Clinical Scenario

A 47-year-old man who is a worker on demolition sites, comes to you for review. He is having increasing tiredness, lethargy, headache, and abdominal pains over the past 3 months. On examination, he is hypertensive with a blood pressure of 160/90 mmHg, he has a regular pulse of 82 beats per minute and he looks pale.

Investigations

Hb - 9.7 g/dl

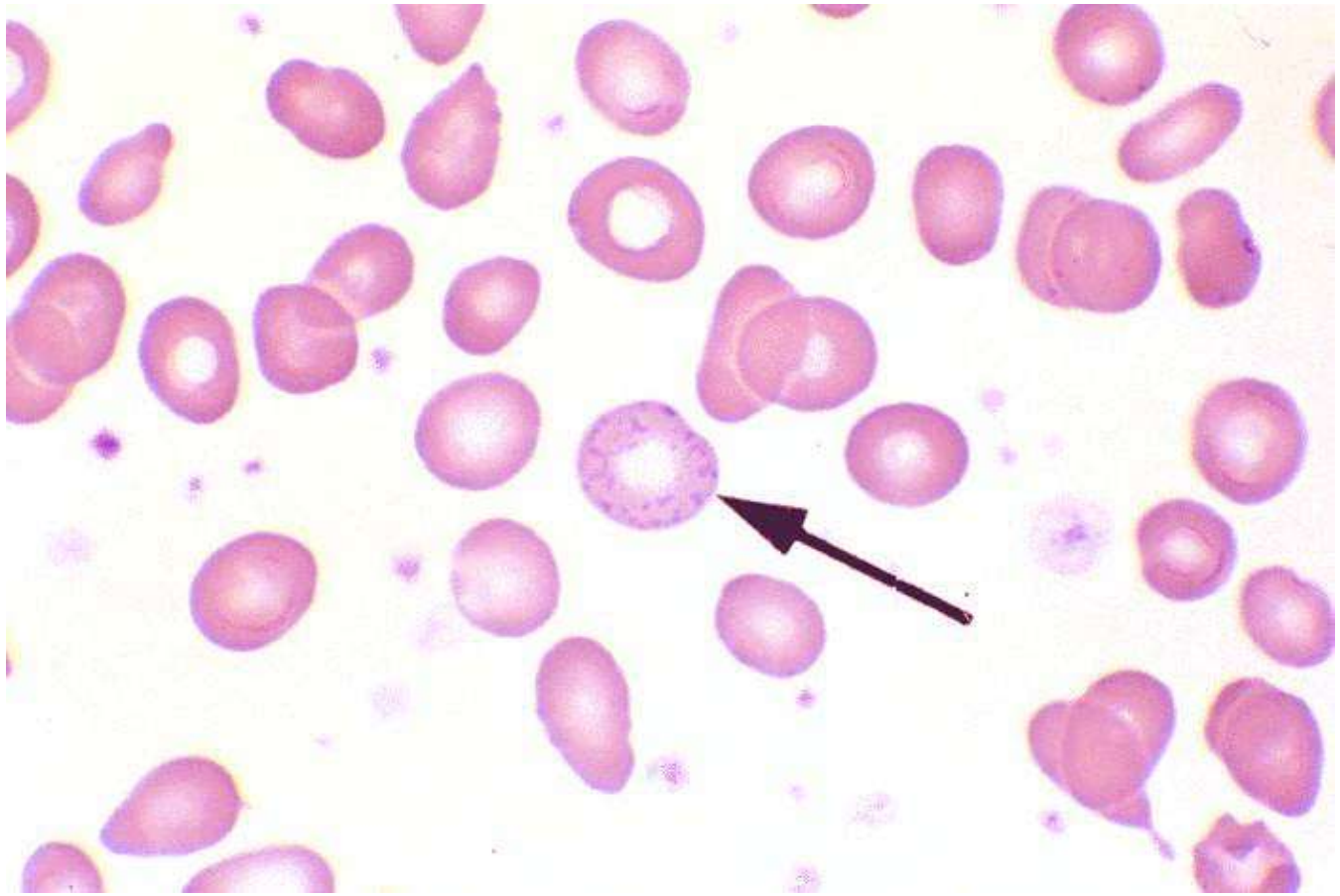
MCV 78 fl

Platelets 175,000/µL

WBCs 6000/ µL

Lead level - 100 µg/dl

Peripheral blood film reveals basophilic stippling.

