

Interest of Hyperbaric Oxygen Therapy in Childhood Carbon Monoxide Poisoning: A Retrospective Study

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Authors' contributions

This work was carried out in collaboration among all authors. Author ZI designed the study and wrote the first draft of the manuscript. Authors AR and FZA wrote the protocol and performed the statistical analysis. Authors MC, AK, MS, RA, AH and AA managed the analyses of the study and the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Carbon monoxide poisoning is one of the common causes of poisoning in patients. However, few studies have focused on the pediatric group. We performed this study in order to clarify the clinical characteristics of pediatric patients with Carbon monoxide poisoning.

Methods: We retrospectively reviewed the medical records of pediatric patients (age < 14 years) diagnosed with CO intoxication and admitted in the Hyperbaric Oxygen chamber of the military hospital MOHAMED V of RABAT between January 2018 and March 2020, epidemiologic and clinical data were collected, analyzed and compared with literature.

Results: A total of 112 children with CO poisoning were identified. Their ages ranged from one to 14 years, symptoms were nausea and vomiting (31%), headache (23%), altered consciousness (19%), dizziness (15%) and seizure (8%). Most events (67.8%) occurred during winter, (100%) of cases were recorded as accidental poisoning. Only one child developed delayed neurologic sequelae, with no death enregistered in our series.

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The most common cause of intoxication was improperly vented exhaust produced by gas, hot water heaters (98%).(And) (20%) of patients needed two or more sessions of Hyperbaric Oxygen therapy.

Conclusion: The treatment of Carbon monoxide poisoning is a race against time. Patients requiring Hyperbaric Oxygen therapy must be carefully selected within the first few hours, and kept under close observation. Children with Carbon monoxide poisoning had good outcomes in this series.

Keywords: Carbon monoxide; poisoning; children; neurologic defects.

ABBREVIATIONS

- CO : Carbon Monoxide*
- HbCO : Carboxyhemoglobin*
- DNS : Delayed Neurologic Sequelae*
- HBOT : Hyperbaric Oxygen Therapy*

1. INTRODUCTION

Carbon monoxide (CO) is an important source of poisoning worldwide [1]. In the United States, about 50,000 emergency department visits annually are attributed to CO poisoning, resulting in about 1000-2000 deaths annually. In England, 4000 emergency department visits and 40 deaths are noted annually [2]. Most cases are caused by the incomplete combustion of carbon-containing materials. Carbon monoxide is a toxic, colorless, odorless, tasteless and non-irritating gas, not perceptible by humans [3].

CO intoxication causes tissue hypoxia and direct damage at the cellular level, and can result in a variety of acute symptoms, including headache, seizure, lethargy and coma. Although most victims recover after prompt treatment, some may develop long-term neuropsychiatric deficits [1].

Children may be more vulnerable to carbon monoxide poisoning because of their increased metabolic demand and their inability to vocalize symptoms or recognize a dangerous exposure, and newborn infants are more vulnerable to carbon monoxide poisoning because of the persistence of fetal haemoglobin [4].

The aim of this study was to describe and analyze the causes, clinical characteristics and

benefits of hyperbaric oxygen therapy in CO poisoning in children.

2. METHODS

Our study include all pediatric patients (birth to 14 years old) admitted in Hyperbaric Oxygen chamber of the military hospital MOHAMED V of RABAT with a diagnosis of CO poisoning between January 2018 and March 2020. The diagnosis of COP was made with history of poisoning following exposure to a source of CO.

For each case we recorded The age and sex of the patient, cause of CO exposure and the mechanism (voluntary or accidental), date (month, and season), presenting signs and symptoms, time lapse between CO exposure and admission in the hyperbaric chamber, single or multiple sessions of Hyperbaric Oxygen therapy HBOT, initial Carboxyhemoglobin HbCO levels, and imaging findings if made. Cases with incomplete data, for those who abandoned treatment early were excluded, a total of 112 patients receiving HBOT included in the study.

3. RESULTS

A total of 112 children with CO poisoning were identified. There were 51(45.5%) males and 61 females (55.5 %). The ages ranged from one to 14 years Table 1.

