© Pediatr Med Rodz 2019, 15 (2), p. 157–163
DOI: 10.15557/PiMR.2019.0027

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Received: 14.01.2019
Accepted: 07.02.2019
Published: 31.07.2019

Assessment of hyperbaric oxygen efficacy in children with carbon monoxide poisoning

Ocena skuteczności tlenoterapii hiperbarycznej u dzieci po zatruciu tlenkiem węgla

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Abstract

Carbon monoxide (CO) poisoning is an important clinical problem. The symptoms of poisoning are non-specific, particularly in the paediatric population. Currently, the use of hyperbaric oxygen (HBO) is considered to be more justified with increasing severity of the patient's clinical status and with shorter time elapsed from CO exposure. **Aim of the study:** The aim of this study was to assess the efficacy of hyperbaric oxygen therapy in children with symptoms of carbon monoxide poisoning. **Material and methods:** The study was a retrospective analysis of medical records of children hospitalised for carbon monoxide poisoning at the Department of Paediatrics, Paediatric Nephrology and Allergology of the Military Institute of Medicine, Poland, between January and December 2018. The following data were analysed: laboratory test results, CO poisoning circumstances, the manner of reporting to the Department, the number of hyperbaric chamber sessions needed to relieve the symptoms completely, the occurrence of complications and the duration of stay at the Department after treatment completion. **Results:** The analysis of the results did not demonstrate any relationship between carboxyhaemoglobin level and poisoning manifestations and CO poisoning severity. However, a statistically significant positive correlation was demonstrated between carboxyhaemoglobin level and HCO_3– level and a statistically significant negative correlation was found between carboxyhaemoglobin level and capillary pH. No statistically significant differences were found between children with mild poisoning and those with severe poisoning in terms of potassium level, capillary pH, and HCO_3– and glucose levels. At the same time, it was demonstrated that hyperbaric therapy is a highly effective and safe method. **Conclusion:** Regardless of the causes of CO poisoning in the paediatric population, hyperbaric therapy is a highly effective and safe method. One session in a hyperbaric chamber is sufficient to relieve the symptoms completely and prevent delayed sequelae of poisoning and CO side effects.

Keywords: hyperbaric therapy, carbon monoxide poisoning (CO), children, carboxyhaemoglobin, hyperbaric chamber

Streszczenie

Zatrucie tlenkiem węgla (CO) stanowi istotny problem kliniczny. Objawy zatrucia są niespecyficzne, zwłaszcza w populacji dziecięcej. Obecnie uznaje się tym większą zasadność zastosowania tlenoterapii hiperbarycznej (*hyperbaric oxygen*, HBO), im cięższy jest stan kliniczny pacjenta i im krótszy czas upłynął od momentu przerwania ekspozycji na CO. **Cel pracy:** Celem niniejszej pracy była ocena skuteczności leczenia tlenem hiperbarycznym dzieci z objawami zatrucia tlenkiem węgla. **Materiał i metody:** Badanie zostało przeprowadzone jako analiza retrospektywna dokumentacji medycznej dzieci hospitalizowanych z powodu zatrucia tlenkiem węgla w Klinice Pediatrii, Nefrologii i Alergologii Dziecięcej Wojskowego Instytutu Medycznego w okresie od stycznia do grudnia 2018 roku. Analizie poddano informacje dotyczące wyników badań laboratoryjnych oraz okoliczności zatrucia CO, sposobu zgłoszenia się do Kliniki, liczby sprężeń w komorze hiperbarycznej niezbędnych do całkowitego ustąpienia objawów, występowania powikłań oraz okresu pobytu w Klinice po zakończonym leczeniu. **Wyniki:** Przeprowadzona analiza wyników nie wykazała związku między stężeniem karboksyhemoglobiny a prezentowanymi objawami zatrucia oraz stopniem ciężkości zatrucia CO. Wykazano natomiast istotną statystycznie dodatnią korelację pomiędzy stężeniem karboksyhemoglobiny a stężeniem HCO_3– oraz ujemną pomiędzy stężeniem karboksyhemoglobiny a pH krwi włośniczkowej. Nie odnotowano istotnych statystycznie różnic między grupą dzieci z łagodnym zatruciem i z ciężkim zatruciem w zakresie wartości stężenia potasu, pH krwi włośniczkowej, stężeń HCO_3– oraz glukozy. Jednocześnie wykazano, że terapia hiperbaryczna cechuje się wysoką skutecznością i dużym bezpieczeństwem. **Podsumowanie:** Niezależnie od przyczyn zatrucia CO w populacji dziecięcej terapia hiperbaryczna jest metodą wysoce skuteczną i bezpieczną. Jeden zabieg w komorze hiperbarycznej pozwala uzyskać całkowite ustąpienie objawów i zapobiega późnym następstwom zatrucia czy efektom ubocznym działania CO.