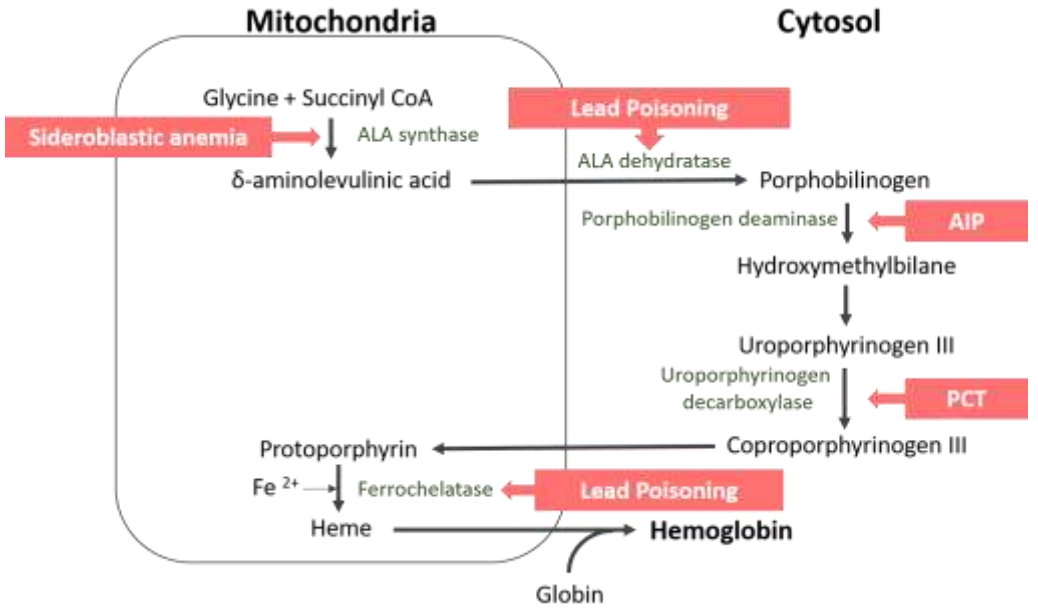


Lead Toxicity *or* Poisoning

Abdominal pain + Neurological signs = Acute porphyria , Lead poisoning

- Chronic occupational exposure
- Leaded paint
- Water contaminated by lead pipes
- Use of kohl cosmetics

- Lead poisoning affects liver, kidneys, nervous system, bone, teeth & heme.
- Directly toxic to cell membranes through free radicals
- Interferes with DNA transcription, enzymes, & NMDA receptors in nervous system
- Most characteristic - Defective ALA dehydratase & ferrochelatase function during heme synthesis
  - Anemia
  - Build-up of heme precursors, such as ALA which may be directly or indirectly harmful to neurons
  - Elevated ALA results in lead poisoning's symptoms similar to acute porphyria



