SAWA UNIVERSITY

HISTOPATHOLOGY
1
PHYSICAL AND CHEMICAL INJURIES

Sawa University — جامعـــــة ســـــــاوة الاهلية

College of health and medical techniques — كليـــــة التقنيـــات الصحيـة والطبيـة

Department of Medical Laboratories — قسم تقنيات المختبرات الطبية

Third Stage — المرحلـــــــــــة الثالثة

الجانب النظري — محاضرة رقم 10

Theoretical — Lecture No. 10

تدريسي المادة : م.د. قاسم عباس محمد.

OBJECTIVE

- Identify the major types of physical and chemical injuries.
- Describe the histopathological changes associated with burns, radiation, toxins, and mechanical trauma.
- Recognize clinical correlations and complications resulting from exposure to harmful physical and chemical factors.

Introduction

- Physical and chemical injuries represent a group of non-infectious, non-immune, non-genetic causes of tissue damage.
- They directly harm cells via: Membrane disruption, Protein denaturation, DNA damage, Oxidative stress, Ischemia.
- These injuries vary in severity depending on:
  - Dose / intensity
  - Duration of exposure
  - Tissue vulnerability
  - Presence of underlying diseases

Classification of Physical and Chemical Injuries

A. Physical Injuries :

1.Mechanical trauma

2. Thermal injuries (Heat & Cold)

3.Electric injuries

4. Radiation injury

5.Barotrauma

6. Pressure-related injuries

7.Noise injury

B. Chemical Injuries :

1.Caustic acids and alkalis

2. Heavy metals

3.Alcohol

3. Drugs (therapeutic & illicit)

5.Environmental toxins

6. Pesticides & industrial chemicals

A. Physical Injuries :

1. Mechanical Trauma

Examples : Abrasion, Contusion, Laceration, Incised wound, Puncture wound, Crush injury, Fractures

Histopathology

- **Disruption of tissue architecture**
- **Hemorrhage (extravasation of RBCs)**
- **Interstitial edema**
- **Neutrophilic infiltration after 4–24 hours**
- **Fibroblast proliferation during healing**

2. Thermal Injuries

A. Burns (Heat Injury): Degree of Burn

1.First Degree (Superficial) : Epidermis only, Redness, no blisters

Histopathology: vascular dilatation, mild edema

2.Second Degree (Partial Thickness). Involves epidermis + part of dermis, Blister formation

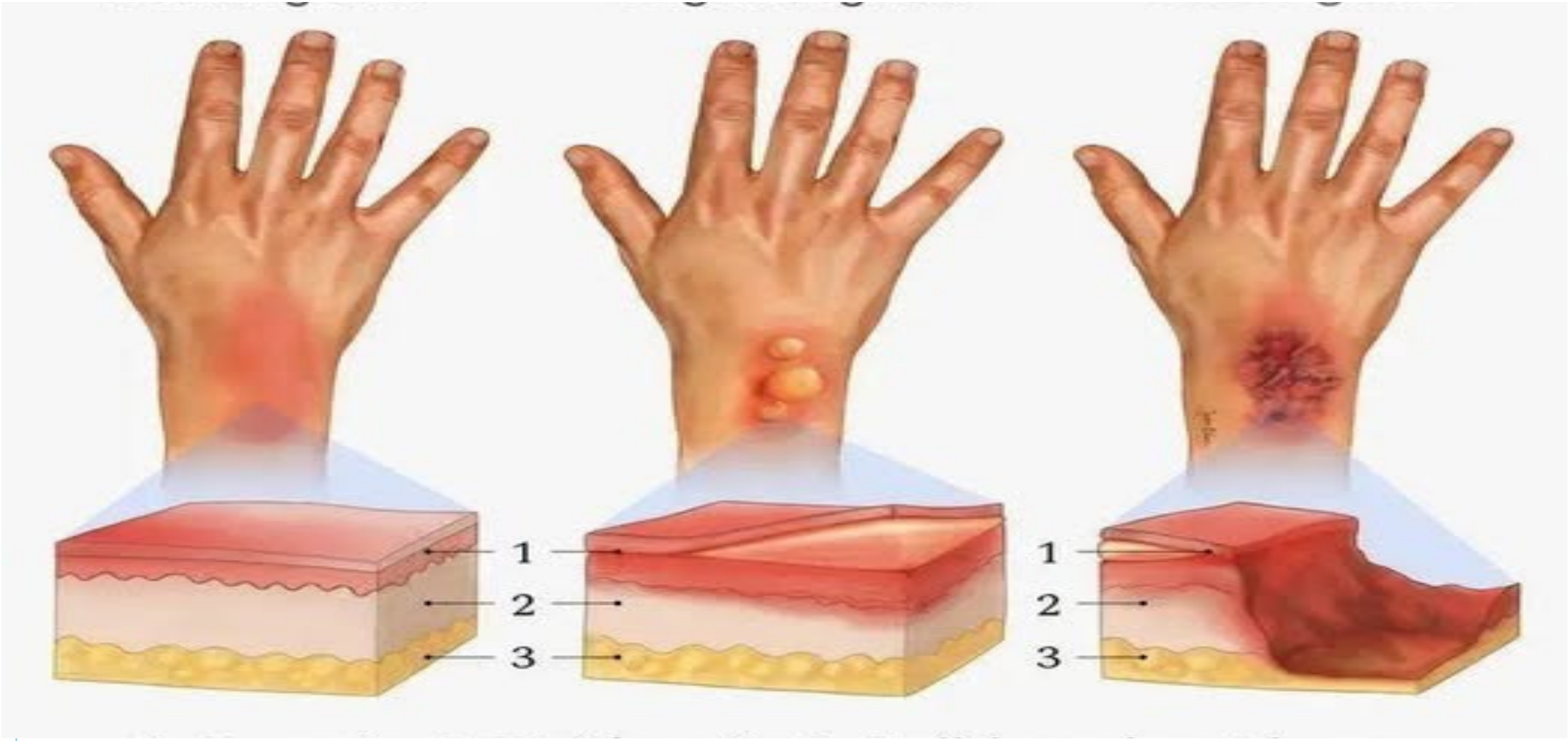
Histopathology: separation between epidermis/dermis, edema, acute inflammation