Słowa kluczowe: hiperbaria, zatrucie tlenkiem węgla (CO), dzieci, karboksyhemoglobina, komora hiperbaryczna

INTRODUCTION

Carbon monoxide (CO) is the most common agent in inhalation poisonings globally[1]. According to the Polish State Fire Service data, during the 2017/2018 heating season in Poland there were 4,343 incidents caused by the harmful effects of CO in which 2,659 individuals were affected and 71 persons died[2]. CO poisonings occur mainly in the autumn and winter. They are usually associated with faulty heating equipment and ventilation systems; they are also found in individuals evacuated from burning buildings.

Carbon monoxide is a colourless, odourless and tasteless gas. It is formed as a result of incomplete combustion of hydrocarbon-containing products. It is produced by the human body as a byproduct of haemoglobin degradation. Under physiological conditions CO is present in non-smokers at a 1–3% concentration and its level can be as high as 10–15% in smokers[3]. Carbon monoxide enters the body through the airway and, to a lesser extent, through the skin and mucous membranes, although the percutaneous route does not have a significant effect on the course of intoxication. Carbon monoxide is removed from the body predominantly through the respiratory tract as CO; only 1% is oxidised to carbon dioxide (CO_2). The amount of CO absorbed by the human body depends on respiratory minute volume, the degree of alveolar-capillary diffusion, the duration of exposure and ambient CO concentration. Carbon monoxide is characterised by 250–300-stronger affinity for haemoglobin than oxygen and forms carboxyhaemoglobin (COHb) with it. The rate of CO binding with haemoglobin is high at the initial phase of exposure and rises until an equilibrium is achieved between blood COHb level and CO concentration in the polluted air[1,4].

The clinical manifestation of CO poisoning may be non-specific[4–6]. Cells with the relatively highest metabolic activity and, consequently, substantial perfusion, are at the highest risk of CO toxicity. Therefore, cardiomyocytes and central nervous system cells are most often affected. Patients complain of headache, dizziness, nausea, emotional instability and confusion; in more serious cases, poisoned individuals lose consciousness. In infants frequent vomiting may be mistaken for gastrointestinal infection[3]. CO exposure may result in muscle damage, including myocardial damage. Cardiac myoglobin binds CO three times more strongly than skeletal myoglobin; this is manifested on electrocardiogram as ST segment depression, atrial fibrillation or ventricular arrhythmia. As a result of skeletal muscle damage and breakdown, myoglobinuria may occur; acute renal failure associated with rhabdomyolysis may even develop. There were cases in which myotonia developed in individuals poisoned with CO who were still conscious, which sometimes made it difficult or impossible for them to leave the room. Other reported effects of CO poisoning include solid organ congestion and disseminated intravascular coagulation[1,4,7].

