# General Pathology

**م.د. أحمد تركي هاني**
**MBChB. MSc. PhD.**

# Introduction & Subspecialties

م.د. أحمد تركي هاني
MBChB. MSc. PhD.

## Pathology

Study of disease: **Etiology** (Cause), **Pathogenesis** (Mechanism), **Morphology** (Structural Change), and **Clinical Manifestations**.

## Clinical Pathology

Analysis of **body fluids** (blood, urine) and tissues using lab methods (chemistry, hematology, microbiology).

## Molecular Pathology

Study of disease at the **molecular level** (DNA, RNA, proteins) for diagnosis and targeted therapy (e.g., cancer genetics).

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# Cell Response to Injury

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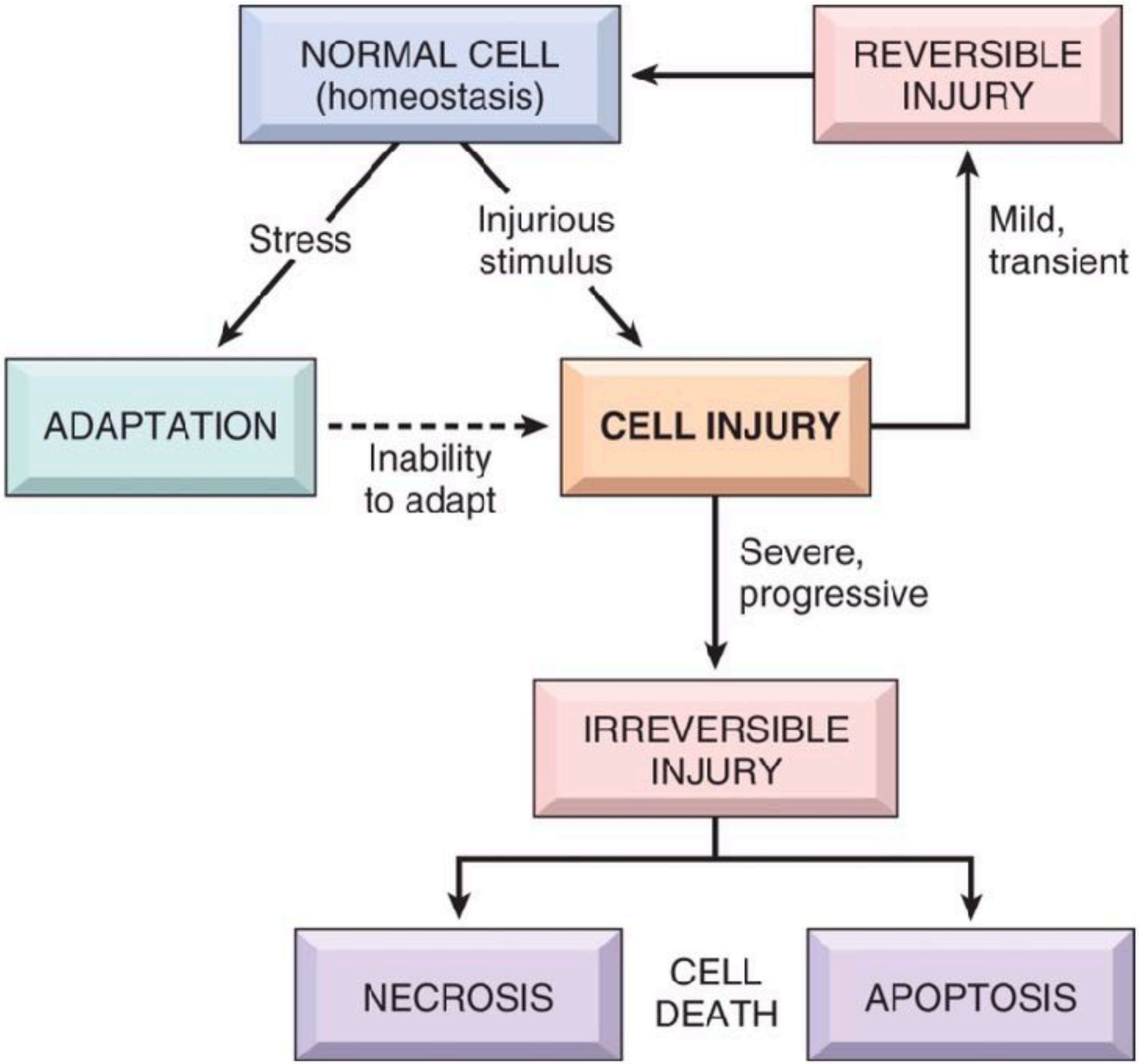
**MBChB. MSc. PhD.**

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# Cell Injury Overview

Cell damage occurs when cells are exposed to harmful stimuli.

It can be **reversible** or **irreversible** depending on the severity and duration of the insult.

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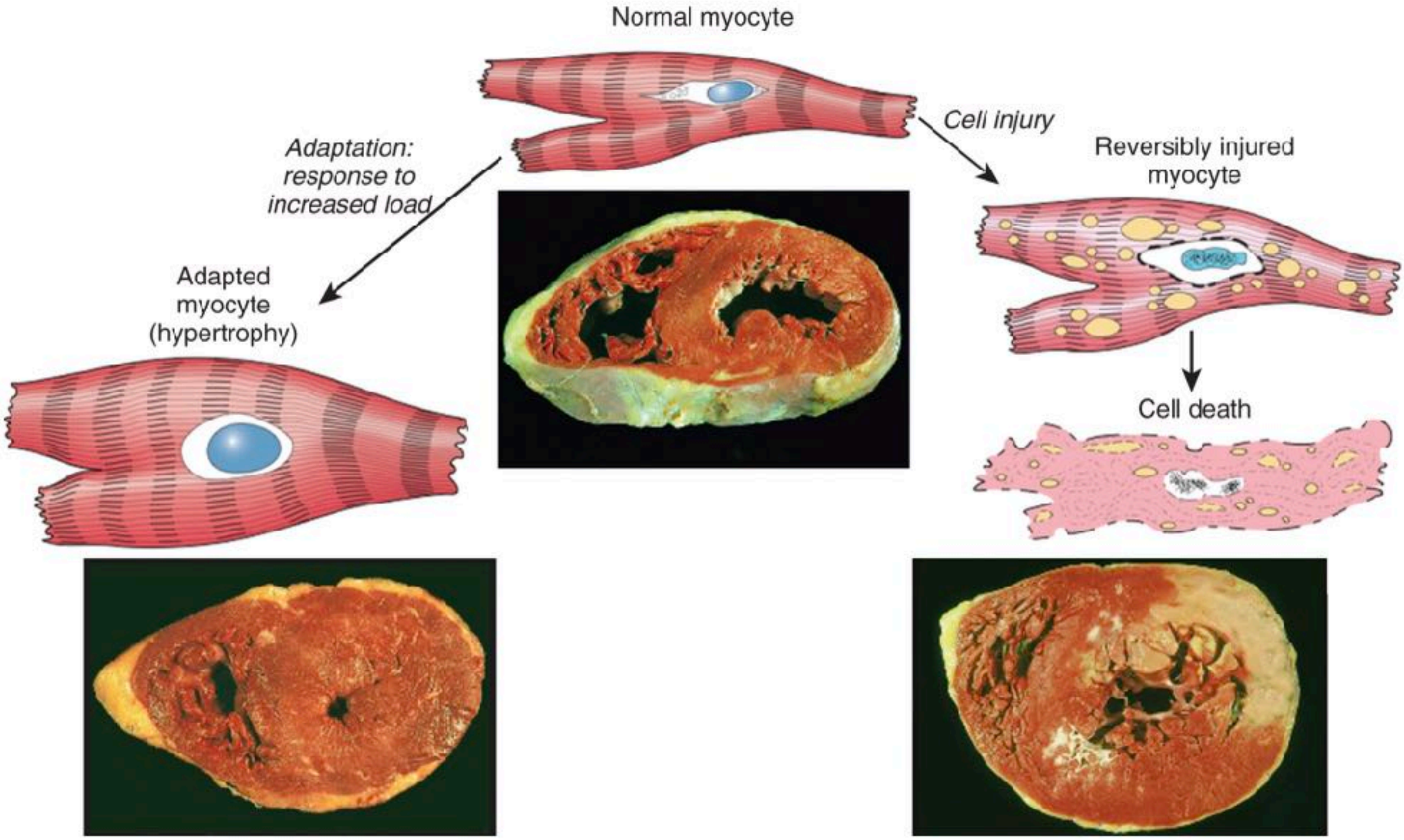


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# Reversible Cell Injury

This is a **mild or short-term injury** where cells can recover if the stressor is removed.

