Cyanide Poisoning: Pathophysiology and Clinical Manifestation



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Pathophysiology

Inhibits Complex IV of Oxidative Phosphorylation

Inhibited-chains with (4) Fork and Oxidative-Ox Pi badge

The primary mechanism of cyanide toxicity is inhibition of Complex IV (cytochrome c oxidase) in the electron transport chain within mitochondria. By binding ferric iron (Fe3+) in cytochrome a?, cyanide prevents the reduction of oxygen to water, halting oxidative phosphorylation and ATP synthesis. This results in cellular hypoxia despite normal oxygen delivery, leading to rapid energy failure and lactic acidosis.

Forces Cells Into Anaerobic Metabolism

Ant-robe

By blocking oxidative phosphorylation, cyanide prevents aerobic metabolism and forces cells to rely solely on anaerobic glycolysis for ATP production. This process generates lactic acid, resulting in severe lactic (metabolic) acidosis with an elevated anion gap. Despite normal oxygen tension, tissues are unable to utilize oxygen, a state termed histotoxic hypoxia.

Presentation

Exposure to Fire Fumes

Fire Fumes

In industrialized countries, one of the most common causes of cyanide exposure is inhalation of smoke from domestic or industrial fires. Combustion of nitrogen-containing synthetic materials such as plastics, wool, silk, and polyurethane releases cyanide gas, which can be rapidly absorbed through the respiratory tract.

Industrial Exposure

Industrial Machine

Cyanide exposure can occur in various occupational settings, including metal extraction and mining, electroplating, photography, jewelry production, and plastics or rubber manufacturing. Accidental or intentional ingestion of cyanide salts or exposure to sodium nitroprusside infusions in high doses or prolonged use can also cause toxicity.

Headache and Confusion

Head-egg on Confucius

Early central nervous system manifestations include headache, dizziness, anxiety, and confusion, progressing to seizures, coma, or loss of consciousness in severe poisoning. Neurologic symptoms arise from impaired oxidative metabolism and subsequent energy depletion in neurons.

Arrhythmias

Broken Arrhythmia-drum

Cyanide poisoning produces arrhythmias secondary to myocardial hypoxia. Common findings include bradycardia, ventricular tachycardia, ventricular fibrillation, and pulseless electrical activity (PEA). The underlying mechanism is impaired ATP generation and lactic acidosis disrupting cardiac conduction.

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"Cherry Red" Skin

Skin-suit Guy with Cherries

A characteristic feature of cyanide poisoning is “cherry red” discoloration of the skin and mucous membranes, resulting from increased venous oxyhemoglobin. Because tissues cannot extract oxygen, venous blood retains a bright red appearance similar to arterial blood.

Almond Breath/Taste

Almond Breath

Some patients develop a bitter almond odor on the breath or report a similar taste due to cyanide’s volatile compounds. However, not all individuals can detect this odor because of genetic variability in smell perception.

Acute Kidney Injury (AKI)

Acute-sign Kidney Injured

Cyanide toxicity can cause acute kidney injury due to hypoxic tubular injury and accumulation of nephrotoxic metabolites generated during detoxification, primarily thiocyanate. Prolonged hypoperfusion or direct mitochondrial dysfunction within renal tubular cells may contribute to renal impairment.

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