

HM/CH-1/L-7

MORPHOLOGY OF CELL INJURY

MORPHOLOGIC FORMS OF CELL INJURY

MECHANISMS

1. Reversible cell injury
2. Deranged cell metabolism
3. Irreversible cell injury
4. Programmed cell death
5. Residual effects
6. After-effects

NOMENCLATURE

1. Retrogressive changes(degenerations)
2. Intracellular accumulations
3. Cell death-necrosis
4. Apoptosis
5. Subcellular alterations
6. Gangrene, pathologic calcification

DERANGED METABOLISM: INTRACELLULAR ACCUMULATION

- *Accumulation of constituents of normal cell metabolism:*
fat, proteins, CH
- *Accumulation of abnormal substances:*
storage diseases
- *Accumulation of pigments:*
endogenous, exogenous



A. ENDOGENOUS PIGMENTS

1. Melanin
2. Melanin-like pigment
 - a. Alkaptonuria
 - b. Dubin-Johnson syndrome
3. Haemoprotein-derived pigments
 - i) Haemosiderin
 - ii) Acid haematin (Haemozoin)
 - c. Bilirubin
 - d. Porphyrins
4. Lipofuscin (Wear and tear pigment)

B. EXOGENOUS PIGMENTS

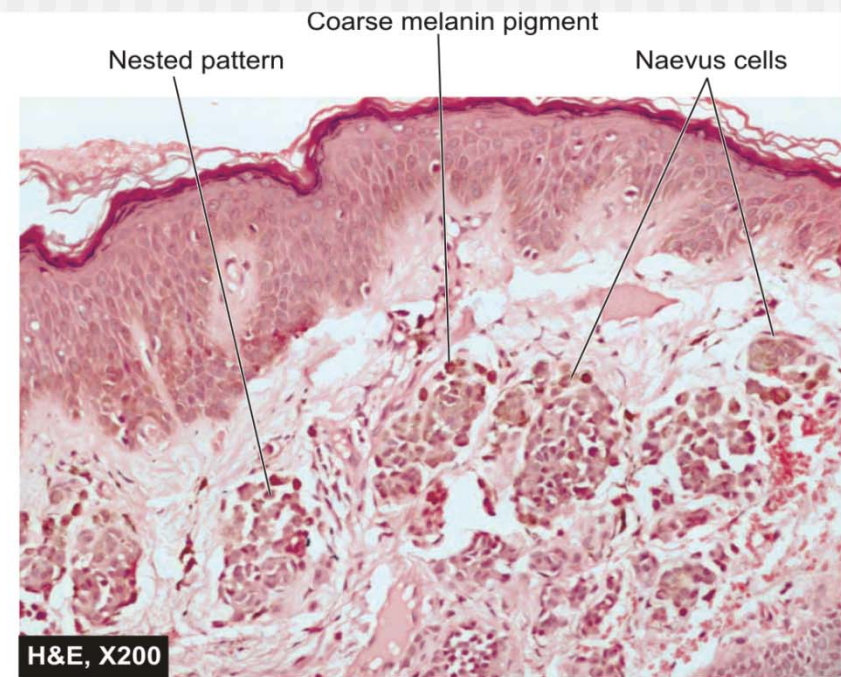
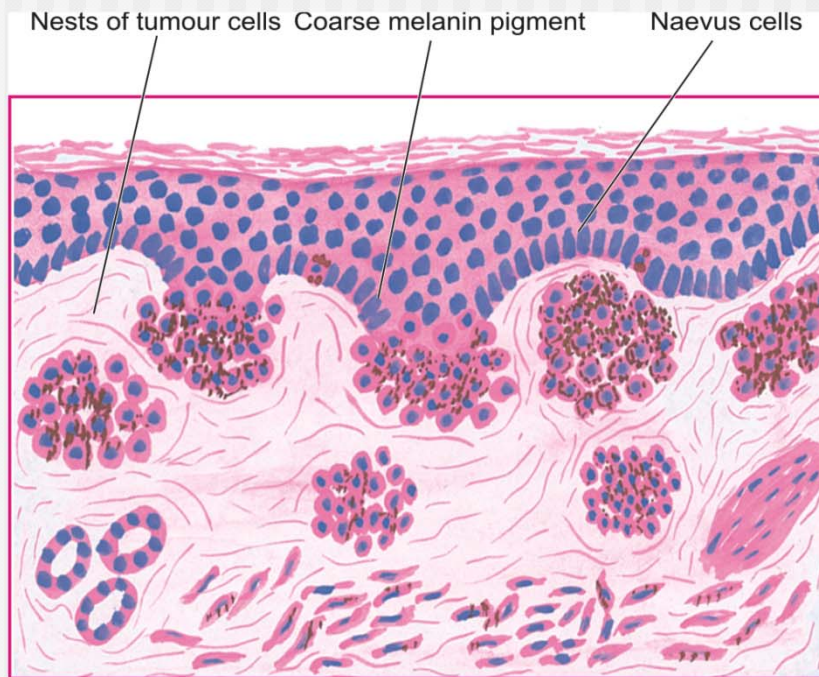
1. Inhaled pigments
2. Ingested pigments
3. Injected pigments (Tattooing)

