Cyanide Poisoning Diagnosis and Treatment



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Lab Findings

Lactic Acidosis
Lemon-lake-up arrows
Patients with cyanide poisoning will characteristically have an elevated blood lactate, leading to a lactic acidosis. Because cyanide blocks oxidative phosphorylation (the electron transport chain), the peripheral tissues are unable to utilize oxygen and must switch to anaerobic metabolism which produces lactate.

Elevated Venous PO2
Up-arrow-O2 tank-vine
Patients with cyanide poisoning will characteristically have an elevated venous O2 concentration that may be near or at the level of the arterial O2 concentration. This occurs as a result of the peripheral tissue's inability to utilize oxygen, causing the oxygen to remain bound to hemoglobin and returning into venous circulation instead of being unloaded in the peripheral tissues.

Check Carboxyhemoglobin
Car-box-he-man-globe
Patients who are exposed to cyanide via the inhalation route as a result of exposure to products of combustion should have their carboxyhemoglobin concentration checked. This assesses for concomitant carbon monoxide poisoning, which is commonly seen in tandem with cyanide poisoning in patients exposed to toxic fumes.

Management

Sodium Nitrate PLUS Sodium Thiosulfate
Salt-shaker-nitro plus sign sodium-thigh-sulfur match
One of the antidotes for cyanide poisoning consists of the combination of sodium nitrate (also known as amyl nitrate) and sodium thiosulfate. Sodium nitrate acts by converting hemoglobin into methemoglobin, for which cyanide has a higher affinity than it does to complex IV of the electron transport chain. As a result, cyanide will essentially unbind complex IV and instead bind to methemoglobin, forming cyanomethemoglobin. Sodium thiosulfate then converts this into thiocyanate by donating a sulfate group, allowing the bound cyanide to be excreted in the urine.

Methemoglobinemia
metal he-man-globe
Sodium nitrite acts by converting hemoglobin to methemoglobin, to which cyanide binds with higher affinity compared to complex IV of the electron transport chain. While this is intentional and allows for the eventual elimination of cyanide, this may result in methemoglobinemia and all the subsequent undesireable results. Recall that methemoglobin essentially "traps" hemoglobin in the oxidized Fe3+ state, making the unloading of oxygen very difficult. For this reason, hydroxocobalamin is the preferred antidote for cyanide poisoning when it is available.

OR Hydroxocobalamin (First Line)
hydra-cobra-man holding oar and number-one-foam-finger
Another antidote for cyanide poisoning is hydroxocobalamin, a precursor of vitamin B12 which acts by directly binding cyanide and forming cyanocobalamin, which is immediately able to be excreted in the urine. Since hydroxocobalamin does not affect tissue oxygenation and acts rapidly, it is the preferred antidote for treatment of cyanide poisoning.

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