1. Basic Human Pathology Lecture #1 Cellular Adaptations to Disease / Cell Injury & Death I

Basic Human Pathology Lecture #1
Cellular Adaptations to Disease / Cell Injury & Death I

2007
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2. Definitions

Definitions

• Pathology
  – ‘The study of disease’
    • Clinical pathology
      – Laboratory procedures
    • Anatomic pathology
      – Structural abnormalities at the cellular and tissue level

• Etiology
  – The cause or causes of any disease.

• Pathogenesis
  – The mechanisms for the development of the disease.
3. Definitions (cont’d)

Definitions (cont’d)

- **Homeostasis**
  - The “steady state” that cells exist in normally
  - An equilibrium of the cells with their environment for adequate function
  - When disturbed there is a predisposal for the onset of pathology

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4. Review: Tissue, Cellular, and Plasma Membrane Anatomy and Te...

Review: Tissue, Cellular, and Plasma Membrane Anatomy and Terms

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5. Definitions

Definitions

- **Relationships exist between cells and with the vasculature**
- **Tissue changes may be from either of the following:**
  - Parenchyma
    - The specific, unique functioning tissue of an organ
  - Stroma
    - The connective tissue framework and blood vessels of an organ
    - Not specific to the organ

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6. Parenchyma and Stroma

Parenchyma and Stroma

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7. Plasma Membrane

8. Reaction of Cells to Stimuli

**Reaction of Cells to Stimuli**

- **Adaptation to Environmental Stress**
  - Cells can adapt to stimuli by either hypofunctioning or hyperfunctioning.
  - A persistent sublethal injury can cause - - - - >
  - **Hypertrophy**
    - Increase in the size of an organ or tissue due to an increase in the size of the cells
    - Ex. - work hypertrophy of muscle
  - **Hyperplasia**
    - Increase in the size of an organ or tissue caused by an increase in the number of cells
    - Ex. – glandular proliferation in the breast during pregnancy
    - Can occur with hypertrophy
    - Ex. – uterine enlargement during pregnancy

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9. Hypertrophy - Masseter Muscle

Hypertrophy – Masseter Muscle

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10. Hypertrophy

Hypertrophy

Source: TUSDM

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11. Hypertrophy – Skeletal Muscle

**Hypertrophy – Skeletal Muscle**

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12. Left Ventricular Wall Hypertrophy of Cardiac Muscle –...

**Left Ventricular Wall Hypertrophy of Cardiac Muscle –**

Macroscopic and microscopic

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13. Hypertrophy – Cardiac Muscle

Hypertrophy – Cardiac Muscle

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Normal  Hypertrophy

14. Nodular Hyperplasia – Prostate

Nodular Hyperplasia – Prostate

Macroscopic

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15. Nodular Hyperplasia – Prostate

Nodular Hyperplasia – Prostate

Microscopic

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16. Hypertrophy and Hyperplasia

Hypertrophy and Hyperplasia

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Hypertrophy and Hyperplasia During Pregnancy

Uterine myometrium - macroscopic

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Physiological Hyperplasia –

Endometrium of Uterus During Menstruation

Macroscopic

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19. Physiological Hyperplasia – Endometrium of Uterus

Physiological Hyperplasia – Endometrium of Uterus

Microscopic

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20. Developmental causes of reduced cell mass

Developmental causes of reduced cell mass

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21. Adaptation to Environmental Stress (cont’d) –

Adaptation to Environmental Stress (cont’d) –
Developmental Causes of Reduced Cell Mass

❖ Agenesis
   • Failure of formation of embryonic cell mass (anlage)

❖ Aplasia
   • Failure of differentiation to organ specific tissues
     • Ex. - kidney
   • Failure of cell production
   • During fetal development aplasia results in agenesis
   • Later in life, aplasia can be caused by permanent loss of precursor cells in proliferative tissues such as bone marrow

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22. Adaptation to Environmental Stress (cont’d) –

Adaptation to Environmental Stress (cont’d) –
Developmental Causes of Reduced Cell Mass

❖ Dysgenesis
   • Failure to undergo structural organization of tissues into an organ

❖ Hypoplasia
   • Decrease in cell production that is less extreme than that found in aplasia – failure of growth to full size
     • Ex. – Turner syndrome and Klinefelter syndrome
     • Partial lack of growth and maturation of gonadal structures

