Features	First Intention	Second Intention
1) Cleanness	Clean	Unclean
2) Infection	Generally uninfected	May be infected
3) Margins	Surgically clean:	Irregular
4) Healing	Scanty Granulation tissue	Granulation tissue fill the gap
5) Healing period	Short	Long
6) Healing direction	Direct healing	From the bottom of edge.
7) Sutures	Used	Not used
8) Outcome	Neat & clean scar.	Contracted Irregular wound.
9) Complications	Infrequent, epidermal infusion cyst formation.	Suppuration, may require debridement.

Q5:- Caesarean section, the Gynecologist applied neat surgical sutures to the incision.

A - Trace the steps of healing in this patient in chronological order. (3)

The step of healing in this patient in chronological order:

1 → Formation blood clot.

2 → Formation of granulation tissue.

3 → Cell proliferation & Collagen deposition.

4 → Scar formation.

5 → Wound contraction

6 → Connective tissue remolding.

B. Give the brief account of systemic factors affecting the wound healing. (2)

Grand Unit Test Questions (2019)

Q1(a) What are types of Necrosis? Give examples with each type?

- Fibrinoid Necrosis (Hypertension or antigen-antibody reaction)
- Coagulative Necrosis (All ischemic hypoxic injuries)
- Fat Necrosis (Fatty necrosis by pancreatic lipase)
- Gangrenous Necrosis (Lower limb)
- Liquefactive Necrosis (bacterial infection)
- Caseous Necrosis (Tuberculosis)

(b)

(b) Morphology of Any 2 types with examples

Repeat (cell Injury) Unit test 19

Q2 : Female diabetic, cut her hand. Wound failed to heal after two weeks

(a) Cause of delayed healing

(b) List Four local/systemic factors that Influence wound healing

(c) Trace steps of healing in this pathway...

(a)

Metabolic status: can change wound healing
Circulatory status: Inadequate blood supply)
Infection: most Imp cause

(b)

5 Systemic Factors:
6 • Nutrition • Metabolic status • Circulatory status
• Hormones

Local Factors.
• Infection • Mechanical factors
• Foreign bodies • Size, location & type of wound

(b) Trace steps of Healing
Repeat.

Q3:

Neoplasia

Benign Tumors
- Localized

Malignant tumors
• Metastatic

Q4: Sequence of Events in Acute Inflammation?

Later

Q5: (a)

Four granulometous Diseases

- T.B
- Lepropsy
- Immune reactions against Intestinal bacteria
- Disease by Treponema pallidum.

C- What is atrophy enumerate its causes. (2)

"It is decrease in size of organ or tissue due to reduction in size or no. of cells."

Causes:-

→ Immobilization.

→ Loss of blood supply.

→ Loss of nerve supply.

Q4:- A. What are the causes of cell injury? (2)

1) → Toxins 2) → Infectious agents 3) → Physical trauma
4) → Nutritional Imbalance. 5) → Genetic abnormalities
6) → Immunological reactions 7) → Hypoxia & Ischemia.
8) → Radiation 9) → Chemical

B. Mechanism of irreversible cell injury? (3)

→ Inability to restore mitochondrial function.

→ Loss of structure & function of plasma membrane & intracellular membrane.

→ Loss of DNA & chromatin structure & integrity.

Q5:- 39 years old multigrada female gives birth to a baby.

A. What is type of cellular adaptations change will occur in the uterus? (1)

~~trophy~~
→ Hyper~~plasia~~

B. Describe 4 other cellular adaptations with examples. (4)

* Hypertrophy:-
  → Increase in size of cells resulting in an increase in the size of the organ.
  e.g. Increse in size of uterus in pregnancy
* ~~Hyper~~
* Hyperplasia
  → Inc in number of cells in organs either differentiated cells or less differenciated.
  e.g Female breast at puberty
* Meta plasia
  → It is replacement of one adult cell type with other adult cell type to cope with adverse conditions.
  e.g Ciliated columnar epithelial cells replaced by stratified squemous epithelial cell
* Atrophy
  → Shrinkage in size of cell by loss of cell substance
  e.g Muscular atrophy in paralytic patient.

→Inflammation, Healing & Repair←

Q 1: What are components of Acute Inflammation? Describe steps involved in leucocyte recruitment. (3)

. Acute Inflammation has 3 major components
→ Alteration in vascular caliber that leads to increased blood flow
→ Structural changes in microvasculature that permits leukocytes & protein to leave circulation
→ Emigration of leukocytes from micro-circulation their accumulation in focus of injury & their activation to eliminate offending agent.