One hundred percent of cases were recorded as accidental poisoning without any suicide attempt. Glasgow Coma Score (GCS) on presentation was 15 for 100 patients (90%) and ≤14 for the remaining 12 (10%). Presenting signs and symptoms included nausea and vomiting (31%), headache (23%), consciousness disturbance (19 %), dizziness (15 %) and seizure (8 %). Fig. 1

Table 1. Age distribution of children with carbon monoxide poisoning

Age (year)	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Number	19	16	9	11	8	9	7	6	2	10	1	9	3	2

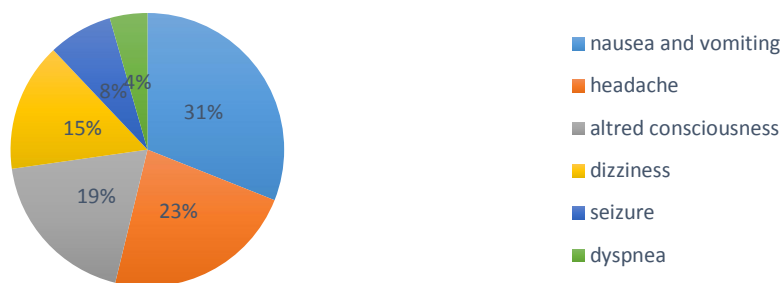


Fig. 1. Symptoms of children with carbon monoxide poisoning

Eight patients (7%) needed intensive care during their acute phases, two of them received mechanical ventilation and one patient presented delayed neurologic sequelae. Seventy patients (62.5%) had initial HbCO levels > 25%.

In 20 patients (17.8%) who underwent electroencephalography (EEG) examination, six had abnormal records.

Eight patients (7.14%) had computed tomography or magnetic resonance imaging examinations, four of them (3.5%) had abnormal findings.

Most events (76 patients, 67.8 %) occurred during winter Table 2.

(60%) were resident in the region of Rabat and presented directly to our hospital, while (40%) were transferred from outside the city, which conditioned the time lapse between CO exposure and admission in the hyperbaric chamber, (90%) within the first 6 hours after exposure Table 3.

A single session of HBOT was administered to 90 patients (80%) and two or more sessions to 22 (20%) Fig. 2. The most common cause of intoxication was improperly vented exhaust

produced by gas hot water heaters 110 (98%), the two remaining cases of CO poisoning were house fires.

4. DISCUSSION

Hyperbaric oxygen therapy increases significantly dissolved oxygen content by the administration of 100% oxygen at pressures two to three times ambient pressure [3].

The purpose of COP treatment is to quickly recover cellular damage and remove the toxic substance by increasing oxygen transport and capacity [5]. The binding of CO to hemoglobin is reversible, and oxygen competes with CO to bind to it [6]. The half-life of CO is 5 hours at room temperature while it is only 90 minutes during normobaric oxygen therapy and 25 minutes during HBOT at 3 atmospheres. This duration is important, particularly for its cardiac effect [7] the recovery period from symptoms is shortened, which reduces long-term neurological complications. As far as possible, HBOT should be administered within the first 6 hours, (90%) in our study. In unconscious patients, HBOT should certainly be the first choice. Its effect is to block the hypoxia induced by CO in the cells [5].

Table 2. Month and season distribution of children with carbon monoxide poisoning

Season	Winter			Spring			Summer			Autumn		
	Jan	Feb	Mar	April	May	June	July	Aug	Sep	Oct	Nov	Dec
	19	32	25	2	5	2	1	1	0	1	10	14