3.Third Degree (Full Thickness): All layers destroyed, Skin becomes dry, leathery

Histopathology: coagulative necrosis, loss of adnexal structures

4.Fourth Degree: Extends to muscle and bone

Systemic Effects: Hypovolemic shock, Sepsis, Hyperkalemia, Myoglobinuria



1. Epidermis **2. Dermis** **3. Hypodermis**

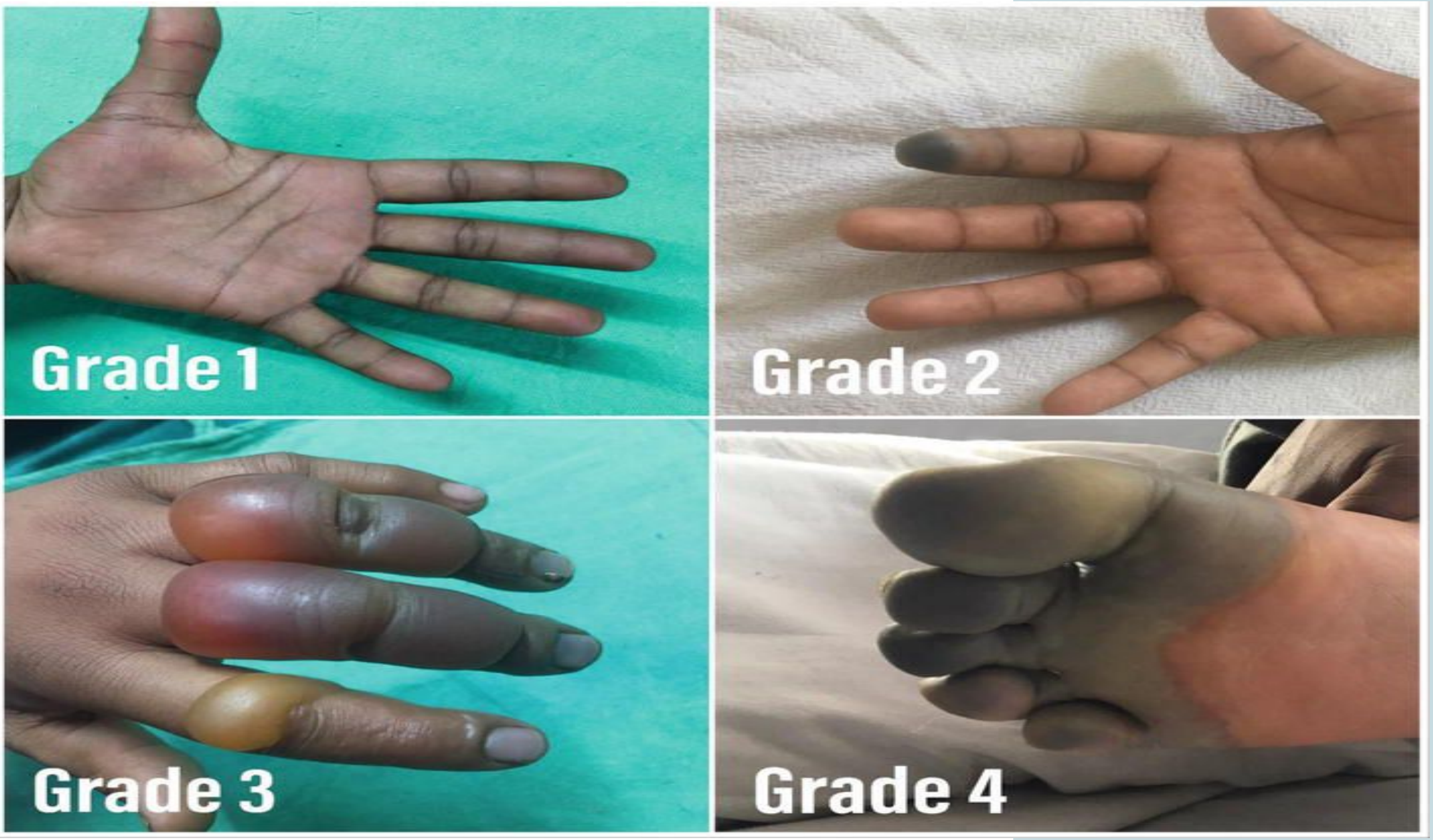
B. Cold Injury (Frostbite)

