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Cyanide Intoxication During Burn Inhalational Trauma: The Silent Heroes Living Among us: Case Report

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ABSTRACT

Here we report the case of a 71 years old gentleman, an unsung hero, who in an attempt to save another life became a surviving victim of household fire. The inhalational injury to his lung in the form of dense black ash filling his lungs was managed using mechanical ventilation and repeat bronchoalveolar lavaging with saline during bronchoscopy. Emerging high anion gap lactic acidosis, with poor oxygen extraction, likely due to inhalational cyanide intoxication was managed using the hydroxocobalamin antidote and bypassing the emerging ATP deficit using creatine phosphate. The oxygen diffusion capacity of the lung during the injury was not overly compromised, increased D dimers suggested the development of microthrombi, singularly not explaining the anaerobic cell respiratory phenotype. The rapidly increasing blood lactate levels, reflecting deviation of the oxidative phosphorylation toward anaerobic glycolysis due to respiratory chain complex IV blockade by inhalational cyanide were promptly reversed using creatine phosphate to regenerate high energy ATP and to scavenge cyanide by binding to hydroxycobalamin.

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Abbreviations

ADP: Adenosine Diphosphate
ATP: Adenosine Triphosphate
Acetyl CoA: Acetyl coenzyme A
CO: Carbon Monoxide
Fe: Iron
HCN: Hydrogen Cyanide
Hgb: Hemoglobin
Iv: Intravenous
Leu: Leukocytes
Ly Abs: Lymphocytes Absolute Numbers
Neu: Neutrophils
OH: Hydroxyl
OXPHOS: Oxidative Phosphorylation
paO2: Oxygen Partial Pressure

Main Article

Inhalational injury during fires eventuates upon inhalation of particular matter containing various chemicals and due to heat effect [1]. Carbon monoxide (CO) and hydrogen cyanide (HCN) intoxications are the main culprits, occurring often concomitantly due to low oxygen environmental burning and presence of nitrogen-containing material [2].

The most common reasons for cyanide intoxication are fires, when nitrile-group containing materials such as plastics (such as polyurethane, vinyl) foams or wool burn [3]. Cyanide, upon ingestion or inhalation, once entering the cell, irreversibly binds to Fe3+ in cytochrome c (complex IV), halting de novo mitochondrial ATP production, and the cells switch to anaerobic glycolysis. Cyanide in limited amounts is broken down by liver rhodanese and sodium thiosulphate to form nontoxic thiocyanate. Another source of potential cyanide toxicity is its industrial application. The worldwide production of hydrogen cyanide is between 1.1 to 2.6 tons yearly, and cyanide is processed to produce plastics, amino acids, reagents for rare metal extraction, such as silver and gold mining under strictly controlled conditions. There is some cyanide exposure from automobile exhausts and cigarette smoke. Cyanide reaches the groundwater from the mining and metal industries [4].

Mass intoxications concluding in death were reported from club fires igniting the soundproofing that was polyurethane material. Any structural fire, in which plastic material burns, releases hydrogen cyanide. Cyanide - Zyclone B - was intentionally used in Nazi concentration camp gas chambers, murdering around 1.1 million Jews [5]. Cyanide was the culprit in the largest environmental catastrophe in Europe after Czernobyl, when a contaminated dam of a goldmining company broke free polluting part of the Tisza and Danube rivers exterminating the fauna for two years [6]. In the not so far past HCN has been used as plant growth regulator to produce equal budding of dormant plants and several inhalational exposures were reported in workers exposed to HCN containing growth regulators [7].

The presence of cyanide is acute and short term. The consequence of cyanide toxicity is tissue hypoxia, despite availability of oxygen, due to inhibition of cytochrome c(complex IV) of the oxidative phosphorylation (Oxphos), designed to create energy gradient through proton and electron transport through the inner mitochondrial membrane. It is the place where oxygen is ultimately reduced with the aid of copper and iron atoms accepting electrons to release water.Tissues are disabled from extracting oxygen leading to deep energy depletion, visually to pinkish skin color. Fire burning usually leads to the combination of CO and CN

poisoning [8]. Depending on the severity of cyanide poisoning death can occur within minutes due to total cell energy depletion and cellular hypoxia. The constituents of the electron transport chain pump hydrogen ions to power the ATP synthase for ATP production, and upon cytochrome IV blockade both O2 reduction and ATP synthesis halts. Consequently, pyruvate is disabled from converting to acetyl-CoA entering the mitochondria, instead is converted to low energy resource lactic acid, with measurable high anion gap metabolic acidosis to emerge. Cyanide intoxication does not negatively influence blood oxygen saturation nor partial pressure, but arteriovenous oxygen partial pressure difference is obliterated due to the emerging lack of oxygen extraction by complex IV of mitochondria [9].