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23. Adaptation to Environmental Stress (cont’d)

Adaptation to Environmental Stress (cont’d)

❖ Atrophy

• Decrease in the size of an organ or tissue resulting from a decrease in the mass of pre-existing cells
• Results most often from disuse, nutritional or oxygen deprivation, diminished endocrine stimulation, aging, and denervation
• Often marked by presence of autophagic granules
  – Intracytoplasmic vacuoles containing debris from degraded organelles

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24. Autophagy and Cell Atrophy

Autophagy and Cell Atrophy

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25. Adaptation to Environmental Stress (cont’d)

Adaptation to Environmental Stress (cont’d)

- General atrophy - involves widespread atrophy of numerous tissues
  - Starvation atrophy
  - Senile atrophy
    - Reduced activity leads to reduction in size of the skeletal muscle fibers

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26. Adaptation to Environmental Stress

Adaptation to Environmental Stress

- Local atrophy
  - Disuse atrophy
    - From inactivity of an organ or part
    - Ex. - an arm in a cast results in loss of muscle due to lack of use
  - Pressure atrophy
    - From prolonged pressure on a local area
    - Ex. - bed ulcers; atrophy of the submandibular gland
  - Endocrine atrophy
    - From deprivation of hormonal stimulation
    - Ex. - lactating breast and uterus after menopause
  - Denervation atrophy
    - Ex. - damage to axons supply muscle; lack of stimulation

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27. Local Atrophy - Disuse

Local Atrophy - Disuse

Source: TUSDM

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28. Atrophy - Endocrine

Atrophy - Endocrine

Normal adrenal

Adrenal atrophy – ACTH ↓

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29. **Atrophy**

### Localized
- Cerebral atrophy in Alzheimer’s disease

### Generalized
- Senile Atrophy

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30. **Hemifacial Atrophy**

### Hemifacial Atrophy

*Source: TUSDM*

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31. Adaptation to Environmental Stress (cont’d)

Adaptation to Environmental Stress (cont’d)

- **Involution**
  - Physiological decrease in the number of cells to their normal number
  - Ex. – thymus gland involutes during adolescence
  - Ex. – myometrium involutes during post partum

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32. Involution – Thymus Gland

Involution – Thymus Gland

Childhood  Adult

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Adaptation to Environmental Stress (cont’d)

**Metaplasia**

- Replacement of one differentiated tissue by another in a hostile environment

**Squamous metaplasia**

- Ex. - Change from columnar ciliated epithelium to squamous epithelium at the squamocolumnar junction of the cervix
- Associated with chronic irritation (e.g., bronchi with long term use of tobacco); vitamin A deficiency
- Often reversible

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Adaptation to Environmental Stress (cont’d)

**Metaplasia – cont’d**

- Osseous (cartilaginous) metaplasia
  - Formation of new bone (cartilage) at sites of tissue injury such as ill fitting dentures
- Myeloid metaplasia (extramedullary hematopoiesis)
  - Proliferation of hematopoietic tissue in sites other than the bone marrow, such as the liver or spleen
  - --> hepatosplenomegaly such as during sickle cell anemia

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35. Cellular Adaptations to Disease/Cell Injury and Death I: Slit...

<table>
<thead>
<tr>
<th>Original Tissue</th>
<th>Stimulus</th>
<th>Metaplastic Tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ciliated columnar epithelium of bronchial tree</td>
<td>Cigarette smoke</td>
<td>Squamous epithelium</td>
</tr>
<tr>
<td>Transitional epithelium of bladder</td>
<td>Trauma of bladder calculus</td>
<td>Squamous epithelium</td>
</tr>
<tr>
<td>Columnar epithelium in gland ducts</td>
<td>Trauma of calculus</td>
<td>Squamous epithelium</td>
</tr>
<tr>
<td>Fibrocollagenous tissue</td>
<td>Chronic trauma</td>
<td>Bone (osseous) tissue</td>
</tr>
<tr>
<td>Oesophageal squamous epithelium</td>
<td>Gastric acid</td>
<td>Columnar epithelium</td>
</tr>
<tr>
<td>Columnar glandular epithelium</td>
<td>Vitamin A deficiency</td>
<td>Squamous epithelium</td>
</tr>
</tbody>
</table>

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36. Respiratory Epithelium Prior to Metaplasia

Respiratory Epithelium Prior to Metaplasia

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37. Cellular Adaptations to Disease/Cell Injury and Death I: Sli...

