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Smoke inhalation in children: Focus on management

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Abstract

Smoke inhalation injury is a major determinant of morbidity and mortality in fire victims. We report the case of a 3-year-old child with smoke inhalation, presenting with abnormal consciousness: after a neurological examination, he was diagnosed with severe carbon monoxide and hydrogen cyanide poisoning. Patient was treated with mechanical ventilation, bronchoalveolar lavage, hyperbaric oxygen, and hydroxocobalamin, and then discharged in good clinical condition.

Conclusions: Early diagnosis/treatment of smoke inhalation prevent complications and sequalae.

Keywords: Smoke inhalation; Cyanide; Carbon monoxide; Poisoning; Hyperbaric oxygen.

Introduction

Smoke inhalation is common during fires. It is associated with high mortality, mainly linked to the systemic effects of two asphyxiating components frequently present in inhaled gases: Carbon Monoxide (CO) and Cyanide Acid (HCN) [1]. The management of fire smoke inhalation victims requires a clinical approach to detect poisoning and begin appropriate treatment. We present the case of a child victim of fire smoke inhalation to emphasize principles of a therapeutic approach.

Case study

A three-year-old child and his one-year-old brother were victims of a fire. Civil protection found the charred little brother, but they were only able to save our patient. The evaluation in the emergency room found a child in coma with a Glasgow Coma Scale (GCS) of 6/15, with hypertonic movement of four limbs. His saturation was 88% in room air, along with stridor, soot in the nostrils, upper and lower extremities without skin burns, but hemodynamic status was otherwise normal. The patient was intubated on arrival and started on mechanical ventilation with an FiO_2 of 1. Tracheal aspiration showed the presence of abundant soot. A bronchoscopy was then performed, revealing the presence of soot throughout the bronchial tree, mainly on the right side. The bronchoalveolar lavage allowed for a complete cleaning of the bronchial tree. Initial assessment showed severe metabolic acidosis (pH= 7.10, HCO_3- = 8.4 Meq/L, PCO_2 = 3.6 KPa, PaO_2=8.4 KPa), Carboxyhemoglobin (COHb) of 39%, lactate at 14.5 mmol/L, high liver enzymes, and CK of 750 U/L. At H6 during admission, the child received hyperbaric oxygen therapy. Given the presence of soot in the nostrils, neurological damage, and very high lactates, cyanide poisoning was strongly suspected; two doses of hydroxocobalamin (CYANOKIT) at a dose of 70 mg/kg were administered. Patient blood gas improved (at H10: pH=7.34, PCO_2= 4.5 KPa HCO_3-=22.44 Meq/L, PaO_2= 15.7 KPa), COHb decreased to 8%, lactate gradually became normal, although we noticed a transient increase in (CPK) without impairment of renal function. The child was extubated in 48 hours, as consciousness after a neurological exam gradually returns to normal.

Discussion

This observation illustrates the seriousness of fire smoke poisoning even in the absence of skin burns, including the importance of rapid management based on the pathophysiologi-

cal effects of this type of poisoning. Smoke inhalation results in two types of physiological damage: 1) airway and lung edaema issues related to thermal and chemical damage; and 2) systemic effects of toxic gases such as CO and cyanide. Early fire fatalities are primarily due to hypoxia, along with a lethal synergistic effect of low Oxygen (O_2) levels (with massive consumption of O_2 during combustion) and inhalation of high concentrations of CO and CA (unable to use O_2 in tissue).

Cyanide is a powerful cellular poison as it binds to the ferric iron of cytochrome oxidase, blocking the mitochondrial respiratory chain, thereby causing oxygen deficiency for tissues and lactic acidosis. In the blood, cyanide has a propensity to bind to bivalent iron in hemoglobin, preventing the binding of oxygen to hemoglobin and hindering tissue oxygenation [2,3]. Cyanide poisoning is manifested by asthenia, headaches, confusion, and polypnea. In severe cases, convulsions, coma, apnea, cardiorespiratory arrest, and lactic acidosis is seen. Cyanide poisoning should be suspected in the presence of two of these criteria: signs of neurological impairment (convulsions, coma); haemodynamic disorders; soot in the mouth or sputum and metabolic acidosis, with lactates >8 mmol/l [3]. Our patient had three of them.