ENDOGENOUS PIGMENTS:

Melanin

- Generalised hyperpig:
 - Addison's disease
 - Chloasma
 - Ch arsenic poisoning
- Focal hyperpig:
 - Café-au-lait spots
 - Peutz-Jeghers syndrome
 - Melanosis coli
 - Melanotic tumours
 - Lentigo
 - Dermatopathic LN
- Generalised hypopig:
 - Albinism
- Localised hypopig:
 - Leucoderma
 - Vitiligo
 - Acquired causes

Melanin in intradermal naevus



Melanin-like pigments:

Alkaptonuria

- Autosomal recessive
- Deficiency of oxidase enzyme required for breakdown of HA
- Accumulation of HA in tissues (ochronosis)
- Alkaptonuria (homogentisic aciduria)

Dubin-Johnson syndrome

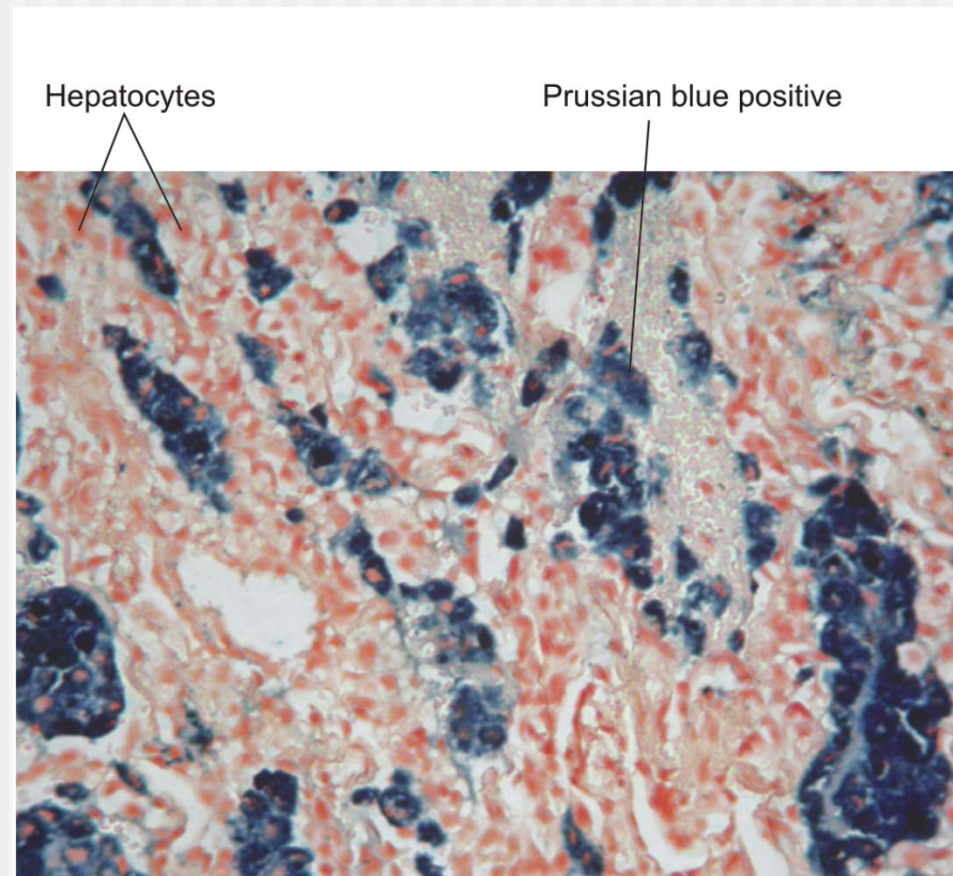
- Autosomal recessive form of hereditary conj hyperbil.