* Steps ~~of~~ Involved In Leukocyte recruitment
→ Leukocyte adhesion to endothelium.
→ Leukocyte migration through endothelium.
→ Chemotaxis of leukocytes towards site of injury.

Q: Discuss the outcome of acute inflammation? (2)

1→ Complete resolution:-
Resolution involves removal of cellular debris and microbes by macrophages and resorption of edema fluids by lymphatics

2- Healing by connective tissue replacement :-
Connective tissue grows in area

of damage converting it into a mass of fibrous tissue called organization.

3 → Progression of response to chronic inflammation:-

Acute to chronic transition occcurs when acute inflammatory response cannot be resolved as a result of either persistance of injurious agents or interfere with normal process of healing.

Q2:- b) What are the principal mediators of inflammation? Briefly discuss from where they are derived with examples of each. (3)

Mediators	Sources	Action
1- Histamine	Mast cells, basophils, platelets.	Vasodilation
2- Serotonin	Platelets	Vasoconstriction
3- Prostaglandin	Mast cells, leukocytes	Vasodilation, pain, fever
4- NO (Nitric Oxide)	Endothelium, macrophages	Vascular smooth muscle relaxation, Killing of microbes.
5- Cytokines (TNF, IL-1, IL-6)	Macrophages, Endothelial cells, mast cells.	metabolic abnormalities, hypotension, shock
6- Chemokines	Leukocytes, activated macrophages	Chemotaxis, leukocyte activation

Q4:- Describe the difference b/w healing by primary intention & secondary intention? (5)

Unit # 2019

"Cell Injury"

Q1:- Write down the morphology of necrotic cell?. (1)

* Cytoplasmic Changes:-
  - → Inc.↑ eisonophilia
  - → Glassy Homogenous appearance
  - → Myelien figures are prominent.
  - → Dilatation of mitochondria
  - → Disruption of lysosomes.
* Nuclear Changes:-
  - → Pyknosis (Nuclear shrinkage & inc.↑ Basophilia DNA condensing into dark shrunken mass)
  - → Karyolysis (Digestion of DNA by DNAase)

Q2:- Give account of morphological patterns of 2 types of necrosis?. (4)

* Coagulative necrosis:-

"Underlying tissue architecture is preserved for several days after cell death because of denaturation of not only structural proteins but enzymes therefore blocking proteolysis of cells."

* ~~Liquefactive necrosis~~
* Gangrenous necrosis:-

It refers to condition of limb that has lost blood supply & undergone

coagulative necrosis. When bacterial infection is superimposed, morphological appearance changes to liquefactive necrosis.

Q2:- 60 yrs old, chronic alcoholic, autopsy: liver enlarged, soft & greasy:-

A:- Discuss the pathogenesis of this lesion. (2)

Fatty liver, caused by chronic alcoholism, alcohol causes overproduction of TAGs and their accumulation in liver. Alcohol inhibit fatty acid oxidation ND promotes its deposition.

B:- Microscopic features of this lesion. (3)

Fatty change represents the intracytoplasmic accumulation of triglycerides. At the beginning, the hepatocytes present small fat vacuoles (liposomes) around the nucleus (micro vesicular fatty change). In this stage, liver cells are filled with multiple fat droplets that droplets that do not displace the centrally located nucleus. In the late stages, the size of the vacuoles increases, pushing the nucleus to the periphery of the cell, giving chara

signet ring appearance.

Q3:- Lady has endocervix lined by benign Stratified squamous epithelium with a nabothian cyst and infiltrated with chronic inflammatory cells.

A. What is this phenomenon called? (1)

Metaplasia.

B. Describe mechanism of this change & enumerate one more examples? (2)

Mechanism:- Metaplasia is a reversible change in which one adult cell type is replaced by another cell type. In this type of cellular adaption a cell type sensitive to a particular stress is replaced by another cell type better able to withstand the adverse environment. Metaplasia is thought to arise by reprogramming of stem cells it differentiate along a new pathway rather than a phenotypic change of already differentiated cells.

Other examples:- Epithelial metaplasia is exemplified by the epithelial change in the respiratory tract of cigratte smokers from ciliated columnar to stratified squamous epithelium.

c. what is