**Causes:**

- Hypoxia (low oxygen)
- Physical agents (heat, cold)
- Chemicals or toxins
- Infections

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# Reversible Cell Injury

**Features:**

- **Cell swelling** (due to failure of ion pumps)
- **Fatty change** (especially in liver cells)
- Membrane **blebbing**
- Loss of **microvilli**
- Ribosome **detachment**

**Reversibility**: If the damaging stimulus is removed, normal structure and function can return.

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# Reversible Cell Injury

Mild/short-lived stress. **Cell can recover** if stimulus is removed.

**Cellular Swelling** (Hydropic Change, due to pump failure and influx). **Fatty Change**.

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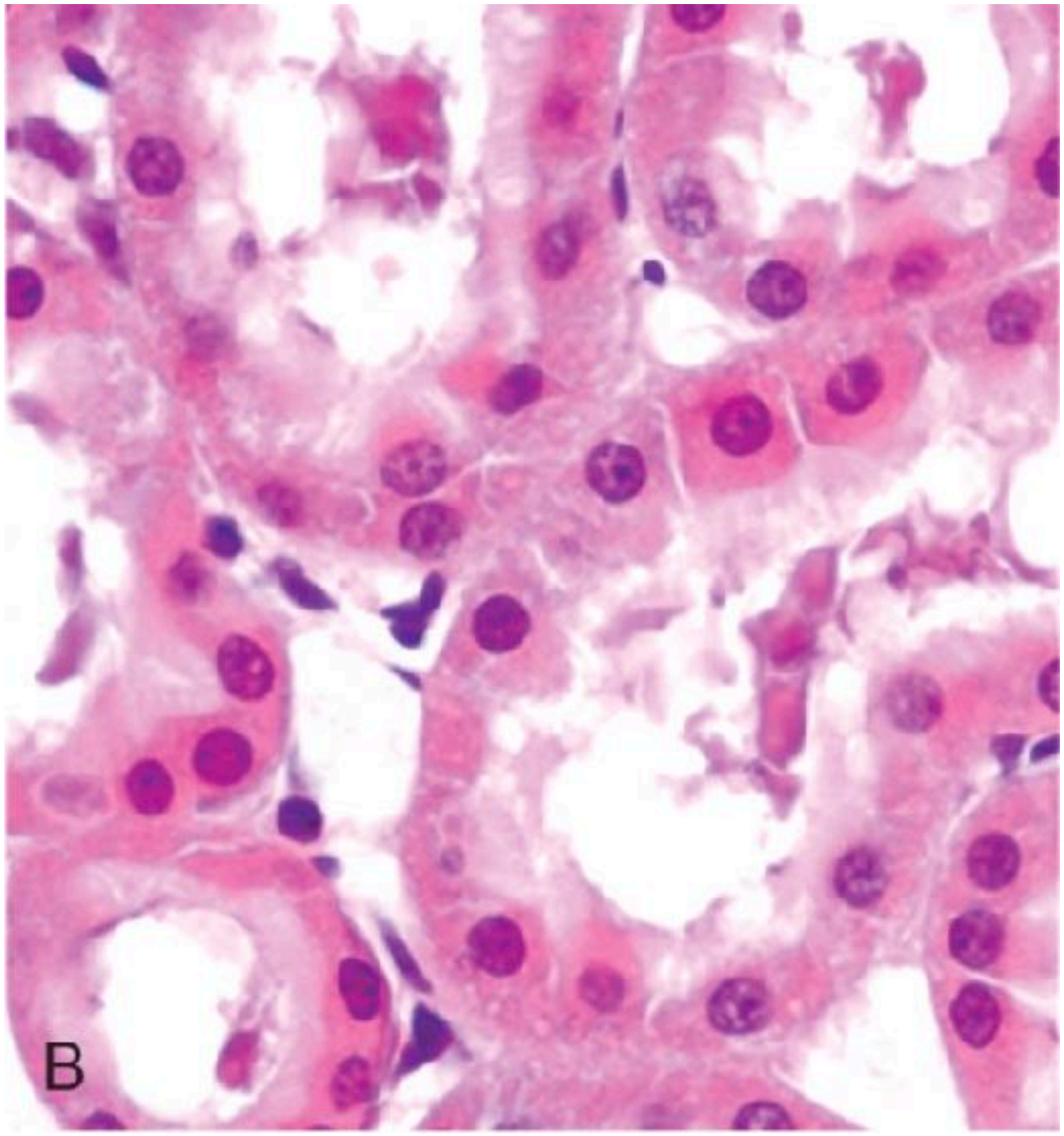
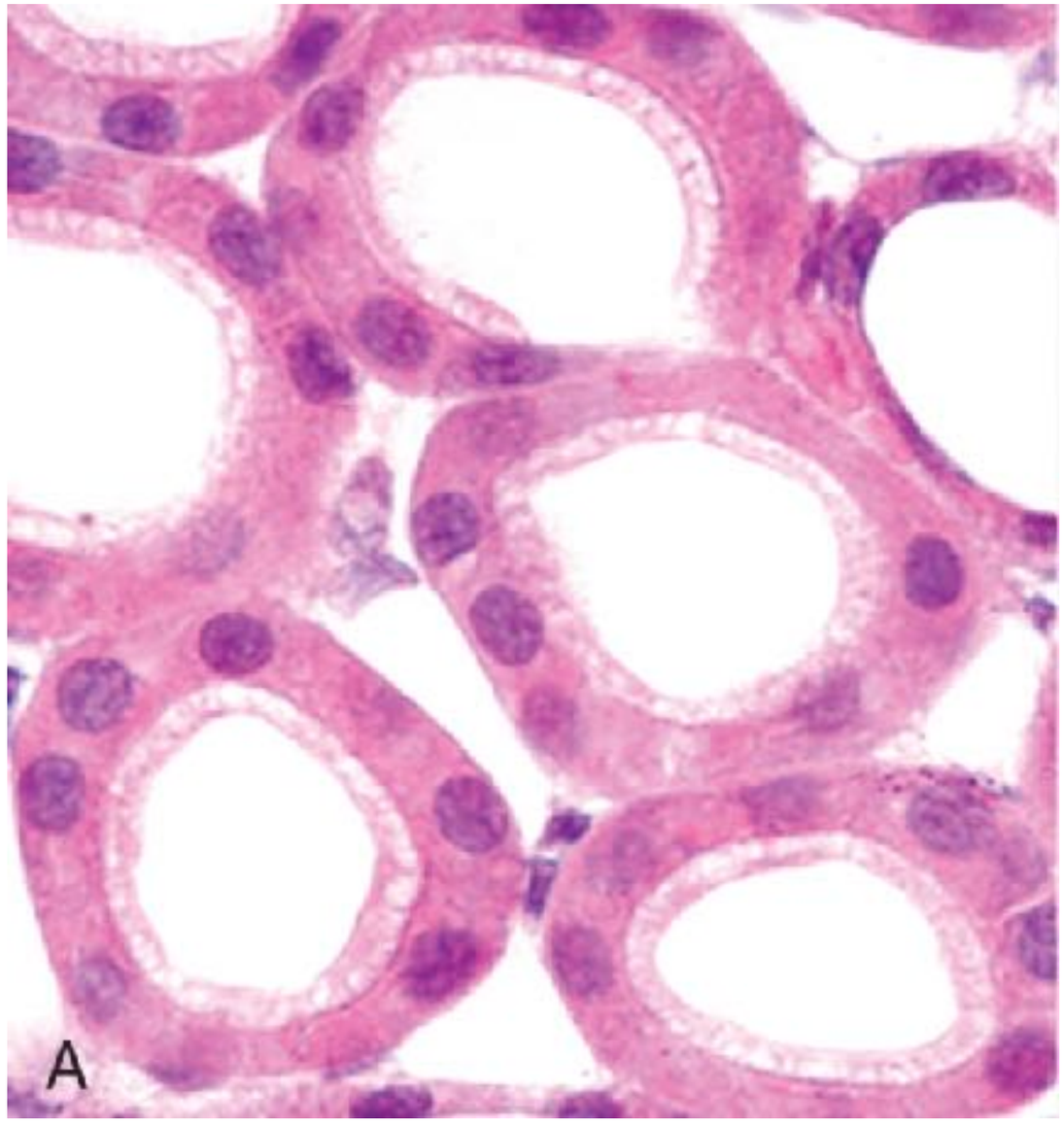
## Early (reversible) ischemic injury showing surface blebs, increased eosinophilia of cytoplasm, and swelling of occasional cells.




Reversible injury




Normal Renal tubular cells

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# Irreversible Cell Injury

Occurs when damage surpasses the cell's ability to recover, leading to **cell death** (necrosis or apoptosis).