Table 3. Time lapse between CO exposure and HBOT

Lapse (hour)	1	2	3	4	5	6	7	8 OR more
	0	10	38	36	17	7	2	2

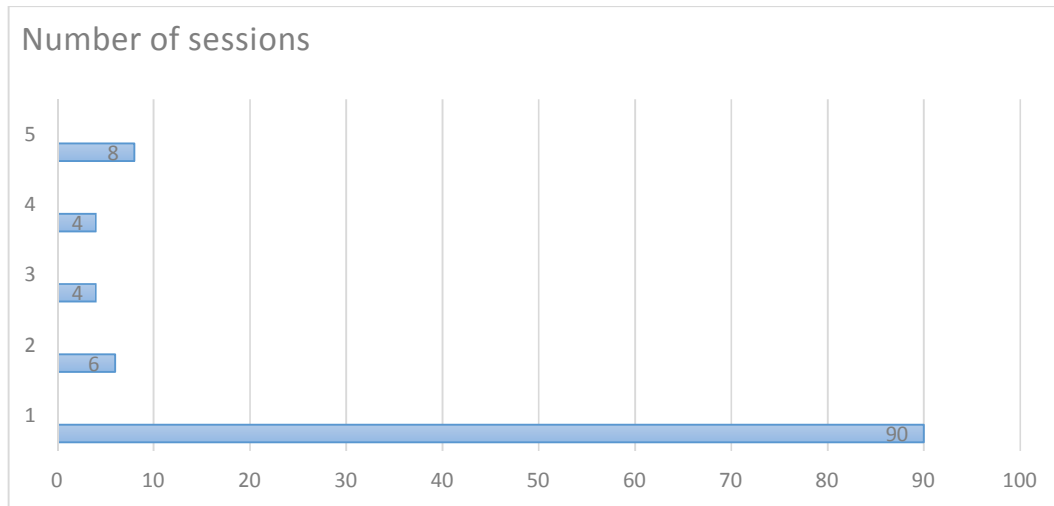


Fig. 2. Number of hyperbaric oxygen therapy sessions

In our series there was no death, the overall case-fatality rate for CO poisoning was reported to be as high as 30%. However, most previous studies enrolled patients with CO poisoning from all causes, including those from fire accidents. In addition to the toxic effects of CO, fire accidents tend to be accompanied by various morbidities, including burn injury, massive fluid loss, cardiac insults, inhalation injury, and hydrogen cyanide intoxication. Thus, the morbidity and mortality rate would be overestimated. [2-8-9].

The causes of COP vary considerably from country to country and from season to season [6]. Suicide is the leading cause of COP in the United States, followed by accidental poisoning [5]. In the present study the most common cause of intoxication was a poorly ventilated exhaust produced by gas water heaters 110 (98%), and just two cases were house fires. Most causes of pediatric CO poisoning are non-intentional, which might explain the lower mortality rate in the studies including only children than in adult ones [2].

Intoxicated children may show a variety of nonspecific symptoms, such as vomiting, headache or dyspnea, and other symptoms, such as dizziness, patient symptoms and findings in the current study were similar to those reported in other studies. [10-11-12].

No relationship was found between COHb levels and clinical outcomes at the time of presentation of the COP. [13-14-15], only Keles, et al. reported that there was a relationship between

the severity of neurological signs and the level of COHb [14].

Delayed onset of neuropsychiatric syndrome is a distinctive feature in victims of CO intoxication. Its pathophysiology is uncertain, but several mechanisms have been proposed, including postischemic reperfusion injury, the effects of CO on the vascular endothelium and oxygen-radical-mediated brain lipid peroxygenation, and nitric oxide-related central nervous system damage [13]. DNS was estimated to occur in 10–40% of adult survivors of CO intoxication, and was usually noted after lucid periods, children have a much lower incidence of DNS than adults, with incidences of 2 to 10% reported in previously published pediatric studies [16-17]. Raphael et al. observed that the development of DNS was more common in patients with a history of consciousness disturbance, than in those without consciousness disturbance [18].

Certain tissues, such as the hippocampus and basal ganglia, are most commonly injured because of their higher sensitivity to ischemic injury [2]. In our study 8 patients (7%) underwent brain imaging studies because of persistent neurological symptoms. Four (3.5%) patients had typical hypoperfusion changes over the basal ganglia. Of 20 (17.8%) patients who underwent EEG examination, six (5.3%) had pathological results, including four with focal epileptiform discharges.

Poor general status on presentation, being unconscious, cardiac arrest, coma, metabolic

acidosis and a high COHb level increase mortality [15]. HBO treatment should be planned according to the clinical status of the patient and not only on the basis of COHb level. If the patient shows any of these problems, HBOT is necessary [5].

5. CONCLUSION

The treatment of COP is a race against time. Many effects of CO resemble those associated with various acute illnesses in childhood, making it easy to overlook the possibility of CO intoxication. Patients needing HBOT must be carefully selected within the first few hours, and kept under close observation. Late symptoms and signs such as neurological sequelae can be avoided by early diagnosis and rapid decision to administer HBOT.

CONSENT

It is not applicable.

ETHICAL APPROVAL

This study was carried out after authorization from the departments concerned and the Hospital. The study guaranteed the confidentiality of our data which was collected and processed in absolute anonymity.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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