38. Esophageal Mucosa Metaplastic to Stratified Squamous Epithel...

Esophageal Mucosa Metaplastic to Stratified Squamous Epithelium

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39. Ductal – Squamous Metaplasia

![Ductal – Squamous Metaplasia](image)

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40. Squamous Metaplasia

![Squamous Metaplasia](image)

Source: TUSD

Bladder - transitional epithelium to squamous epithelium

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41. Summary of Adaptation to Environmental Stress

Summary of Adaptation to Environmental Stress

<table>
<thead>
<tr>
<th>Change in size of cells</th>
<th>Reduction in the size of cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrophy</td>
<td>Increase in the size of cells</td>
</tr>
<tr>
<td>Hypertrophy</td>
<td>Decrease in the number of cells</td>
</tr>
<tr>
<td>Change in number of cells</td>
<td>Increase in the number of cells</td>
</tr>
<tr>
<td>Involution</td>
<td>Stable change to another cell type</td>
</tr>
<tr>
<td>Hyperplasia</td>
<td></td>
</tr>
<tr>
<td>Change in differentiation of cells</td>
<td></td>
</tr>
<tr>
<td>Metaplasia</td>
<td></td>
</tr>
</tbody>
</table>

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42. Summary

Summary

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43. Cell Adaptation Key Facts

Cell Adaptation Key Facts

- Adaptable within physiological limits
- **Heat shock proteins** - can respond to injury by producing cell stress proteins, which protect from damage and help in recovery
- Increased demands met by hypertrophy and hyperplasia
- Reduced demand met by atrophy
- **Apoptosis** – cell loss from tissues can be achieved by programmed cell death
- Tissues can adapt to demand by a change in differentiation known as **metaplasia**

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44. Reaction of Cells to Injury

Reaction of Cells to Injury

- **Reversible injury (degeneration)**
  - Cell functions impaired but cell can recover
- **Irreversible injury**
  - Cessation of all cell functions with cellular death
  - **Apoptosis**
    - Programmed cell death
  - **Necrosis**
    - Sum of the degradative and inflammatory reactions occurring after tissue death

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45. Reaction of Cells to Injury on a Biochemical Level

Reaction of Cells to Injury on a Biochemical Level

- **Functional** (biochemical) changes occur before gross morphologic changes appear
- **Ultrastructural** changes occur before light microscopic changes appear
- **Light microscopic** changes occur before gross morphologic changes appear

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46. Reaction of Cells to Injury on a Biochemical Level

Reaction of Cells to Injury on a Biochemical Level

- **Ubiquitin**
  - Marks abnormal proteins for degradation
  - Ex. - heat shock proteins induced by stress
- **Chaperones**
  - Specialized protein
  - Required for proper folding and/or assembly of another protein or protein complex

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47. Function of Ubiquitin in Cell Stress

Function of Ubiquitin in Cell Stress

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48. Protein Kinesis

Protein Kinesis

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49. Reaction of Cells to Injury on a Biochemical Level

Reaction of Cells to Injury on a Biochemical Level

• Disorders characterized by protein folding abnormalities
  - Two known pathogenetic mechanisms
    - Abnormal protein aggregation, examples:
      - Amyloidosis
      - Neurodegenerative diseases
        - (e.g., Alzheimer’s, Huntington’s disease, Parkinson’s, Prion disease)
    - Abnormal protein transport and secretion, examples:
      - Cystic fibrosis
      - Alpha 1-antitrypsin deficiency

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50. Reaction of Cells to Injury on a Biochemical Level

Reaction of Cells to Injury on a Biochemical Level

• Biochemical derangements
  - Oxygen-derived free radicals affect cell structure
  - ATP depletion
    - Needed for energy of all cell functions
  - Loss of calcium homeostasis
    - Calcium enters via membranes and also increases within the cell (cytosolic calcium)
    - Calcium activates enzymes capable of degrading cell membranes
  - Defects in membrane permeability
    - Sodium plus other accumulations change the osmotic balance
      - Water enters cells
      - Cloudy swelling

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51. Free Calcium’s Role as a Destructive Agent

52. Reversible Cellular Changes and Accumulations

- Hydropic degeneration (hydropic change)
  - Only the cytoplasm is involved
  - Water accumulates and the cell swells
    - Large vacuoles in the cytoplasm
  - Light microscopy
    - Cytoplasm is pink and granular
  - Electron microscopy (ultrastructural)
    - Organelles are swollen
    - Ribosomes displaced
    - Lysosomal activity very apparent
53. Hydropic Change

Hydropic Change

Source: TUSDM

Kidney - microscopic

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54. Hydropic Change - Leukoedema

Hydropic Change - Leukoedema

Source: TUSDM

Oral epithelium - microscopic

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55.