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**MBChB. MSc. PhD.**

# Irreversible Cell Injury

**Outcome**: Cell death with inflammation (**necrosis**) or programmed cell death without inflammation (**apoptosis**).

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# Irreversible Cell Injury

**Hallmarks:**

- **Severe membrane damage**
- **Calcium influx**, activating destructive enzymes
- Mitochondrial dysfunction (ATP depletion)
- **Nuclear changes**: pyknosis (shrinkage), karyorrhexis (fragmentation), karyolysis (dissolution)

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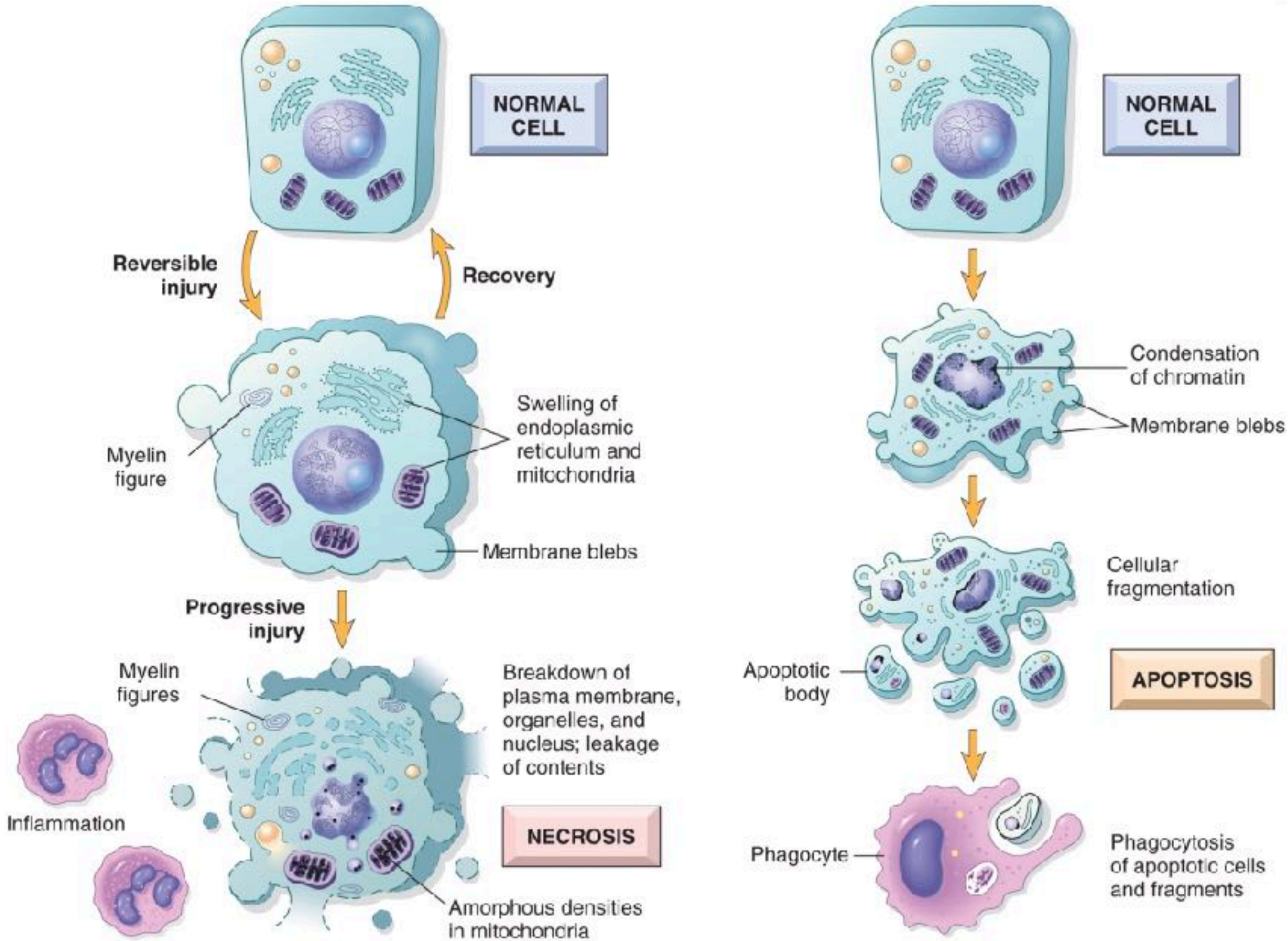


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## Necrosis

**Pathological** (unregulated) death. Cell swells, ruptures, and leaks contents, causing **inflammation**.

**Pyknosis** (nuclear shrinkage), **Karyorrhexis** (fragmentation), **Karyolysis** (dissolution).

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## Apoptosis

**Programmed** (regulated) cell death. Cell shrinks, forms **apoptotic bodies**, and is phagocytosed **without inflammation**.

Cell shrinkage and chromatin condensation.

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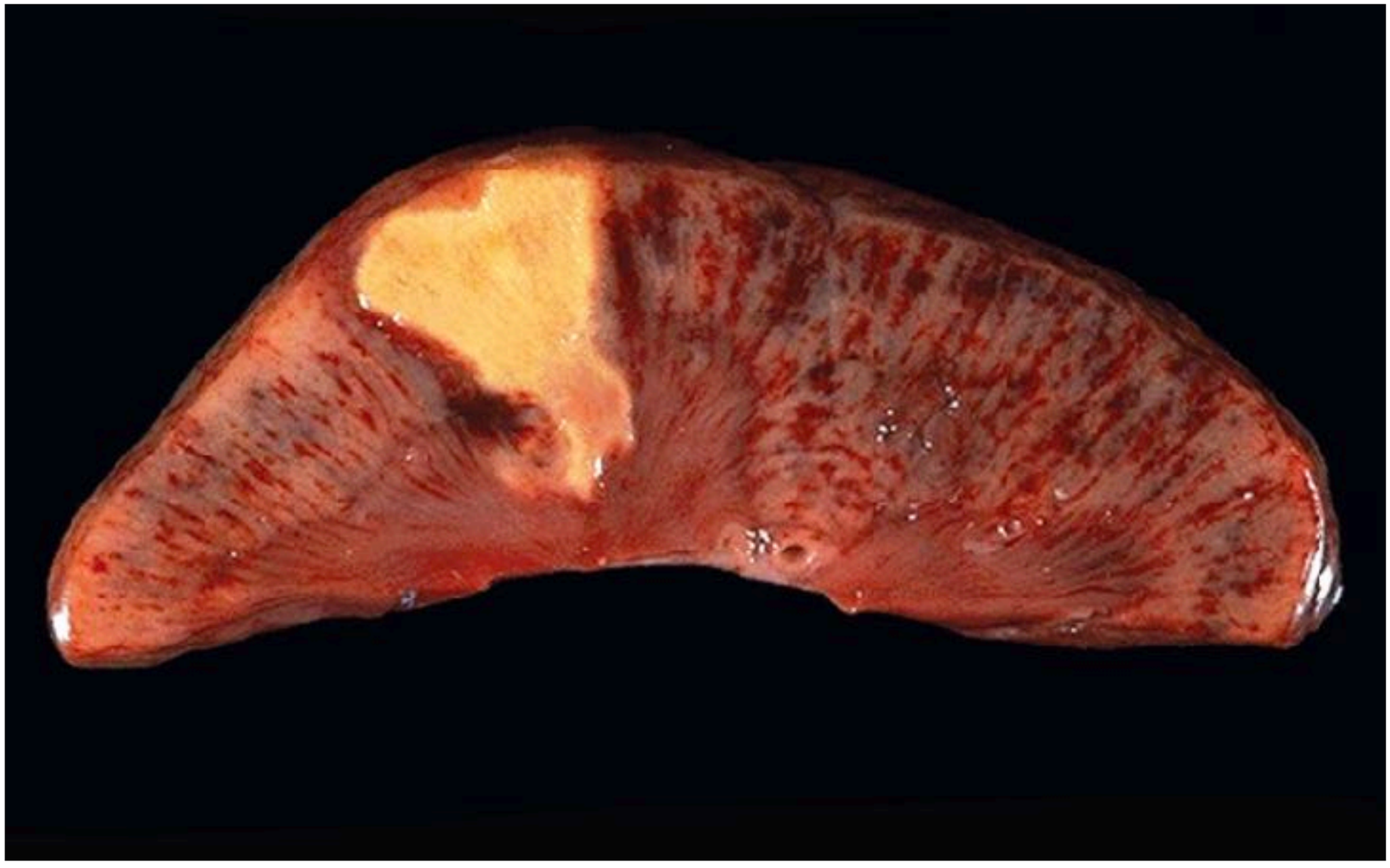
## Wedge shaped kidney infarct (yellow) with preservation of the outlines.



