

**Q6** How are necrosis and apoptosis different from each other?

**A6** During **apoptosis** (also often referred to as programmed cell death) cascades of enzymes are activated within the cells, thus producing degradation of cellular material, including nucleic acids, proteins etc. Following apoptosis, the apoptotic bodies and even whole cells still undergoing apoptosis, are removed by adjacent phagocytic cells. Thus, apoptosis results in removal of dead cells and their contents with little, if any, spilling of dead material into the extracellular milieu. Hence, there is no stimulus to the activation of inflammation.

**Necrosis**, on the other hand is a largely unregulated process, with cell swelling, calcium ion influx, activation of lysosomal enzymes, loss or reduction of ATP production by mitochondria, membrane injury and eventually membrane rupture leading to release of partially degraded cellular material provoking **inflammation**.