Hydropic Degeneration - Leukoedema

Oral epithelium - macroscopic

Source: TUSDM

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56.

Reversible Change – Cell Degeneration

Basal Cell Layer of epithelium

Source: TUSDM

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57. Reversible Cellular Changes and Accumulations

Reversible Cellular Changes and Accumulations

- Fatty change (steatosis, fatty metamorphosis)
  - Characterized by accumulation of intracellular parenchymal triglycerides; nucleus is displaced and the cell swells
  - Observed frequently in liver, heart, and kidney
    - Ex. – in liver secondary to alcoholism, diabetes mellitus, malnutrition, obesity, poisoning
- Results from imbalance among the uptake, utilization, and secretion of fat
  - Increased transport of triglycerides (fatty acids) to affected cells
  - Decreased mobilization of fat from cells
    - Most often due to decreased production of apoproteins for fat transport
  - Decreased use of fat by cells
  - Overproduction of fat in cells

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58. Fatty Change

Fatty Change

Liver microscopic

Source: TUSD

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59. Fatty Change

Fatty Change

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Normal / Fatty change

Source: TUSDM

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60. Fatty Change - Liver

Fatty Change - Liver

Source: TUSDM

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61. Fatty Change - Liver

62. Reversible Cellular Changes and Accumulations

- **Hyaline change**
  - Homogeneous, glassy, eosinophilic appearance in H&E stained tissue sections
  - Caused most often by nonspecific accumulations of proteinaceous material
  - Ex. - glomeruli tufts in diabetic glomerulosclerosis
63. Reversible Cellular Changes and Accumulations

Reversible Cellular Changes and Accumulations

- Accumulation of exogenous pigments
  - Naturally colored substances not requiring tissue stain to be seen
    - Pulmonary accumulations of carbon, silica, and iron dust
    - Plumbism (lead poisoning)
    - Algeria (silver poisoning)
      - May cause a permanent gray discoloration of the skin and conjunctiva

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64. Hyaline Change – Liver Cell

Hyaline Change – Liver Cell

Mallory Bodies

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Reversible Cellular Changes and Accumulations

- Accumulation of endogenous pigments
  - Melanin
    - Most common; brown pigment
    - Formed from tyrosine via tyrosinase
    - Synthesized in melanosomes of melanocytes within the basement membrane of the epidermis and choroid of the eye
    - Transferred by melanocytes to adjacent clusters of keratinocytes and macrophages (melanophores) in the subjacent dermis

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Reversible Cellular Changes and Accumulations

- Accumulation of endogenous pigments
  - Melanin
    - Seen also in neoplasms
      - Ex. - melanocytic nevus, melanotic macule
      - Ex. - melanoma

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67. Labial Melanotic Macule – Focal Melanosis

Labial Melanotic Macule – Focal Melanosis

Source: TUSDM

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68. Melanin Pigmentation – Labial Melanotic Macule

Melanin Pigmentation – Labial Melanotic Macule

Source: TUSDM

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Reversible Cellular Changes and Accumulations

**Bilirubin**
- Catabolic product of the heme moiety of hemoglobin and myoglobin
  - In pathologic conditions, accumulates and stains the blood, sciera, mucosa, and internal organs producing a yellow discoloration (jaundice)
    - **Hemolytic jaundice**
      - Destruction of red blood cells
    - **Obstructive jaundice**
      - Intra- or extrahepatic obstruction of the biliary tract
    - **Hepatocellular jaundice**
      - Parenchymal liver damage

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Reversible Cellular Changes and Accumulations

**Hemosiderin**
- Iron-containing pigment; aggregates of ferritin
- In tissue appears as golden-brown amorphous aggregates
  - **Prussian blue dye – positive blue color stain reaction**
- Exists normally in small amounts as physiologic iron stores within tissue macrophages of the bone marrow, liver, and spleen

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71. Hemosiderin in Liver Cells

Hemosiderin in Liver Cells

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72. Hemosiderin – Lung Alveoli