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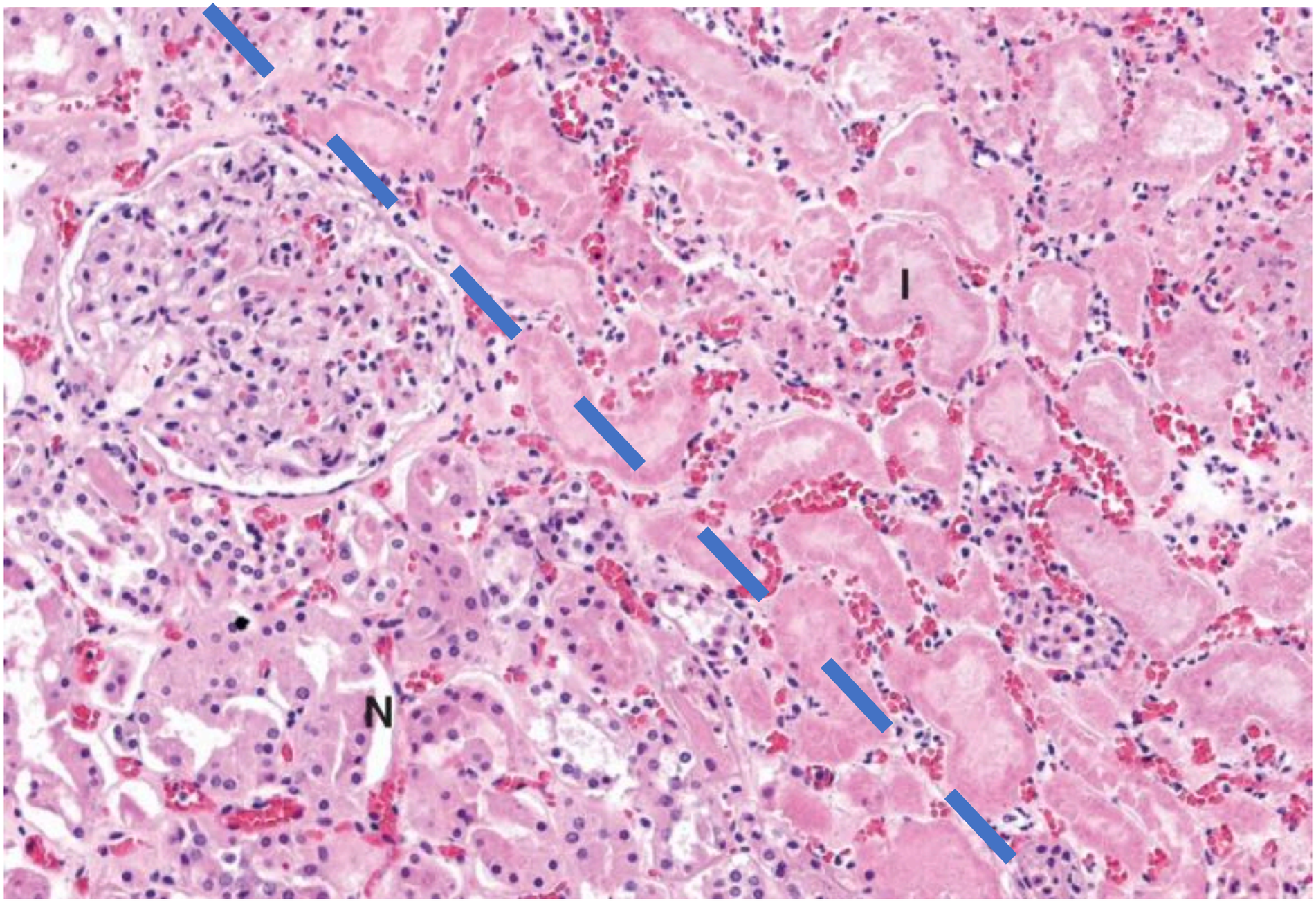
# Microscopic view of the edge of kidney infarct.

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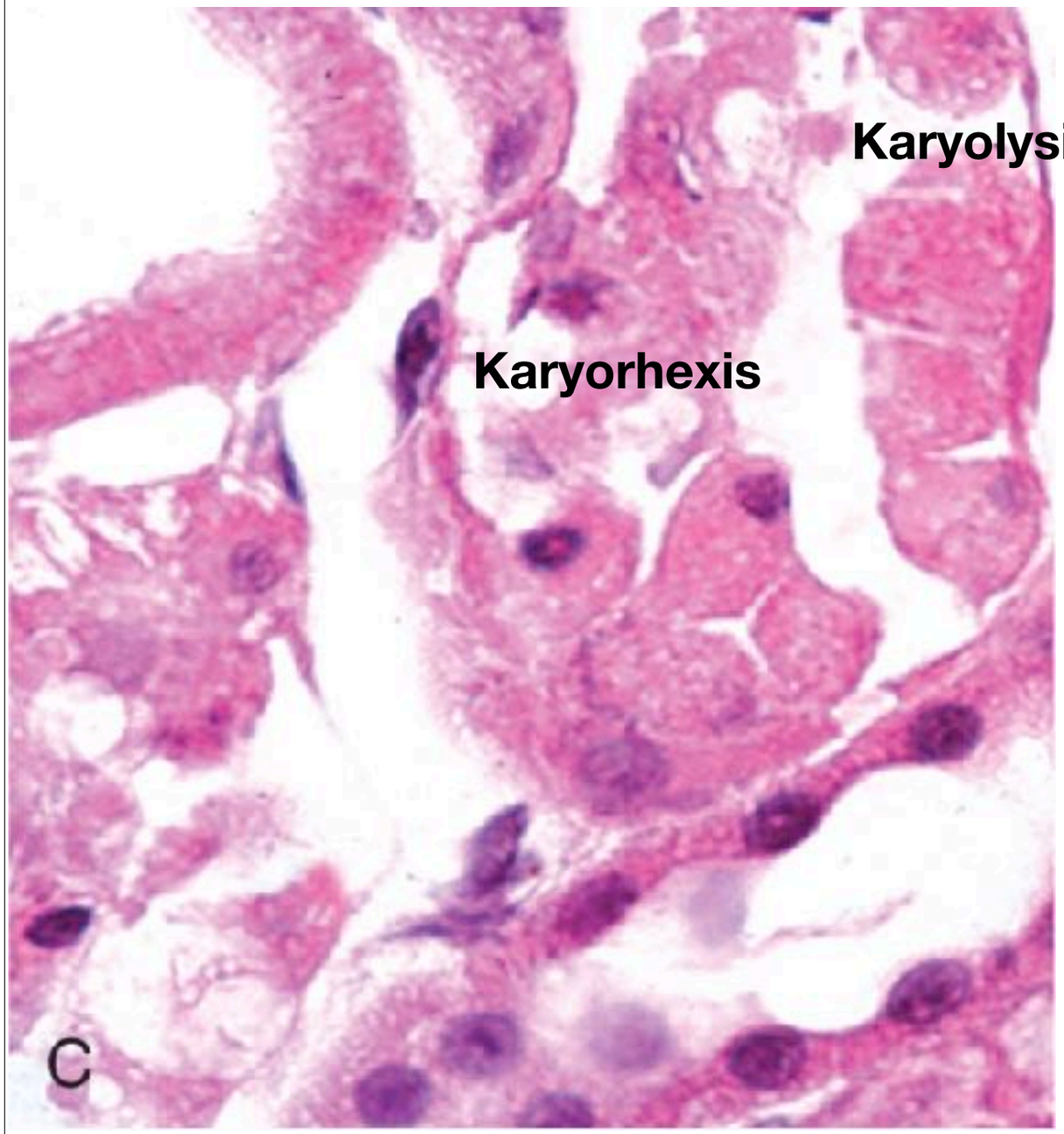
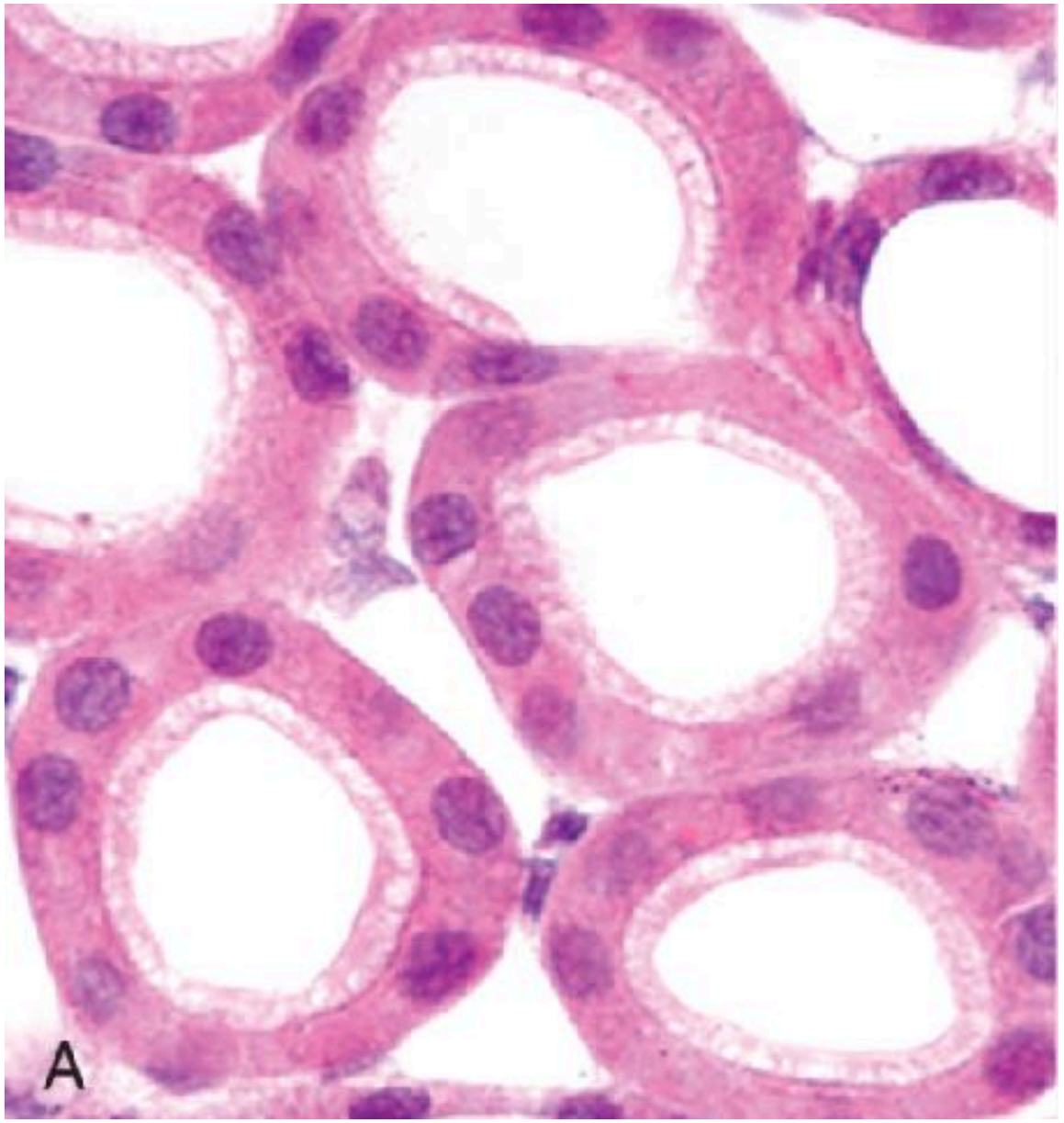
**Necrotic (irreversible injury of epithelial cells, with loss of nuclei and fragmentation of cells and leakage of contents.**