Mechanism

- Ice crystal formation → direct cell rupture
- Vascular stasis → ischemia
- Reperfusion injury

Histopathology

- **Endothelial swelling**
- **Thrombosis of small vessels**
- **Coagulative necrosis of skin and digits**


Grade 1
Grade 2
Grade 3
Grade 4

3. Electrical Injuries

Mechanism: Heat generation, Disruption of nervous system, Ventricular fibrillation

Histopathology

- Coagulative necrosis of skin
- Charred tissue
- Thrombosed vessels
- Sometimes minimal external injury with severe internal burns

4. Radiation Injury

Types: A. Ionizing radiation: X-rays, γ-rays B. Non-ionizing: UV radiation

Mechanism: DNA breaks, Free radicals → oxidative stress, Cell cycle arrest and apoptosis

A. UV Radiation

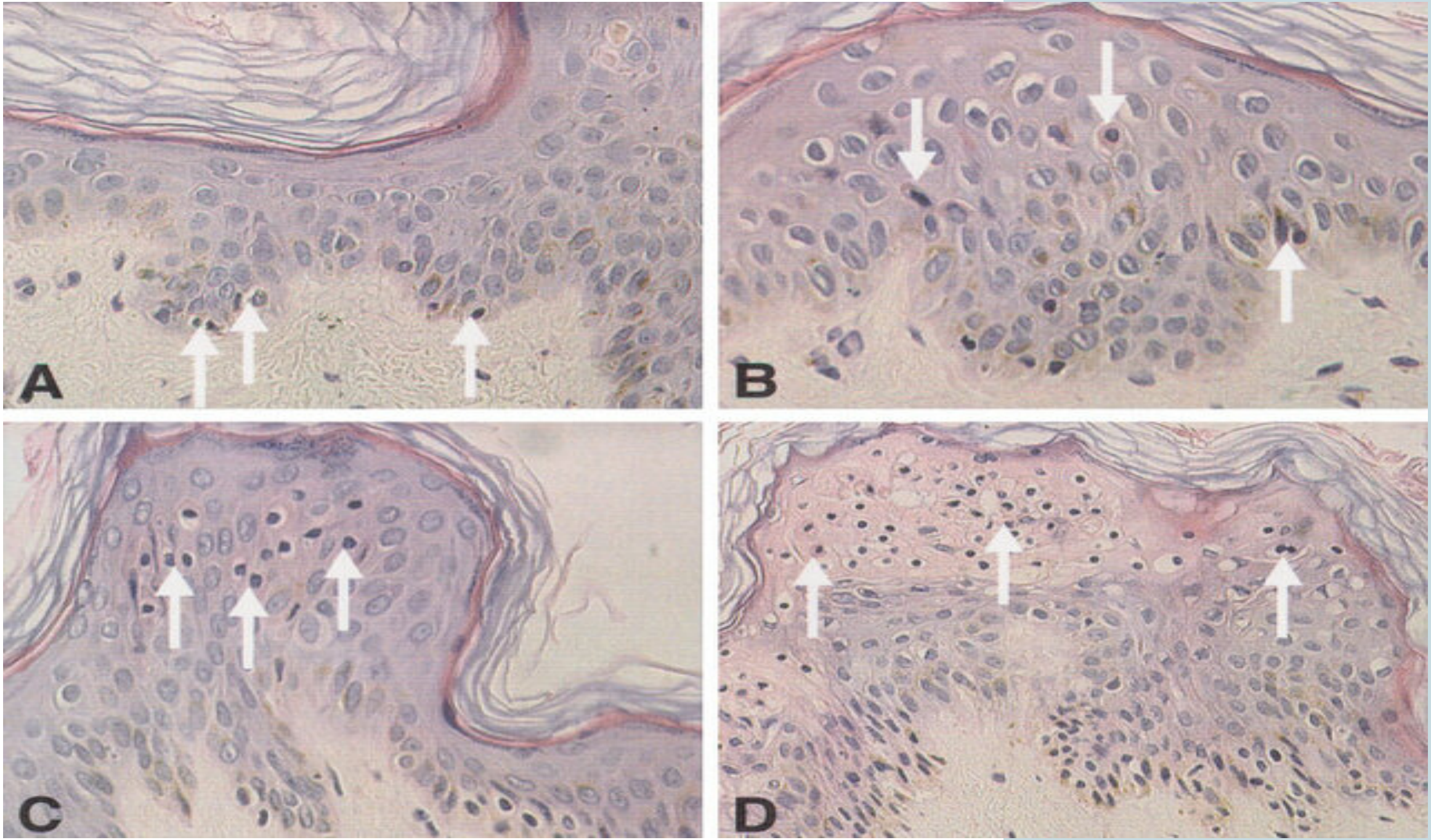
Histopathology: Nuclear enlargement (sunburn cells), Dysplasia in chronic exposure, Elastosis of dermis, Increased melanocytes.