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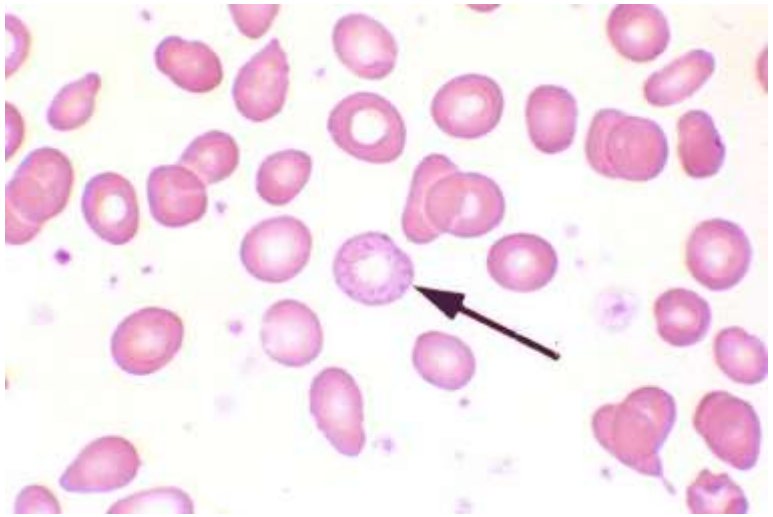
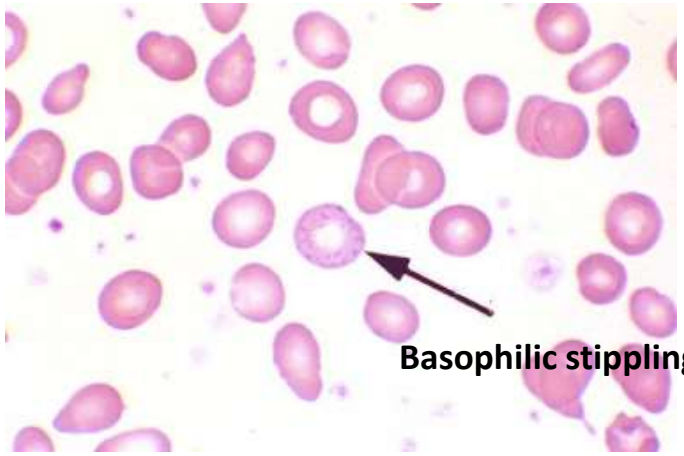
Features:

- Fatigue
- Abdominal pain
- Headache and encephalopathy
- Peripheral neuropathy (mainly motor neuropathy)
- Nephrotoxicity
- Constipation
- Blue lines on gum margin (only in 20% of adult patients & very rare in children)
- Hypertension
- Microcytic anaemia with basophilic stippling
- Hypocalcaemia

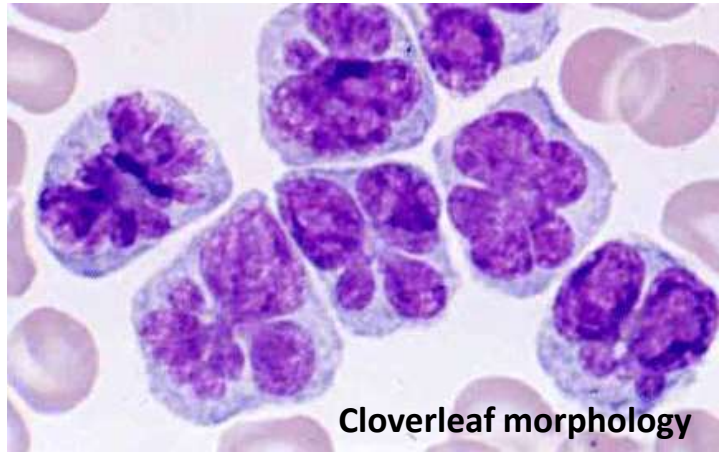


Investigations

- Blood **Lead level** - used for diagnosis
  - > 10 µg/dl – Significant
- **Full blood count (FBC)** - Microcytic Anemia
- **Blood film** - *Basophilic* stippling & *Clover-leaf* morphology
- **Renal, liver function tests** & serum **Calcium** levels
- Raised **serum and urine levels of delta aminolaevulinic acid**, making it difficult to differentiate from acute intermittent porphyria
- **Urinary coproporphyrin** - also increased
- **Urinary porphobilinogen & uroporphyrin** levels are normal to slightly increased
- **X-rays** - Lead accumulate in metaphysis of the bones



Basophilic stippling



Cloverleaf morphology

Management

- Prevent further exposure
- **Chelation Therapy**
  - Dimercaptosuccinic acid (DMSA)
  - D-penicillamine
  - EDTA (in acute cases)
  - Dimercaprol