**Necrosis**




**Normal Renal tubular cells**

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# Accumulations

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# Deposits and Pigmentation

Pigmentation refers to the accumulation of coloured substances in cells or tissues.

**Common types:**

- **Endogenous pigments**: Made within the body
- **Exogenous pigments**: From outside sources

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# Deposits and Pigmentation

Deposits may also include abnormal **accumulation of substances** like:

- Lipids (fatty liver)
- Proteins (amyloidosis)
- Minerals (calcification)

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# Internal (Endogenous) Pigmentation

Comes from the body's own metabolism.

**Examples:**

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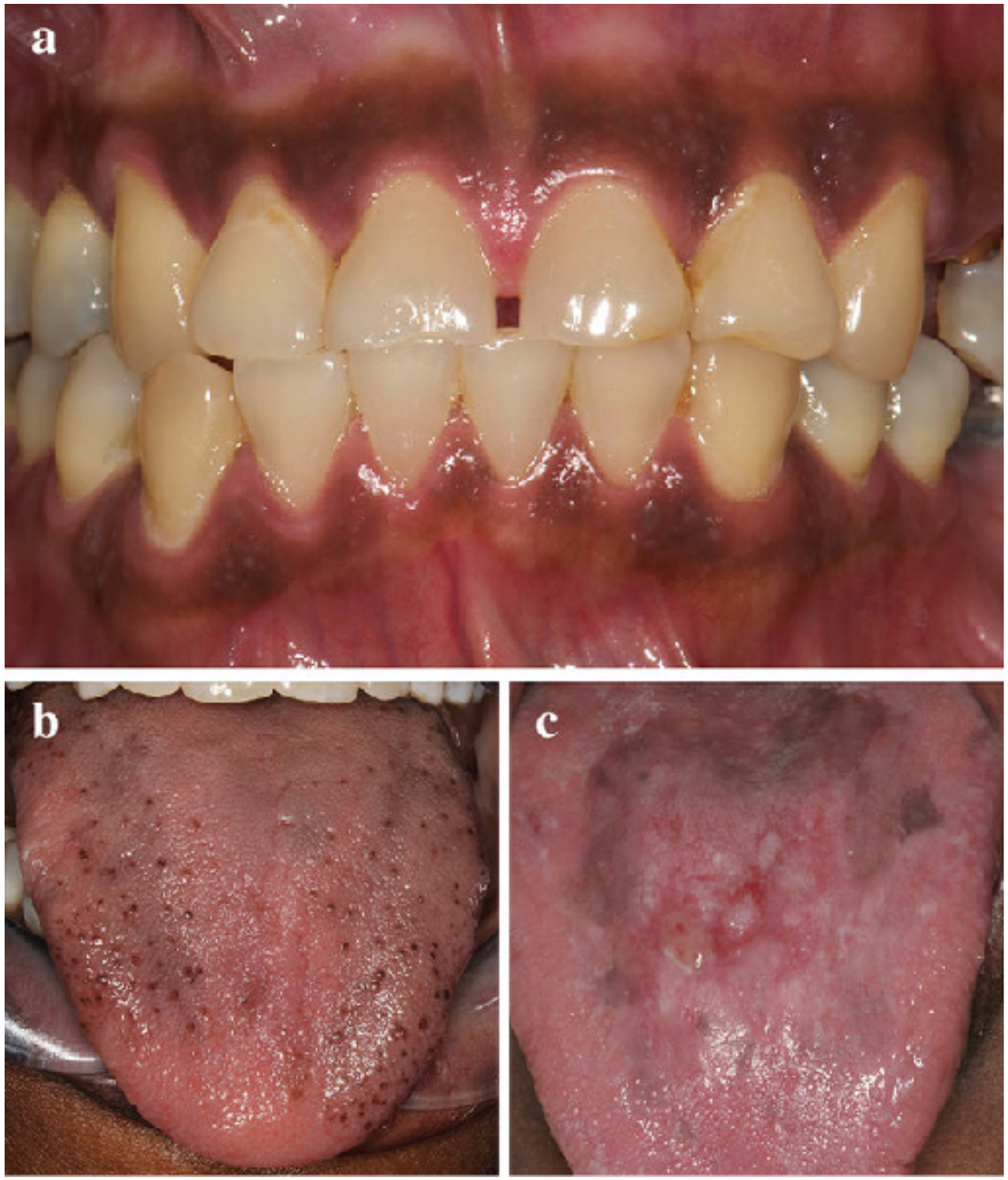
- **Melanin**: Brown-black pigment in skin, eyes (produced by melanocytes)