The most important element of a rescue operation is to leave the rooms where CO exposure occurred. The victims should receive 100% oxygen therapy using an oxygen mask with a reservoir bag or a self-inflating bag for supported ventilation. Ventilating the patient with 100% oxygen increases the gradient of partial oxygen pressures in the alveolar air and pulmonary capillary blood, which makes it possible to reduce the mean COHb half-life from approximately 5–6 hours for atmospheric oxygen breathing to 30–90 minutes. Currently, the recommended standard is to use hyperbaric oxygen therapy[4,6,8]. The more severe the patient's clinical status and the shorter the time elapsed from CO exposure are, the more justified hyperbaric oxygen therapy is.

For adult patients, the accepted threshold of indication for hyperbaric oxygen therapy for CO poisoning is a COHb level above 16 or the presence of clinical symptoms. In the paediatric population, as in geriatric patients, the criteria depend on the observed clinical symptoms, while COHb has exclusively prognostic significance[4,6,9].

The very idea of using a hyperbaric method originated in the 17th century. In 1662 Nathaniel Henshaw was the first to construct a hyperbaric chamber that was used as a spa treatment rather than a caisson or a diving bell. In the 1930s Edgar End was the first to attempt to treat CO poisoning in a hyperbaric chamber; however, this method was documented only in 1960 by Smith and Sharp[4,10]. Since that time, the design of the hyperbaric chamber has been improved and it is being applied in new fields of medicine.

AIM OF THE STUDY

The aim of this study was to assess the efficacy of hyperbaric oxygen therapy in children with symptoms of CO poisoning.

MATERIAL AND METHODS

The study was a retrospective analysis of medical records of children hospitalised for CO poisoning at the Department of Paediatrics, Paediatric Nephrology and Allergology of the Military Institute of Medicine, Poland, between January and December 2018.

The study group included 17 children: 9 girls (53%) and 8 boys (47%) with CO poisoning. The mean age of the study group was 9 ± 5 years.

The study group of children with symptoms of CO poisoning was divided into three categories depending on the severity of the clinical presentation (Tab. 1):

- mild poisoning;
- moderately severe poisoning;
- severe poisoning.

The classification into a given category depended on the sum of points assigned to different symptoms (Tab. 2).

DOI: 10.15557/PiMR.2019.0027

Severity of poisoning	Total score
Mild	0–3
Moderately severe	4–6
Severe	>6

Tab. 1. Clinical division of CO poisoning

Symptom	Number of points
No complaints	0
Headache/dizziness	1
Muscle pain	1
Nausea/vomiting	1
Chest pain	2
Disorientation/confusion	2
Collapse	4
Confusion/loss of consciousness	7

Tab. 2. Number of points for different symptoms

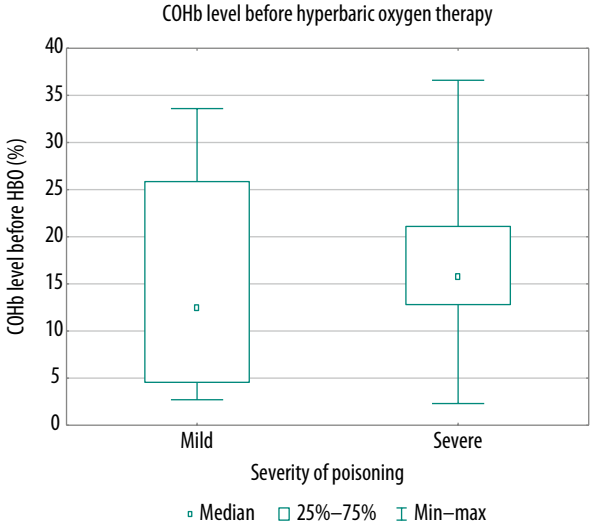

Fig. 1. COHb concentration on admission to the Department in children with mild and severe poisoning symptoms

Laboratory test results (serum potassium and glucose levels, COHb concentration and capillary blood gas test: pH and HCO_3^-), the circumstances of CO poisoning and the manner of reporting to the Department due to symptoms of poisoning were analysed in detail. The number of treatment sessions necessary for the symptoms to subside completely, the presence of complications and the duration of stay at the Department after the end of the treatment were also recorded. In a standard hyperbaric chamber therapy protocol, pure oxygen under the pressure of 2.5 atmospheres absolute (ATA) is utilised. Oxygen is administered through an individual breathing mask if the patient is conscious or through a hyperbaric respirator adapted to an increased density of the breathing gas in unconscious patients[4,6,7,9,11].