CO displaces oxygen from hemoglobin due to 250x higher affinity, but introducing high concentration and high pressure of oxygen displaces CO from hemoglobin and improves tissue hypoxia [10]. More so CO inhibits the oxidoreductase cytochrome P 450, and to a lesser extent inhibits complex IV. In contrast to HCN, with CO intoxication, paO2 levels are maintained, but oxygen saturation is decreased due to the disproportionate binding capacity of Hgb for CO [11,12].

The most favorable antidote for cyanide intoxication in the context of inhalational trauma is hydroxycobalamin, which causes no hypotension and induces no methemoglobinemia, as opposed to sodium nitrite and sodium thiosulfate. Sodium nitrite induces methemoglobin, in which Fe is present in Fe3+ form and binds HCN, while sodium thiosulfate provides sulphur group for liver rhodanes, the enzyme that neutralizes cyanide by converting it to non toxic thiocyanate. Hydroxycobalamin should be administered early based on clinical suspicion before the emergence of end organ failures due to tissue hypoxia. This timing varies based on the severity of intoxication [13,14].

Case Presentation

The 71 old fit patients, with isolated medical history of uncomplicated diabetes mellitus on oral antidiabetics and hypertension on monotherapy. He suffered moderate lung injury due to household fire, an isolated inhalational trauma. Upon admission the patient was conscious, hemodynamically stable, and initially he required low level oxygen support. Presuming the risk of airway swelling and to bronchoscopically dislodge thick and sticky ash, preventive airway intubation and mechanical ventilation with deep sedation was introduced, requiring low dose norepinephrine administration. Initial carbon monoxide level was mildly elevated in arterial blood (10.8%). Timely, predictive intubation is recommended before swelling and airway closure emerges, according to expert opinions during the first 36 hours post injury [13].

Mechanical ventilation did require inspiratory pressures up to 25cm H20, the lungs were mildly noncompliant and inhaled oxygen fraction could be kept below 50%. Upon intubation and deep sedation flexible bronchoscopy was performed, large amounts of black ash diffusely dispersed were encountered and due to heavy adherence to mucosa, partially lavaged using normal saline. Bronchoscopy was repeated three times 24 hourly.

Approximately 12 hours after admission blood gas analysis started exhibiting metabolic lactic acidosis (up to 13mmol/), with worsening anion gap (up to 26.5mmol/l), increased glycemia, and D dimer levels. When lactate level reached 13mmol/l, investigation was initiated to disclose etiology. The blood test showed normal urea, creatinine and hepatic function tests. To exclude arterial thrombosis, whole body CT with three phase contrast was executed, showing Dorso basal lung consolidation. The circumstantial evidence of high anion gap, severe lactic acidosis, and the mechanism of injury suggested cyanide intoxication, shutting down aerobic ATP production and bypassing energy production towards anaerobic route. Cyanide level measurement is lengthy; injury history and basic laboratory investigation is sufficient for substantial suspicion and antidote administration. In severe cases of inhalational injury antidote administration is recommended without laboratory work up, preferentially immediately by emergency medical services [15]. Our treatment began by administering phosphocreatine to enhance ATP recovery and gain time needed for aerobic respiration to recover [16]. Intravenous phosphocreatine was applied, 8 grams daily intravenously (iv) for five days. In the meantime, hydroxycobalamin antidote (Cyanokit) was obtained and 5grs were administered iv during 15 minutes. Ultimately lactate levels normalized. During the emergence of lactic acid metabolic acidosis, blood glucose levels commenced to rise up to 18mmol/l, and corrected using iv short actin insulin therapy. It is likely that the energy deficiency worsened glucose and insulin transfer to cells and utilization in an already predisposed individual. The patient was extubated day 4, and total hospital LOS was 11 days.