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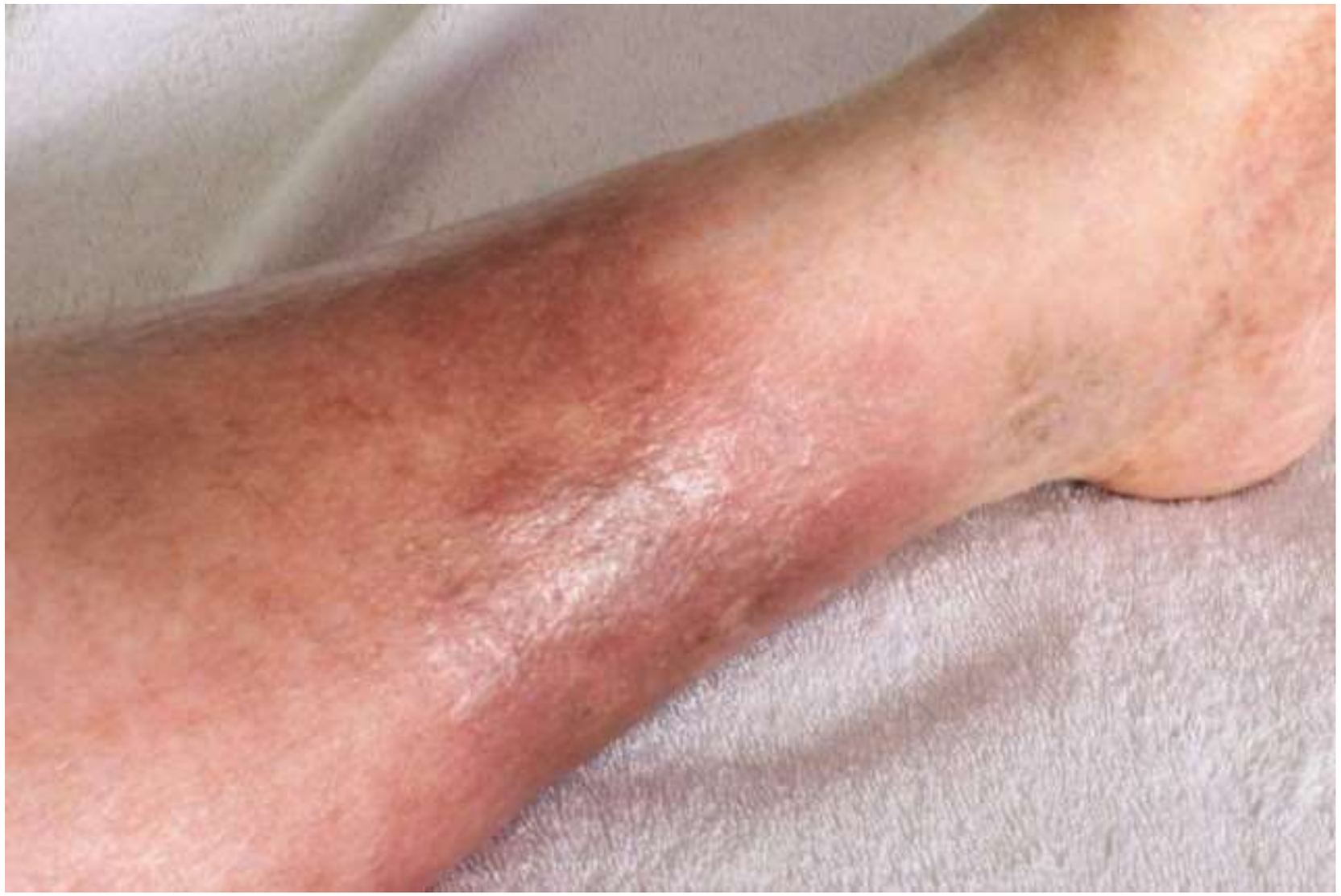
- **Hemosiderin**: Golden-brown pigment from breakdown of hemoglobin (seen in bruises or iron overload)



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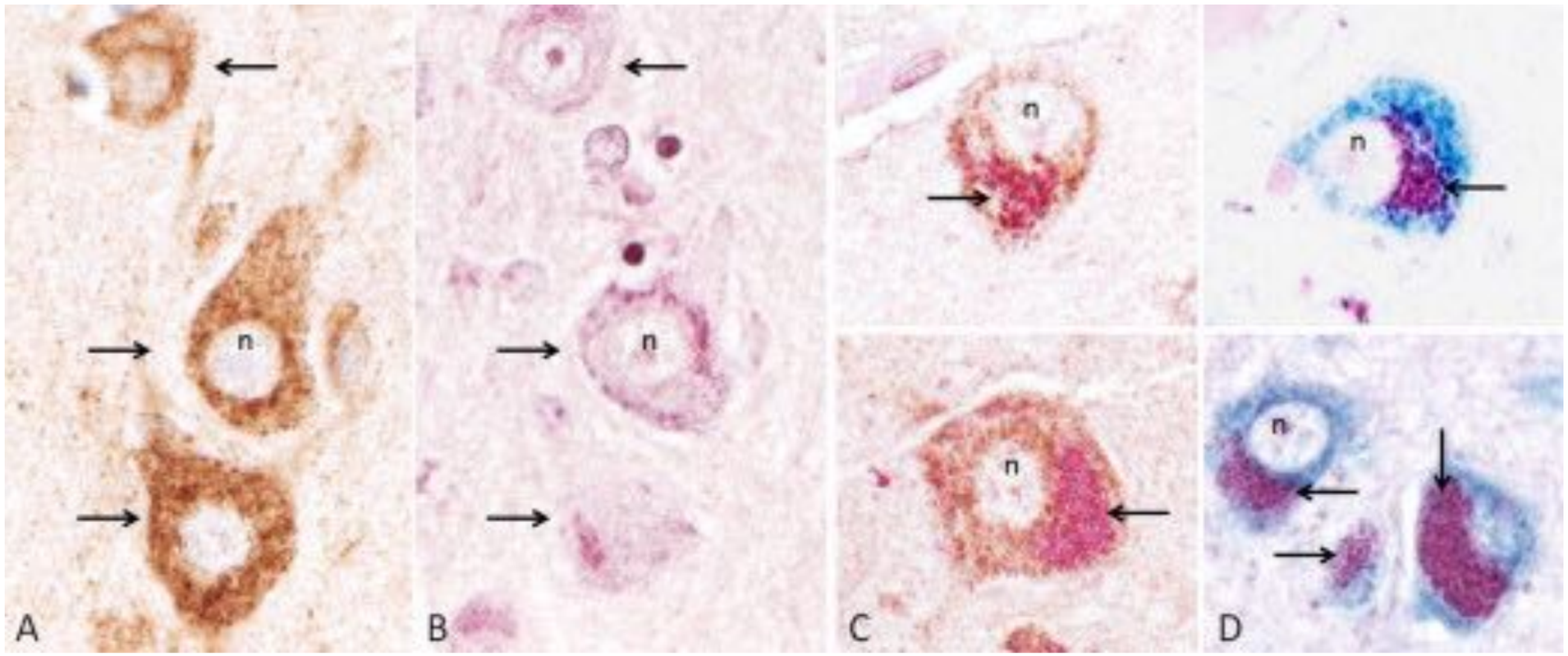
- **Lipofuscin**: "Wear and tear" pigment, yellow-brown, increases with aging

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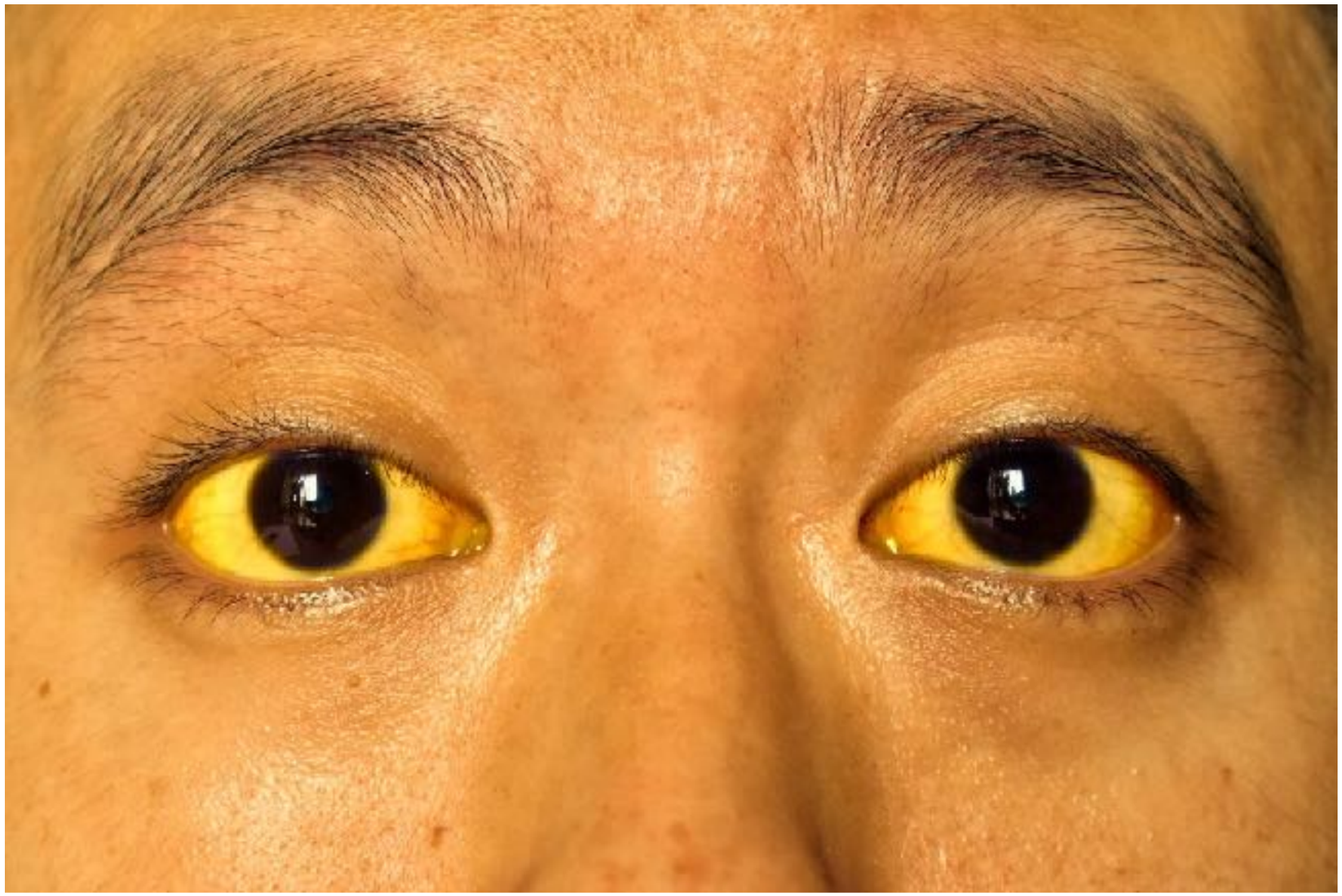
- **Bilirubin**: Yellow pigment from heme breakdown; excess causes jaundice