Statistical analysis was performed on the results using the StatSoft, Inc. (2014) STATISTICA version 12 data analysis software system. Before commencing the analysis, the data were initially verified with a data normality graph and finally with the Kolmogorov–Smirnov and Lilliefors normality tests. Due to the non-normality of some of the variables, non-parametric tests, which do not require normal distribution, were used for the statistical analysis of them. The Student's *t*-test was used to assess variables with a normal distribution. In correlation analysis, Spearman's rank correlation coefficient was calculated for non-normal variables and the Pearson's linear correlation coefficient was determined for variables with a normal distribution. Results with $p < 0.05$ were considered statistically significant.

RESULTS

The medical records of 17 children with CO poisoning treated at the Department of Paediatrics, Paediatric Nephrology and Allergology of the Military Institute of Medicine, Poland, including 9 girls (53%) and 8 boys (47%) were analysed. The mean age of the study group was 9 ± 5 years.

The main cause of poisoning was a faulty gas heater in the bathroom (14 children). Other causes included boiler failure in the boiler room (2 children) and fire in the household (1 child). All children were carried to the Department using medical transport, 8 with mild poisoning symptoms, 1 with moderately severe poisoning and 8 with severe poisoning symptoms.

Median COHb percentage upon reporting to the Department was 16.4% (q_{25} – 7%, q_{75} – 21%). No statistically significant differences were found in COHb values between children with symptoms of mild poisoning and those with severe poisoning (12.4%, q_{25} – 4.5, q_{75} – 25.8 vs. 15.7%, q_{25} – 12.8, q_{75} – 21, respectively) (Fig. 1).

Parameter	Mild poisoning, median (q_{25}–q_{75})	Severe poisoning, median (q_{25}–q_{75})	*p*
K^+ [mmol/l]	4.2 (4.1–4.4)	4.1 (4.0–4.3)	ns.
pH	7.40 (7.4–7.4)	7.41 (7.39–7.42)	ns.
HCO_3– [mmol/l]	25.1 (22.0–26.6)	25.3 (23.4–25.7)	ns.
Glucose [mg/dl]	92 (80–106)	102 (93–113)	ns.
ns. – not statistically significant.			

Tab. 3. Analysed biochemical parameters in children with symptoms of mild and severe poisoning

Parameter	Correlation coefficient	*p*
Duration of hospital stay vs. age	0.05	ns.
Duration of hospital stay vs. COHb	–0.29	ns.
Duration of hospital stay vs. pH	0.0	ns.
Duration of hospital stay vs. HCO_3–	–0.33	ns.
Duration of hospital stay vs. glucose	0.48	ns.
Duration of hospital stay vs. K^+	–0.33	ns.
ns. – not statistically significant.		

Tab. 4. Coefficients of correlation between the duration of hospital stay and the child's COHb level and biochemical markers