Discussion

Hydrogen cyanide toxicity is to be considered during fire and certain industrial accidents. The severity of the toxicity may be mild to fatal, from headache and weakness to suffocation. Diagnosis is based on suspicion and clinical scenarios. Early antidote administration may be a lifesaving decision. Inhalational trauma that initially presents with mild symptoms, may worsen, hence patients require observation and respiratory hygiene. High anion metabolic acidosis with unusually high lactate levels may prompt diagnosis and prevent organ failure. Immediate energy resource administration in the form of phosphocreatine administration, bypassing oxidative phosphorylation promoting ATP regeneration from ADP, during acute stress, when glucose utilization is impaired, may contribute to improved outcome [17]. Using B12 vitamine instead of Cyanokit is not a viable option, because of the lack of free hydroxy (OH) binding cyanide group in the former. During severe intoxication the clinical picture and the laboratory findings may be more complex reflecting trauma, CO intoxication and tissue damage with a severe inflammatory response. The above case presentation due to its relative mildness or moderacy allowed dissecting and demonstrating the cyanide effect on early stages of energy disruption, which due to prompt intervention and its overall moderacy altogether did not lead to major organ dysfunction.

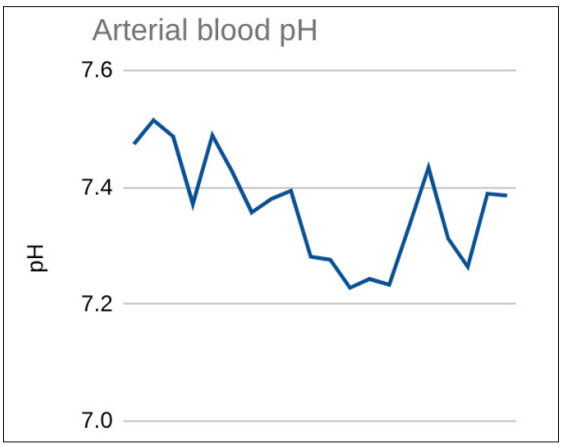

Figure 1: pH Fluctuation During The Demonstrated Observation Period 0-126 Hours Post Admission To IC, Normal pH Range:7.35-7.45

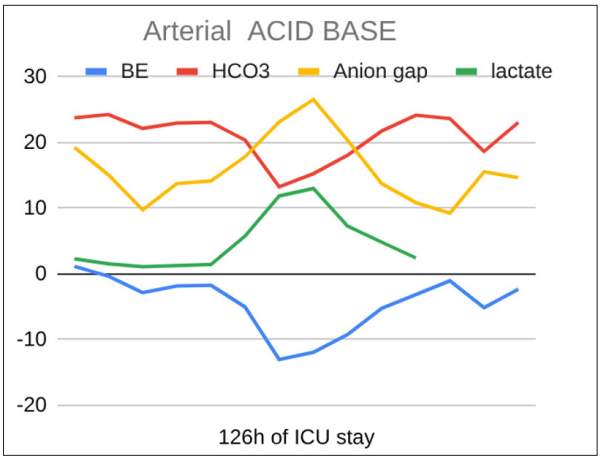

Figure 2: Acid base dynamics, Derangement during the first 126 h after admission to ICU Normal values. Anion gap below 12,BE :+3/-3, Standard HCO3-: 22-28mmol/l, Lactate:0-2mmol/l.

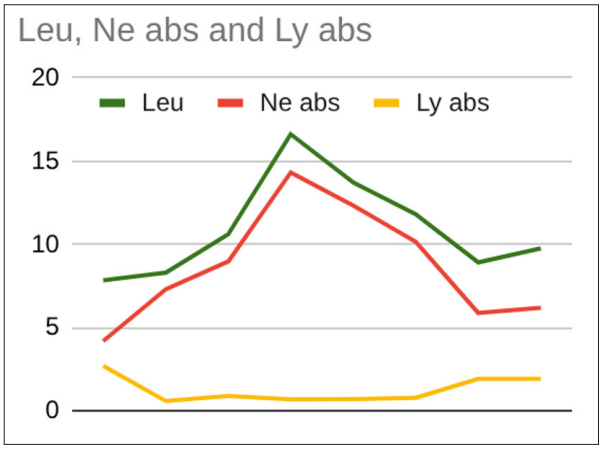

Figure 3: During acute stress leukocyte and neutrophil numbers transiently increase in blood. Normal value: Leukocytes: 4-10*9/l, Neutrophils: 1.5-7.5x10^9/l, Lymphocytes: 1-4.8x10^9/l.