Haemoprotein-derived pigments

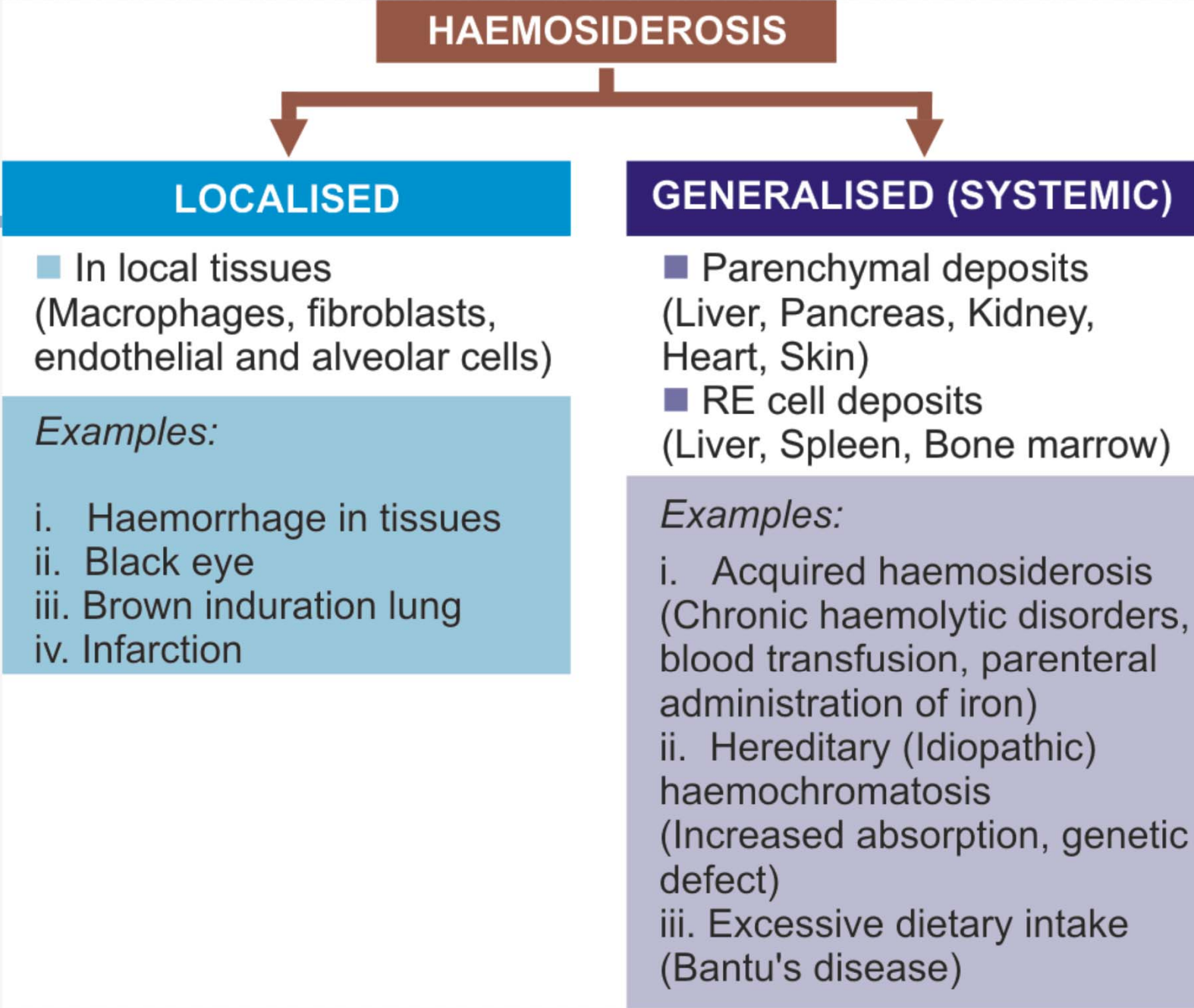
1. Haemosiderin
2. Acid haematin (haemozoin)
3. Bilirubin
4. Porphyrins