 DOI: 10.15557/PiMR.2019.0027

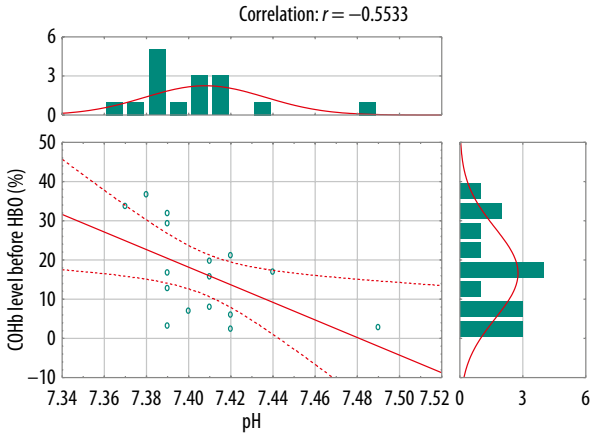

Fig. 2. Correlation between COHb level and capillary pH

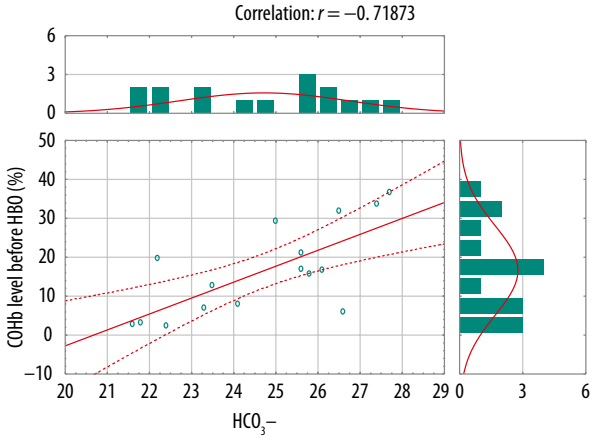

Fig. 3. Correlation between COHb and HCO_3– levels

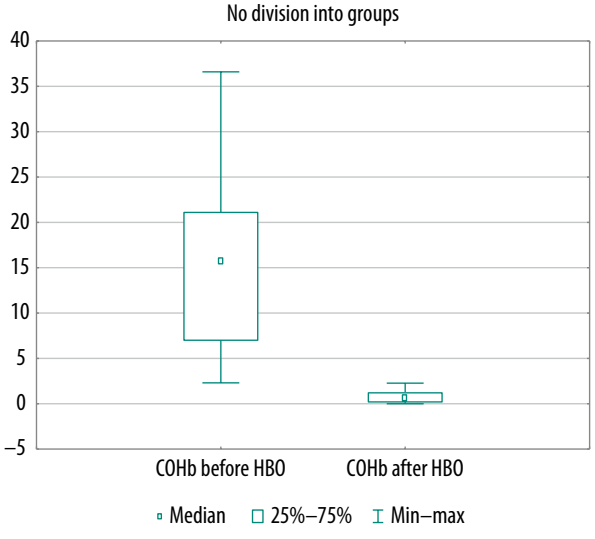

Fig. 4. Percentile COHb values before and after hyperbaric therapy in the whole study group

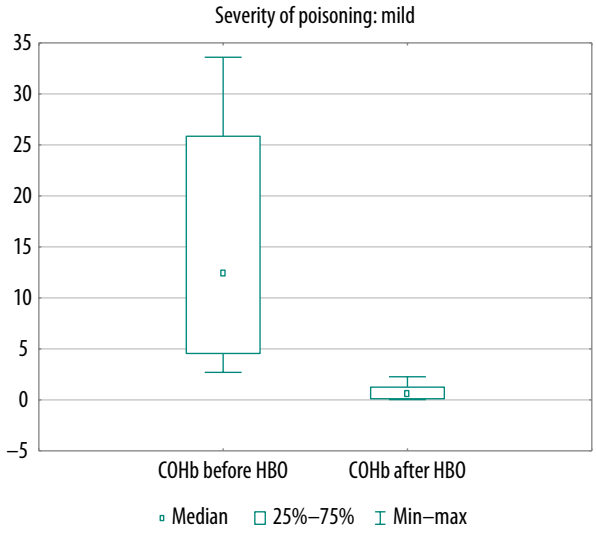

Fig. 5. Percentile COHb values before and after hyperbaric therapy in the group of children with mild poisoning symptoms

In addition, no statistically significant differences were found between children with mild poisoning and those with severe poisoning in terms of potassium level, capillary pH, and HCO_3^- and glucose levels (Tab. 3).

