OP25 CYANIDE POISONING DURING COVID -19 PANDEMIC

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Cyanide poisoning is a rare occurrence. Industrial workers without proper PPE has higher risk of cyanide poisoning. We reported four cases of cyanide poisoning caused by inhalation of acetone nitrile that presented with the complaint of shortness of breath.

A 25 year old previously healthy male presented to the emergency department with sudden onset of shortness of breath associated with dizziness since 8 am. He was triaged to respiratory resus in view of ARDS to rule out COVID-19. He came with 3 other co-workers with the same symptoms. All gave a history of exposure to cyanide during transferring acetonitrile from a big bottle to 180 small bottles. On examination, this patient was confused, PR 130, RR: 30/min, SPO2: 98% on room air, BP: 124/64. Other examination was unremarkable. Patient was given I/V fluids and oxygen via face mask.

His condition worsened 30 min later. He became **obtunded** and GCS score dropped to 11, PR 140, RR 34. SPO2; 84% , BP 119/82, high flow oxygen was increased to 15L/min. Patient was intubated as per SARI protocol. Sodium bicarbonate in a dose of 100mEq over 10 min was administered.

A dose of 5g hydroxocobalamin diluted in 200 ml 5% dextrose IV over 30 minutes was administered 3 hours later after the antidote was obtained from Hospital Kuala Lumpur. Patient was admitted to Intensive Care Unit for close observation. This patient was extubated within the next 48 hours.

Depending on its form, cyanide may cause toxicity through inhalation, ingestion, dermal absorption, or parenteral administration. Clinical manifestations and onset vary widely, depending on the dose and route of exposure, and may range from minor upper airway irritation to cardiovascular collapse and death within minutes. Following an acute exposure, organs and tissues with high oxygen consumption are the first and mostly severely affected.

Cyanide inhibits the action of cytochrome oxidase and carbonic anhydrase. Therefore, blockade of the cytochrome oxidase system causes anaerobic metabolism with resultant lactate production and severe metabolic acidosis.

The monotherapy of intravenous hydroxocobalamin should be effective for severe cyanide toxicity, because of its immediate diffusion into the different tissue compartments. Hydroxocobalamin works by binding cyanide and forms the nontoxic cyanocobalamine, which is renally excreted and has lesser side effects.

Our experience shows that prognosis in cynanide toxicity is reasonably good if rapid supportive intervention provided