Haemosiderin

- Ferritin
- Haemosiderin (Haemosiderosis)



HAEMOSIDEROSIS



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graph TD; A[HAEMOSIDEROSIS] --> B[LOCALISED]; A --> C[GENERALISED (SYSTEMIC)];
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LOCALISED

■ In local tissues
(Macrophages, fibroblasts,
endothelial and alveolar cells)

Examples:

- i. Haemorrhage in tissues
- ii. Black eye
- iii. Brown induration lung
- iv. Infarction

GENERALISED (SYSTEMIC)

■ Parenchymal deposits
(Liver, Pancreas, Kidney,
Heart, Skin)
■ RE cell deposits
(Liver, Spleen, Bone marrow)

Examples:

- i. Acquired haemosiderosis
(Chronic haemolytic disorders,
blood transfusion, parenteral
administration of iron)
- ii. Hereditary (Idiopathic)
haemochromatosis
(Increased absorption, genetic
defect)
- iii. Excessive dietary intake
(Bantu's disease)

Other haemoprotein-derived pigments

2. Acid haematin (haemozoin)

3. Bilirubin

- Conj & unconj hyperbilirubinaemia (jaundice)

4. Porphyrins

- Erythropoietic porphyria: defective haem synthesis in red cells

- Hepatic porphyria: normal erythroid precursors but a defect in synthesis of haem in liver

Lipofuscin (wear and tear pigment)