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# External (Exogenous) Pigmentation

Pigments from outside the body, often environmental.

**Examples:**

- **Carbon (anthracosis)**: Black pigment in lungs from inhaling smoke/pollution
- **Tattoos**: Ink particles taken up by dermal macrophages
- **Heavy metals**: Lead lines in gums, silver (argyria) causing skin and gum discoloration

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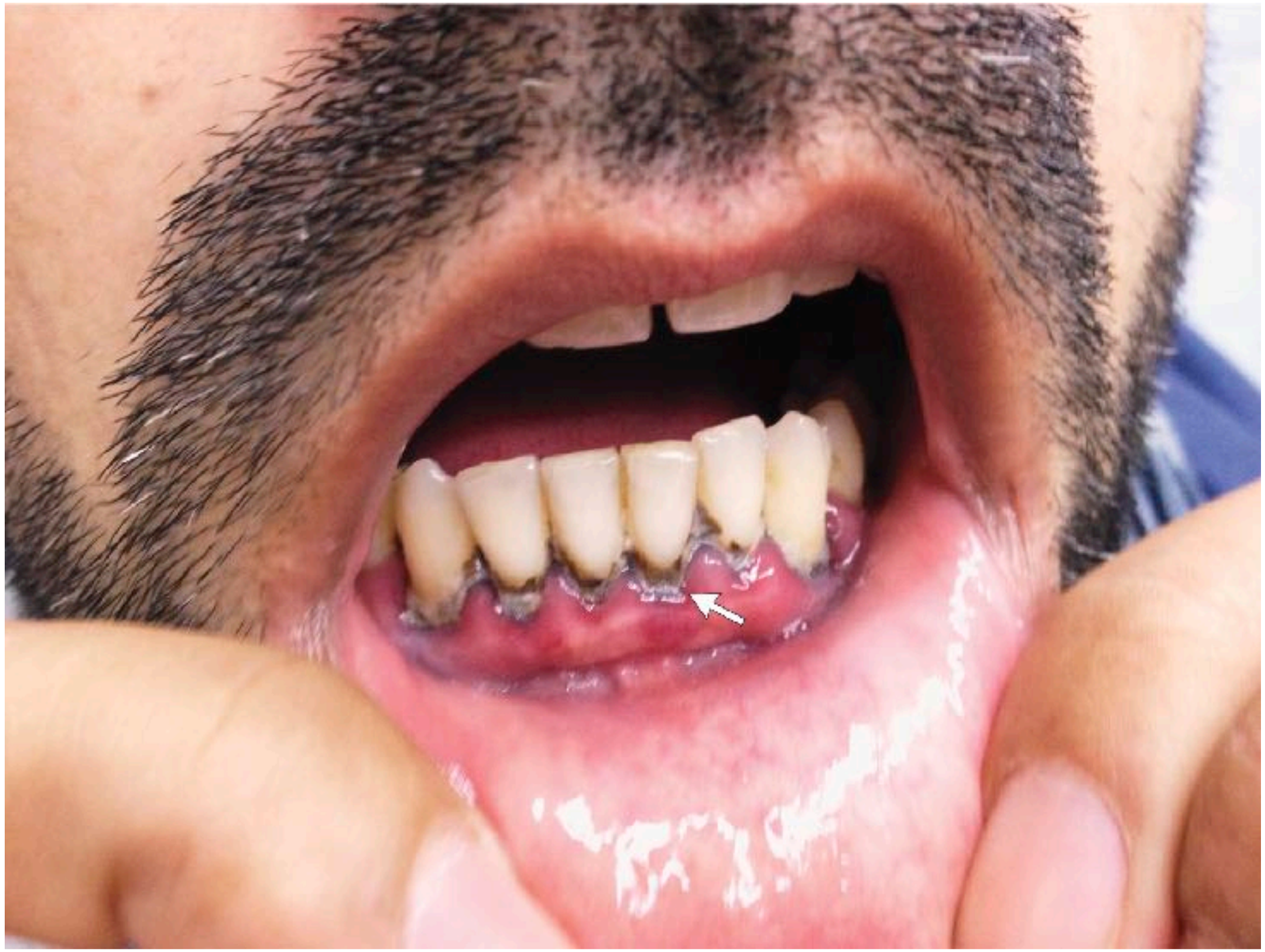
# Lead Line



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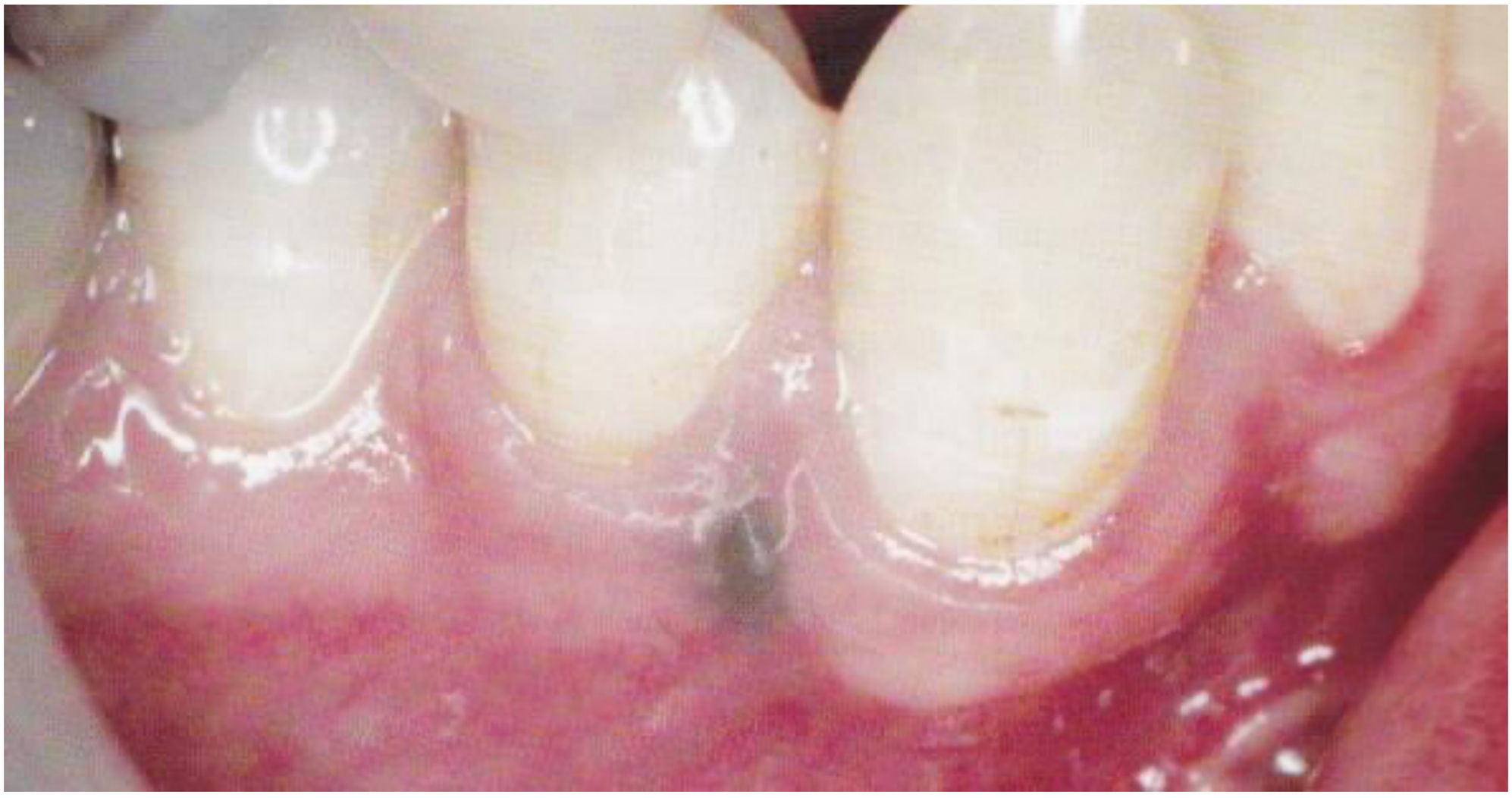
# Amalgam tattoo