Last Second Medicine

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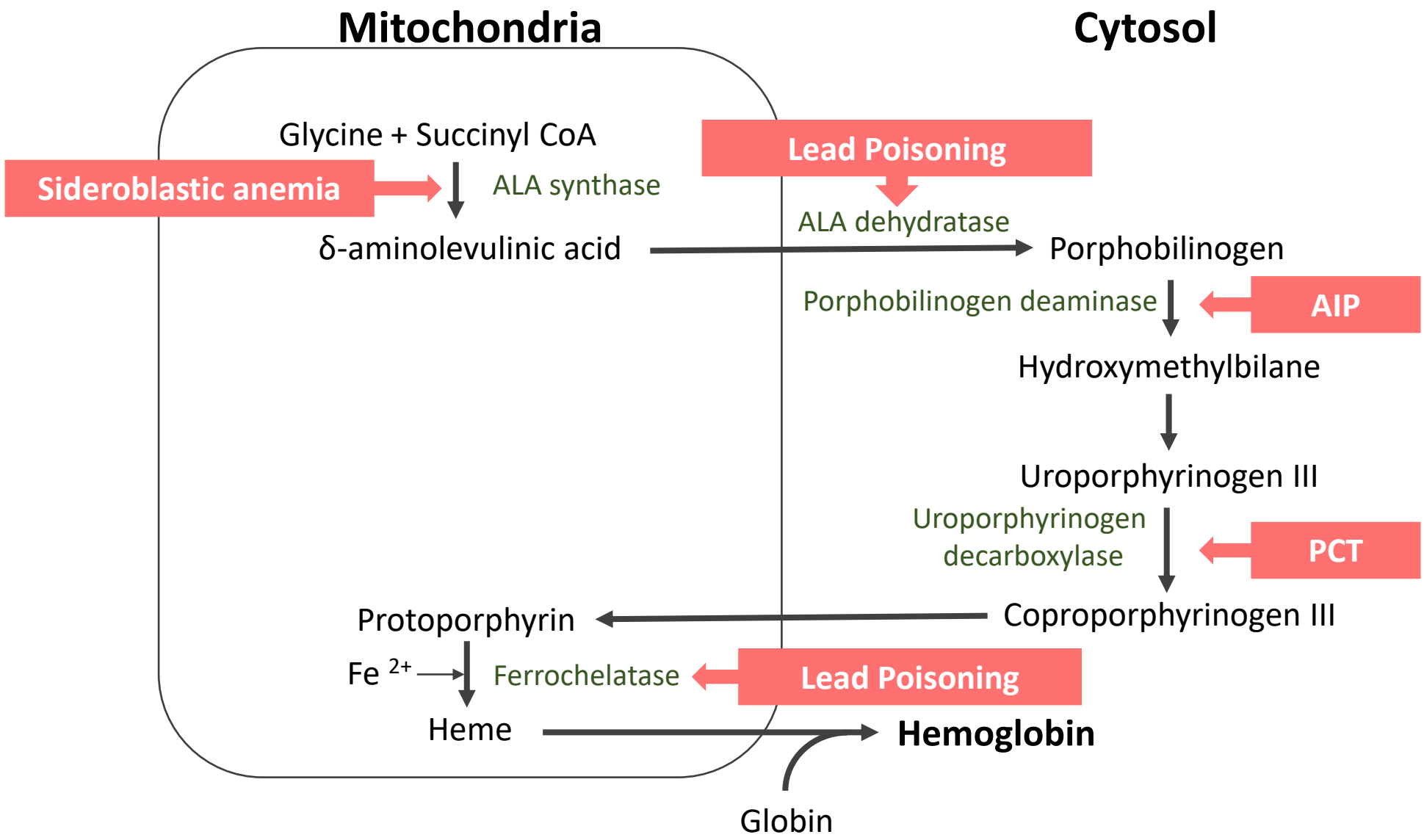
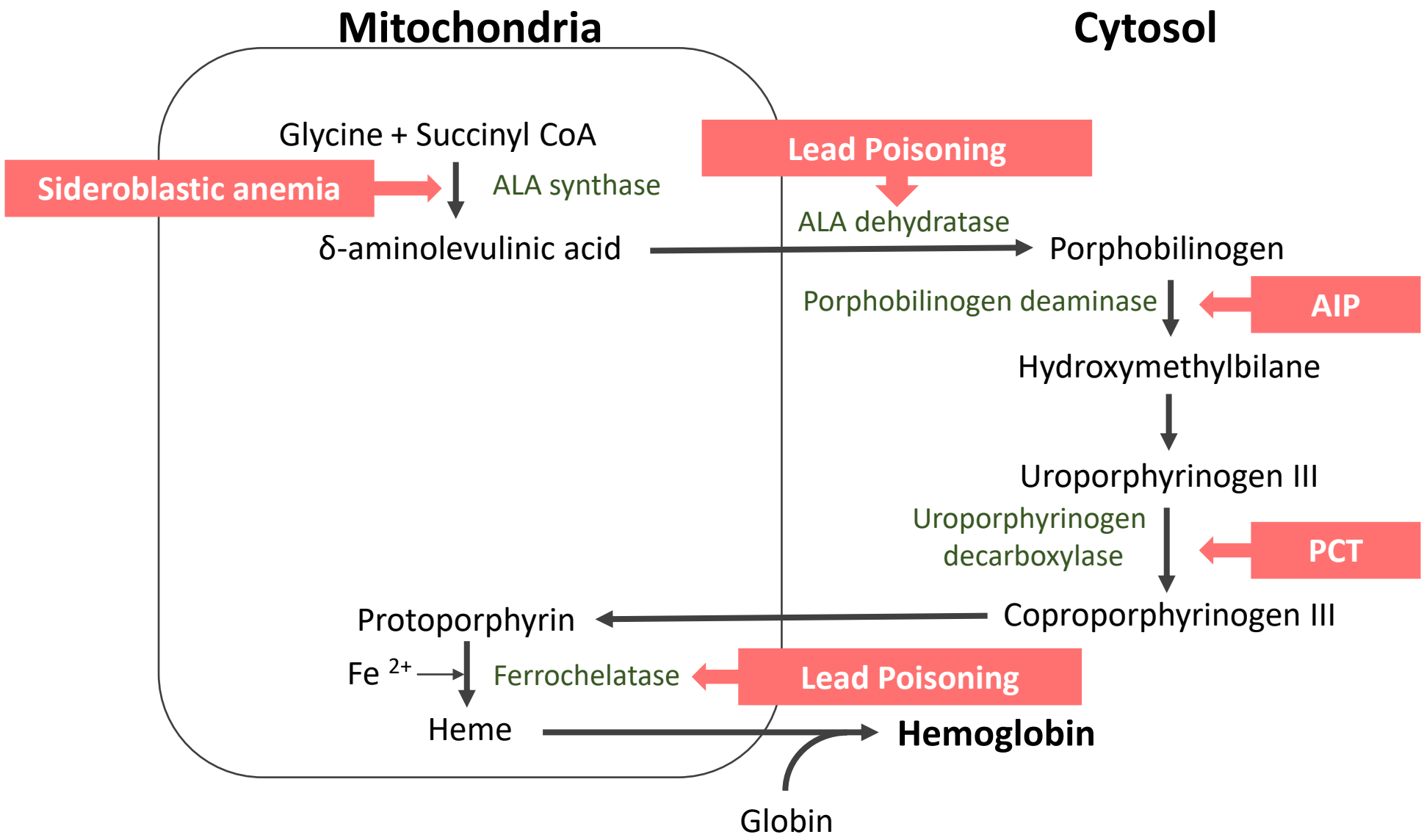

Mitochondria
Cytosol
Glycine + Succinyl CoA
Sideroblastic anemia
ALA synthase
δ-aminolevulinic acid
Lead Poisoning
ALA dehydratase
Porphobilinogen
Porphobilinogen deaminase
AIP
Hydroxymethylbilane
Uroporphyrinogen III
Uroporphyrinogen decarboxylase
PCT
Coproporphyrinogen III
Protoporphyrin
Fe 2+
Ferrochelatase
Lead Poisoning
Heme
Hemoglobin
Globin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