■ LM

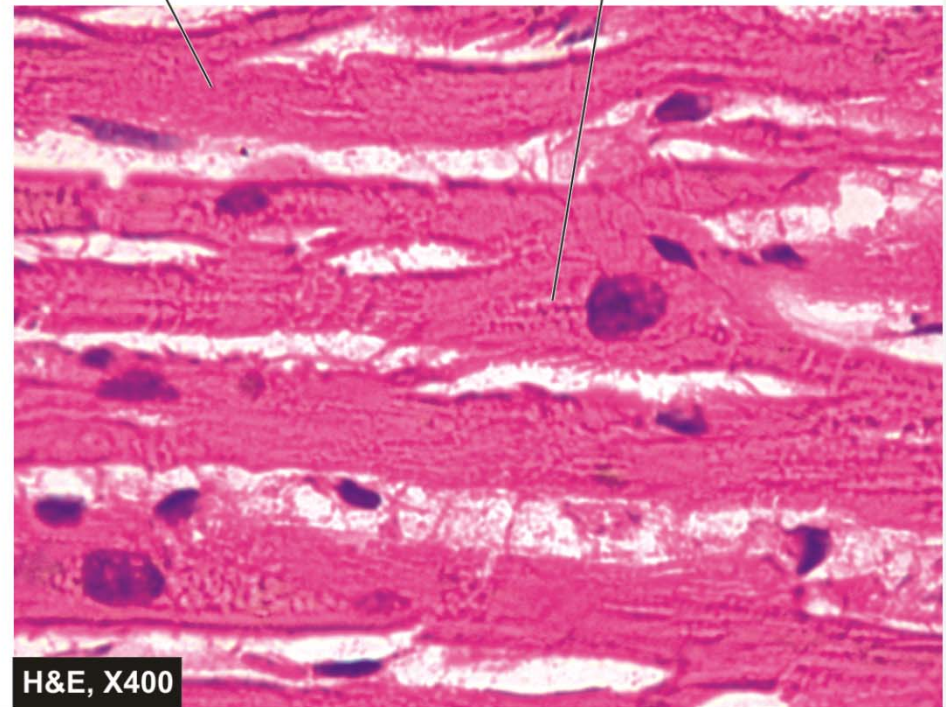
■ EM

Perinuclear lipofuscin granules



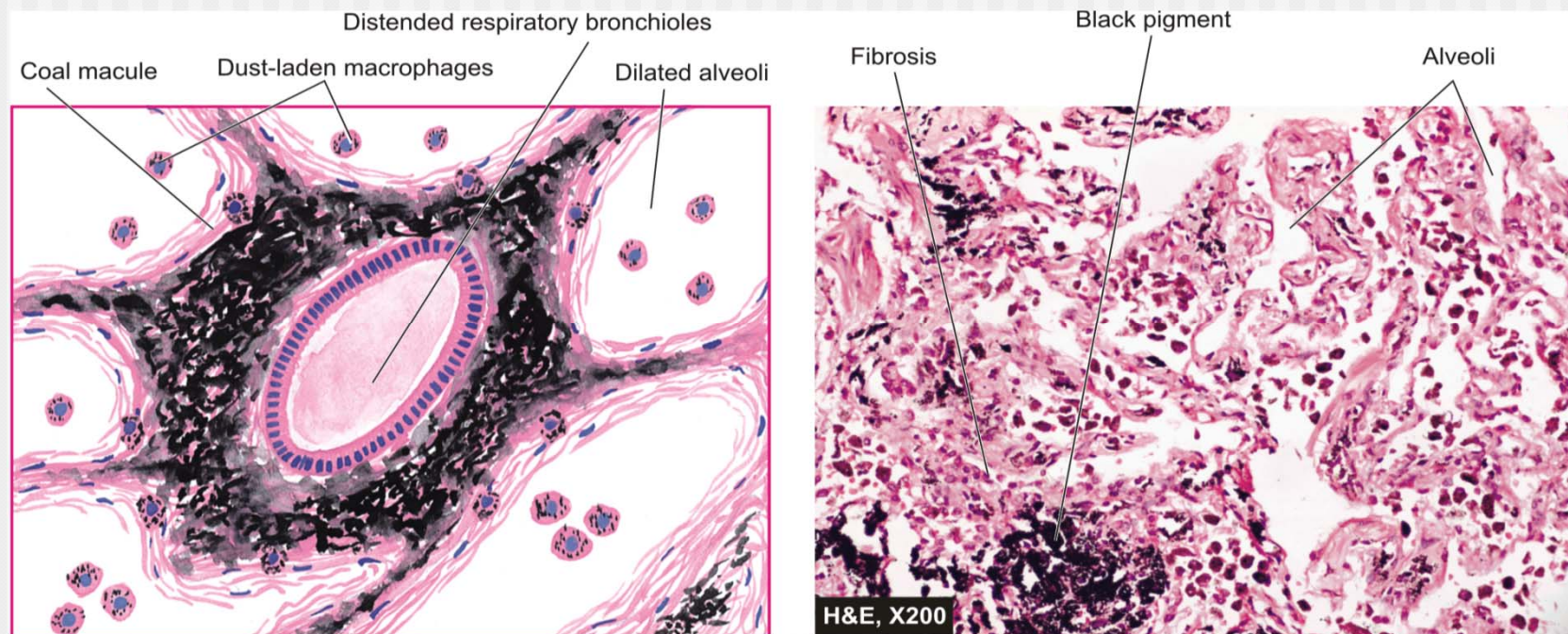
Myocardial fibres

Yellow-brown lipofuscin (perinuclear)



EXOGENOUS PIGMENTS

- Inhaled pigments: various dusts



- Ingested pigments: argyria, ch lead poisoning, carotinaemia
- Injected pigments (tattooing): India ink, carbon