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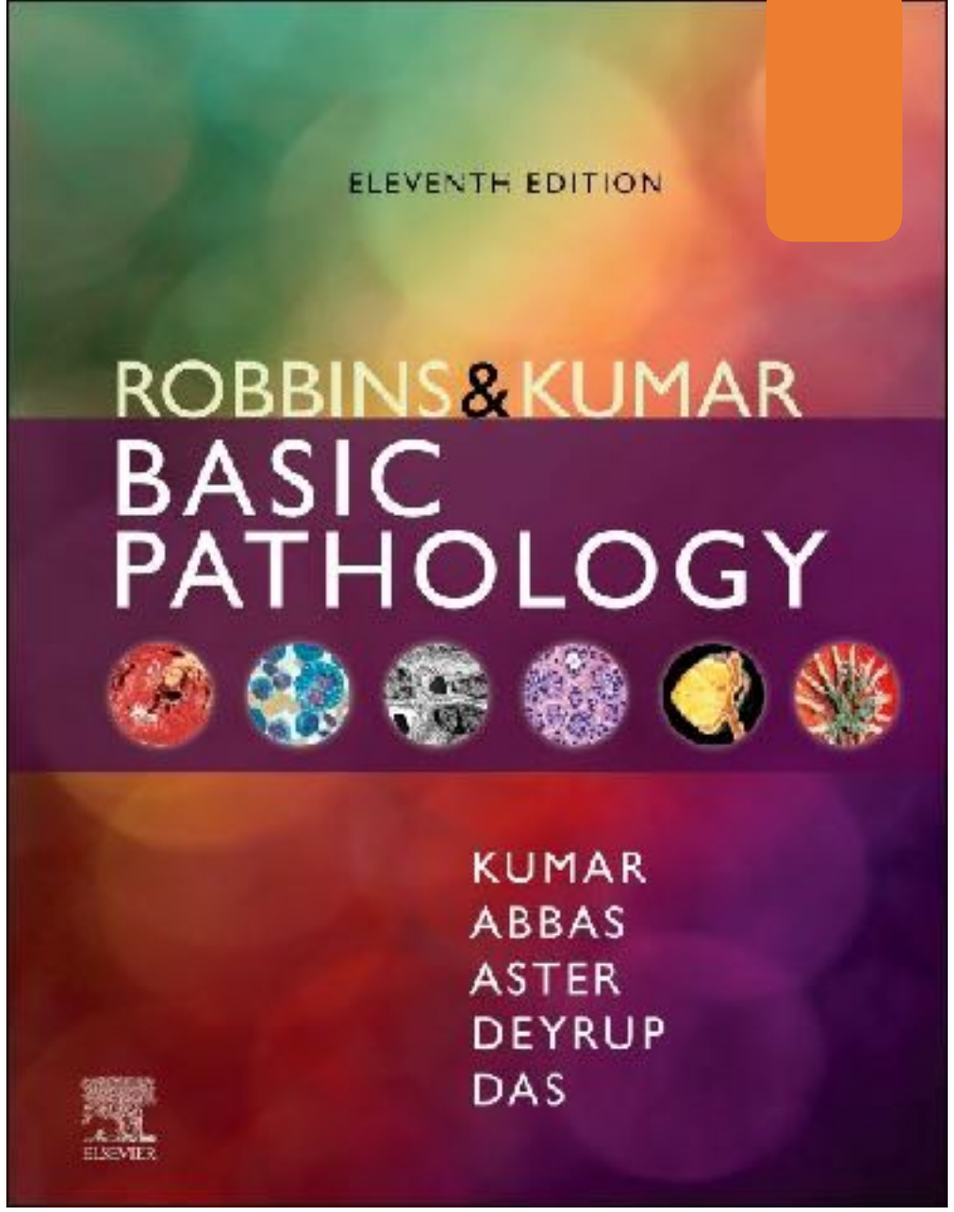


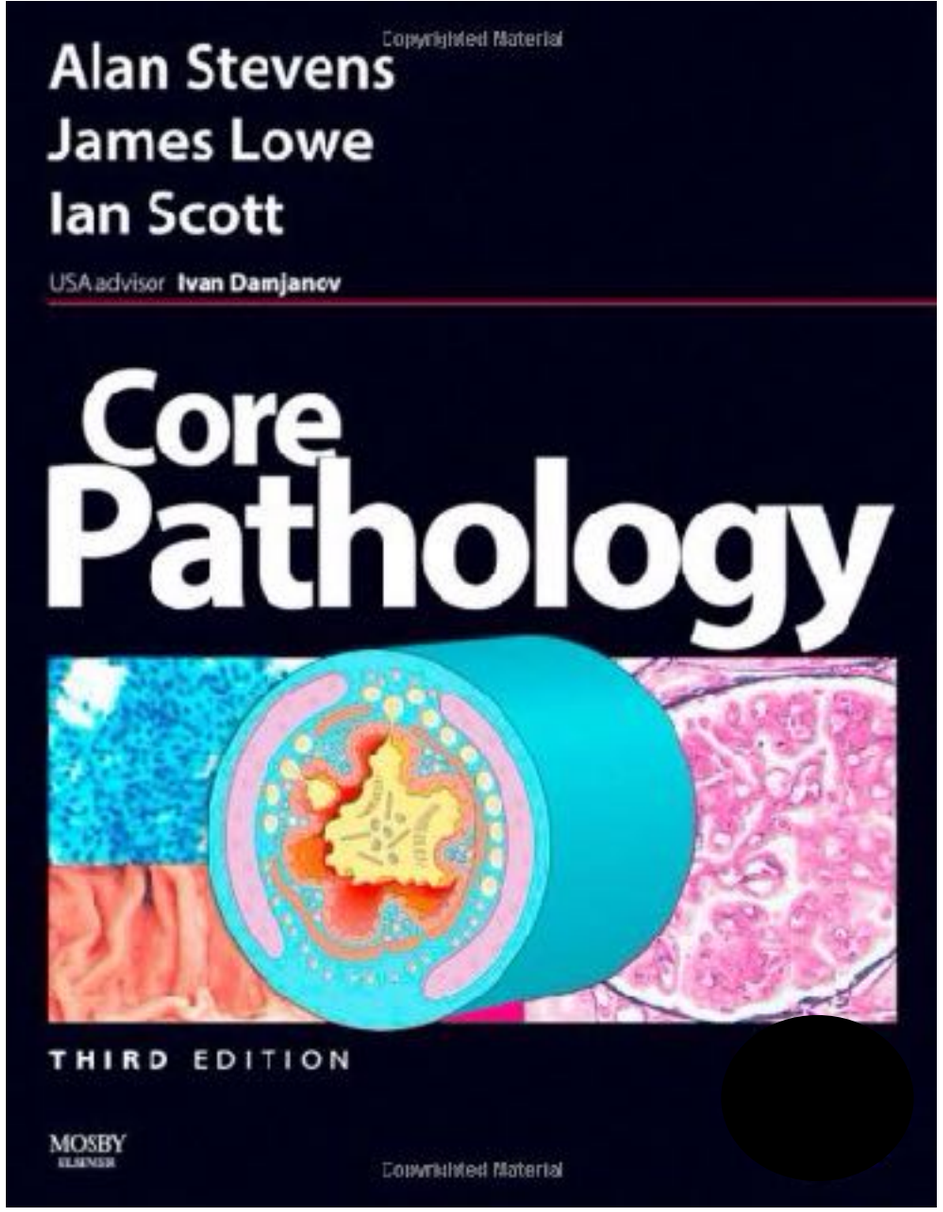


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