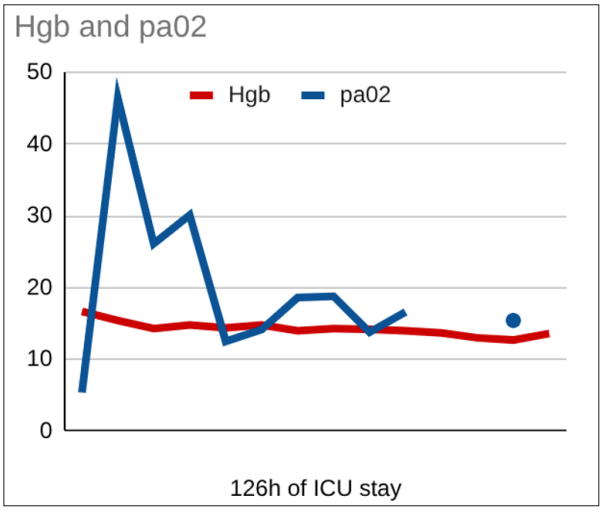

Figure 4: Partial oxygen pressure in blood, kept at high levels to displace CO from hemoglobin, Hemoglobin levels in g/dl during period of observation: Normal values: paO2:10-13.3kPa, Hgb levels:13.2-16.6 g/dl.

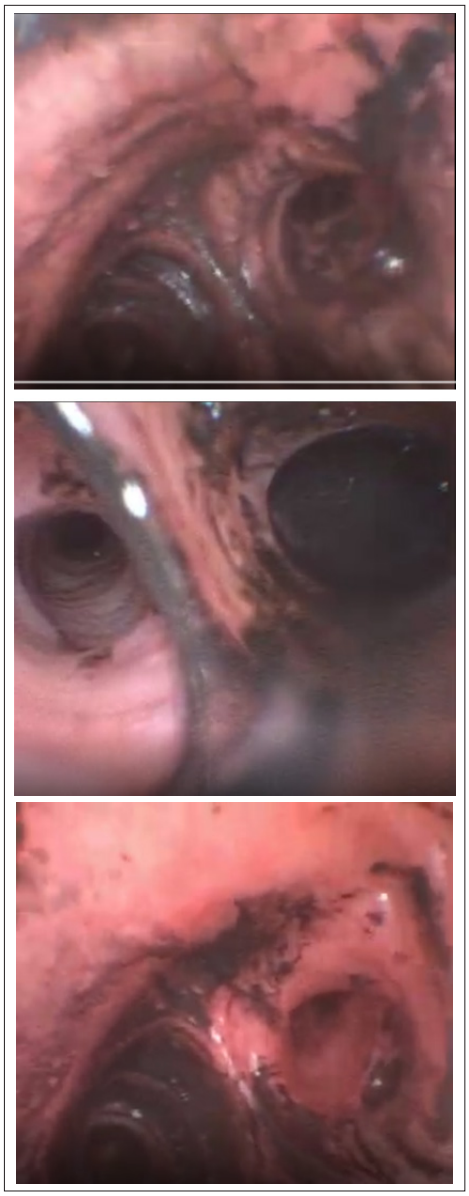

Figure 5: Bronchoscopy depicting thick black patches of ashes covering virtually all segmental bronchi of lung. The photos taken during 2nd bronchoscopy, 24 hours after admission, when soot contamination was already partially cleared by saline lavages, Yet still abundant with emerging hyperemia

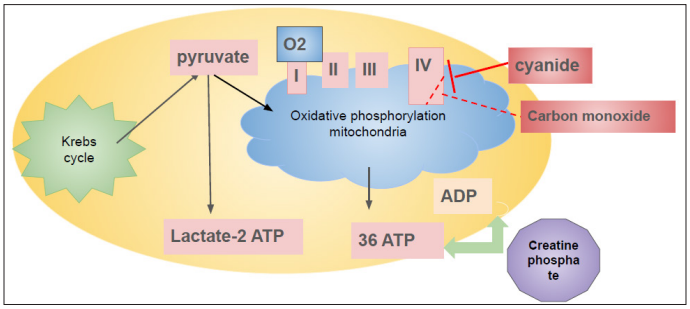

Figure 6: Basic principles of anaerobic and aerobic ATP production, effect of cyanide on complex IV inhibiting mitochondrial electron transport and 36 ATP production, deviating towards anaerobic glycolysis. Cyanide scavenging by OH kobalamin, and bypassing the Oxphos in order to restore ATP using creatine phosphate

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