Carbon monoxide has an affinity for haemoglobin, which is 250 times higher than for oxygen. It forms a complex called carboxyhaemoglobin, unfit for oxygen transport, and responsible for tissue hypoxia. In the cell, it binds to the myoglobin, ensuring the transport of oxygen to the muscles and heart, blocking the oxidation of cytochrome -C of the mitochondrial respiratory chain, rendering these tissue complexes nonfunctional while causing anaerobic metabolism and lactic acid production. It dissolves in plasma, resulting in systemic toxicity [4]. CO poisoning is manifested by headache, nausea, confusion, and visual disturbances. More severe intoxications are manifested by tachycardia, polypnea, convulsions, coma or even death. A patient is considered poisoned with CO if the CO Hb level is greater than 6. There is no connection between the percentage of carboxyhaemoglobin and the subject's prognosis, as explained by tissue toxicity of CO, though it is poorly assessed by CO Hb, and the difference between oxyhaemoglobin and carboxyhaemoglobin cannot be discerned.

The soot is deposited in the respiratory tree, forming an adherent film with irritants on its surface. They are responsible for bronchopulmonary toxicity with increased alveolocapillary permeability, edaema, and bronchoconstriction – as well as significant heat transfer, resulting in thermal and chemical airway burns, which manifest with significant edaema of the upper airways [6]. Presence of soot in the oropharynx has strong sensitivity and negative predictive value in the diagnosis of CO and CA poisoning. Symptomatic treatment coincides with the administration of antidotes: this includes quick assessment and securing the airways by orotracheal intubation for coma, upper airway obstruction, respiratory distress, or moderate to severe facial burns. Airway patency is threatened over time, as is the maintenance of ventilation and oxygenation.

Progressive edaema (or oedema) results from local lesions, skin burns, and a high volume of vascular filling. It usually increases gradually in the first 72 hours. If endotracheal intubation is delayed, this can cause problems due to edaema that swells the tongue and epiglottis. With initial symptoms, the best strategy is to maintain a low decision threshold for intubation [6].

Oxygen is the first treatment in an emergency: normobaric oxygen therapy with a high concentration mask allows us to treat hypoxia and reverse binding of CO with haemoglobin. The duration of oxygen therapy is poorly defined, but in some studies, oxygen is administered until symptoms disappear, while in others it is indicated until the CO Hb level is between 5 and 10%. The European Committee for Hyperbaric Medicine recommends normobaric oxygen therapy for 12 hours; this oxygen therapy is promoted in cases of severe CO poisoning, with two goals: to increase the amount of oxygen dissolved in plasma, being immediately available to cells, and restoring intra mitochondrial energy levels through eliminating the inhibition of cytochrome a3 with CO, reducing initiation of apoptotic phenomena. This remains controversial, as a Cochrane review could not show any beneficial effect for the myocardium [7]. Yet, it is still recommended by the European Committee for Hyperbaric Medicine for those poisoned with CO at risk of immediate- or long-term complications [8]. For children, signs are severe neurological symptoms, syncope, persistence of symptoms after several hours of normobaric oxygen therapy, myocardial ischaemia, arrhythmias, psychiatric symptoms, elevated CO Hb levels, and lethargy or irritability in children under 6 months [9]. Treatment must be done as soon as possible. Beyond 24 hours, even in the absence of symptoms, HBO is no longer recommended (grade C) [8]: it is contraindication in an undrained pneumothorax. In practice, it is essential to note clinical signs of severe CO poisoning to refer the child to a center with a hyperbaric chamber. For unstable patients, the risk/benefit ratio must be carefully assessed, as the patient must be transferred, which can occur in Tunisia with only one hospital supplying HBO.

Bronchial hygiene consists of therapeutic coughing, respiratory physiotherapy, deep breathing exercises, and airway suctioning. Bronchoscopy with bronchoalveolar lavage to remove soot lining the bronchial tree is also sometimes necessary. Victims of fire smoke inhalation will have more sensitivity loss, such that volume resuscitation may be necessary; however, excessive fluid resuscitation may increase pulmonary microvascular hydrostatic pressure and accentuate edaema formation: fluid resuscitation maintains adequate urine output (greater than 1 ml/kg/h).

Hydroxocobalamin (Cyanokit®, EMD Pharmaceuticals, Durham, NC, USA) is the main cyanide antidote for use in Tunisia. It is fast-acting and dissolves in all areas immediately after administration: it combines with cyanide, which forms cyanocobalamin, but does not interfere with tissue oxygenation, thus improving haemodynamic stability. Studies found a survival rate of 67% for CA concentrations >39 micromol/L [10]. Reported side effects are high blood pressure, red discoloration of the skin, and urine in 28% of cases, with rare urticarial reactions or anaphylactic shock [10]. Early empirical treatment with hydroxocobalamin (70 mg/kg) is recommended for coma, cardiorespiratory arrest, or haemodynamic instability.

Conclusion

Rapid diagnosis is critical, along with immediate treatment to save child victims of fire smoke inhalation. Initial manage-

ment is based on assessment and protection of the airways, treatment of CO and CA poisoning, and respiratory support.

Declarations

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• No conflicts of interest to declare.

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