B. Ionizing Radiation: Acute Radiation Injury

Histopathology: Mitotic arrest, Cellular swelling, Apoptosis (especially in lymphocytes)

Tissues Most Sensitive: Bone marrow, GI lining, Gonads, Skin, Lymphoid tissues

Pathologic Features: Atypical mitoses, Chromosomal fragmentation, Atrophy of target organs, Fibrosis in chronic exposure


A
B
C
D

B. Chemical Injuries

1. Acids and Alkalis

Acids: Coagulative necrosis, Eschar formation

Alkalis: Liquefactive necrosis, Deeper penetration, Severe esophageal damage

Histopathology: Necrosis, Inflammatory infiltration, Sloughing of epithelium, Ulceration

2. Heavy Metals

Examples: Lead, Mercury, Arsenic, Cadmium

Histopathology:

Lead: Basophilic stippling in RBCs, Renal proximal tubular injury

Mercury: Nephrotic damage, CNS neuron shrinkage

Arsenic: Skin hyperkeratosis, Squamous cell carcinoma risk

3. Alcohol (Ethanol)

Acute effects: CNS depression, Hepatic steatosis

Chronic effects: Cirrhosis, Cardiomyopathy, Pancreatitis

Histopathology: Fatty liver (macrovesicular steatosis), Ballooning hepatocytes, Mallory bodies, Fibrosis

4. Drugs

Both prescribed and illicit drugs can be toxic:

A. Hepatotoxicity: Centrilobular necrosis (e.g., acetaminophen), Cholestasis, Steatosis, Granulomas
B. Nephrotoxicity: Acute tubular necrosis, Interstitial nephritis
C. Cardiotoxicity: Fibrosis (e.g., anthracyclines)

5. Pesticides & Toxins

Organophosphates

- Inhibit acetylcholinesterase
- Cause bronchoconstriction, paralysis

Histopathology: Non-specific necrosis, Pulmonary edema, Myocardial fatty change

Oxidative Stress as a Common Mechanism

Chemical and physical injuries often share a common pathway:

Reactive Oxygen Species (ROS)

- Lipid peroxidation
- Protein crosslinking
- DNA fragmentation

Histo appearance

- Cytoplasmic eosinophilia
- Pyknosis → karyorrhexis → karyolysis
- Cell membrane blebbing

References

1. Kumar, V., Abbas, A. K., & Aster, J. C. (2023). Robbins & Cotran Pathologic Basis of Disease (11th ed.). Elsevier.

2. Mitchell, R. N. (2023). Basic Pathology. Elsevier.

Any questions?