Hemosiderin – Lung Alveoli

Source: TUSDPM

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73. Reversible Cellular Changes and Accumulations

Reversible Cellular Changes and Accumulations

- **Hemosiderin**
  - **Found in**
    - Week-old hemorrhage
    - Hemolysis
    - Inborn errors of metabolism affecting transport and absorption as in the liver and pancreas
    - Accumulates pathologically in tissue in excess amounts (sometimes massive)
      - Hemosiderosis vs. hemochromatosis

74. Reversible Cellular Changes and Accumulations

Reversible Cellular Changes and Accumulations

- **Hemosiderosis**
  - Accumulation of hemosiderin, primarily within tissue macrophages, without associated tissue or organ damage
    - **Local** - most often from hemorrhage into tissue; derived from breakdown of hemoglobin
    - **Systemic** - generalized; from hemorrhage, multiple blood transfusions, hemolysis, excessive dietary intake; often accompanied by alcohol consumption
Reversible Cellular Changes and Accumulations

Hemochromatosis

- Extensive accumulation of hemosiderin, often within parenchymal cells, with accompanying tissue damage, scarring, and organ dysfunction
- Hereditary type (primary)
  - Most often caused by mutation of Hfe gene, chromosome #6
  - Characterized by liver, pancreas, myocardium, and multiple endocrine glands damage; melanin deposition in skin
  - Triad – micronodular cirrhosis, diabetes mellitus, “bronze diabetes”
  - Elevated serum iron, decreased total iron-binding capacity

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Reversible Cellular Changes and Accumulations

Hemochromatosis – cont’d

Secondary type

- Most often caused by multiple blood transfusions for conditions such as Beta-thalassemia major (a hereditary hemolytic anemia)

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77. Hemochromatosis - Pancreas

Hemochromatosis - Pancreas

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78. Reversible Cellular Changes and Accumulations

Reversible Cellular Changes and Accumulations

❖ Lipofuscin

- Yellowish to light brown, fat-soluble pigment; end product of membrane lipid peroxidation
- “Wear and tear” pigment
- Commonly accumulates in elderly patients
  - Found most often within hepatocytes and at the poles of nuclei of myocardial cells

❖ Brown atrophy

- Accumulation of lipofuscin and atrophy of organs

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79. Lipofuscin – Striated Muscle and Liver

Lipofuscin – Striated Muscle and Liver

Source: TUSDM

Microscopic

Cardiac muscle

Liver

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80. Reversible Cellular Changes and Accumulations

Reversible Cellular Changes and Accumulations

• Pathologic calcifications
  – Abnormal deposition of calcium salts in soft tissue
  – Deep blue-purple in nondecalcified H&E stained tissue
  – May stimulate further bone deposition

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81. Pathologic Calcifications

Pathologic Calcifications

- **Metastatic calcifications**
  - **Caused by hypercalcemia**
    - Most often from hyperparathyroidism
    - Osteolytic tumors with mobilization of Ca\(^{2+}\) and PO\(_4^-\)
    - Hypervitaminosis D
    - Excess calcium intake
      - E.g., milk-alkali syndrome – nephrocalcinosis, renal stones caused by milk and antacid self-therapy for peptic ulcer

82. Metastatic Calcification Hypercalcemia - Lung

Metastatic Calcification
Hypercalcemia - Lung

Source: TUSDM
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83. Pathologic Calcifications

Pathologic Calcifications

❖ Dystrophic calcifications
  • Intracellular or extracellular; gritty
  • Deposition of calcium in tissue altered by injury
    – Areas of old trauma
    – Tuberculosis lesions
    – Affects crucial organs, heart valves, vessels
      » Scarred heart valves
      » Atherosclerosis
  ❖ Not caused by hypercalcemia but calcium attracted by released membrane phosphates
    – Serum calcium concentration normal

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84. Calcific Stenosis of Aortic Valve

Calcific Stenosis of Aortic Valve

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85. Dystrophic Calcification – Medial Sclerosis

86. Dystrophic Calcification – Medial Sclerosis
87. Dystrophic Calcification – Stomach Injury

Dystrophic Calcification – Stomach Injury

Source: TUSDM

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88. Dystrophic Calcification – Periapical and Pulpal Pathoses

Dystrophic Calcification – Periapical and Pulpal Pathoses

Source: TUSDM

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