However, a statistically significant positive correlation was demonstrated between COHb level and HCO_3^- level and a statistically significant negative correlation was found between COHb level and capillary pH (Figs. 2, 3).

In all children, regardless of the baseline COHb level and severity of poisoning, one session in a hyperbaric chamber was sufficient to achieve normal COHb values (Figs. 4–6).

The mean duration of stay at the Department was 2 ± 3 days. One child required longer hospitalisation (12 days) in whom CO poisoning was complicated with pneumonia. No correlation was found between the duration of hospital

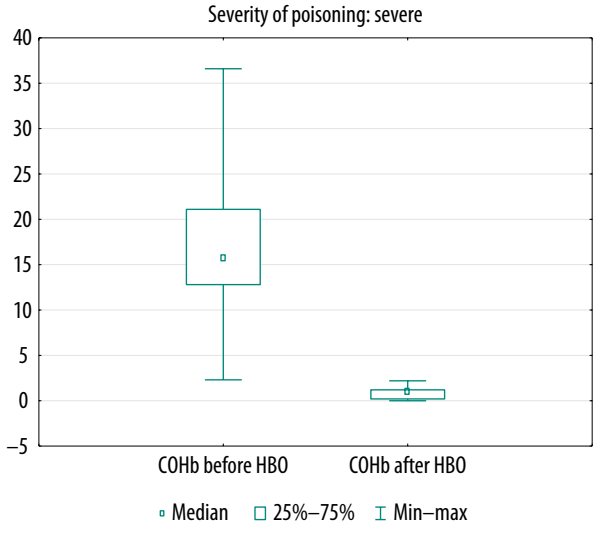

Fig. 6. Percentile COHb values before and after hyperbaric therapy in the group of children with severe poisoning symptoms

DOI: 10.15557/PiMR.2019.0027

stay and the child's age, baseline COHb level and the analysed biochemical parameters (Tab. 4).
No adverse reactions to hyperbaric oxygen therapy were observed in any of the children.

DISCUSSION

Carbon monoxide poisoning is an important clinical problem in the paediatric population[12,13]. CO poisonings occur most frequently due to faulty heating systems, particularly gas heaters, in the autumn and winter. Less common causes include fires (forest, household fires) and suicide attempts[14,15]. The aetiology and pathophysiology of CO intoxication, and diagnostic and therapeutic options have been investigated by the scientific community for years.
When CO poisoning is suspected, it is important to take a thorough medical history quickly and determine whether similar symptoms are present in the individuals in the patient's immediate environment[1].
The most sensitive indicator of CO poisoning is elevated COHb level in capillary blood. Carbon monoxide poisoning is confirmed in adults based on elevated serum COHb values: >3% in non-smokers and >10% in smokers. However, COHb level does not allow one to estimate the severity of poisoning. In children, it is very difficult to confirm the diagnosis based on COHb level; therefore, in paediatrics, clinical symptoms are assumed to be the primary indication for treatment. CO poisoning can be classified into three categories depending on the severity of clinical symptoms: mild (COHb level of 10–30%), moderately severe (30–50%) and severe (50–80%). When CO poisoning is suspected, the use of pulse oximetry is not recommended, since the device does not distinguish between oxyhaemoglobin and COHb and a normal saturation result does not exclude CO poisoning. Other important laboratory parameters include arterial blood gas profile, lactate levels as an indicator of the degree of anaerobic metabolism, and troponin and creatine kinase MB isoenzyme (creatine kinase muscle-brain, CKMB) levels[4,6,7,11]. The results obtained in this study indicate a lack of correlation between COHb level and the presented symptoms of poisoning and no association with the severity of CO poisoning.
However, the analysis found a link between COHb level and the risk of metabolic acidosis: the higher the COHb level, the higher the risk of metabolic acidosis.
Acute CO poisoning usually leads to neurological disorders and/or myocardial dysfunction[12–14,16,17]. Delayed sequelae of poisoning, mainly in the form of neurological disorders, are observed in approximately 10% of patients. Clinical observations and scientific studies on animals conducted globally have inspired a renewed attempt to analyse the influence of CO on the human body, with a focus on the pathophysiology of this gas in a living body[16,18].
The symptoms of CO poisoning are the effect of two toxicity mechanisms. The first one involves oxygen supply (sO_2) restriction, defined as a decreased amount of oxygen supplied to the patient's tissues in a unit of time, which is calculated using the following formula:

$$sO_2 = (HR \times SV) \times (Hgb \times SaO_2 \times 1.34) + PaO_2 \times 0.003$$

where:
- HR – heart rate;
- SV – heart stroke volume;
- Hgb – blood haemoglobin level;
- SaO_2 – arterial oxygen saturation;
- PaO_2 – partial arterial oxygen pressure.

The formation of COHb complexes causes a reduction in oxygen supply, which is inversely proportional to COHb level. This is due to the fact that the concentration of free haemoglobin that could bind oxygen molecules is decreased. This CO poisoning effect leads to COHb level- and half-life-dependent hypoxaemic or anoxaemic changes, particularly in the central nervous system. This mechanism is usually responsible for the spectrum of acute CO poisoning symptoms, which are a life-threatening medical emergency.
The second mechanism of CO toxicity is associated with the effects of CO on cytochrome oxidase complexes. The disruption of mitochondrial function results in the release of reactive oxygen species and increase in oxidative stress at the cellular level. The release of nitric oxide from platelets and endothelial cells increases the formation of oxygen radicals, which may result in damage to vascular endothelial cells[7]. Damage to vascular endothelial cells produces particularly severe symptoms in the brain[9,11,19]. The final effect of this process is lipid peroxidation in the brain. In addition, oxidative damage is exacerbated by cerebral reperfusion and leukocyte adhesion. This process starts on recovery from CO poisoning and results in cognitive impairment, particularly in terms of memory and learning, and in motor dysfunction, which may not produce symptoms during the first days after poisoning[3]. Neonates are more susceptible to CO effects due to the presence of foetal haemoglobin in their blood, which binds twice as much CO as regular haemoglobin. Furthermore, children are more sensitive to the negative effects of CO than adults due to higher respiration and metabolism rates[1,4,6,12,14,20].
Based on current knowledge, patients with the symptoms of severe poisoning (loss of consciousness or neurological dysfunction, circulatory instability or haemodynamic disturbances) should be first referred for hyperbaric oxygen (HBO) therapy[12,17]. Hyperbaric chamber sessions cause a reduction in intracranial pressure by reducing the development of hypoxic brain oedema, which is dependent on oxygen pressure and the hyperbaric chamber pressure[4,17,21]. Regardless of the baseline clinical status and serum COHb level, every patient who has lost consciousness should undergo obligatory hyperbaric oxygen therapy[12,22]. Based on the considerations above, the clinical assessment of a child with CO poisoning should not be based exclusively on COHb concentration, since it does not allow one to

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predict the severity of CO poisoning and its possible sequelae. Because of the lack of clear association between the COHb level and clinical symptoms, every case of CO poisoning in a child should be treated as a life-threatening medical emergency.

Due to the similarity of certain CO poisoning symptoms to acute childhood diseases, the diagnosis of CO poisoning in the paediatric population is difficult. There is also a high risk of overlooking CO intoxication in this age group. Despite the fact that the mortality rate for CO poisoning is lower in paediatric patients than in the adult population, significant delayed neurological sequelae are much more common in children than in adults (epilepsy, cognitive deficits)[23]. Due to the negative effect of CO poisoning on the body of a foetus, neonate and infant, there should be separate recommendations for HBO in children[13–15,22]. The number of publications on the efficacy of hyperbaric oxygen therapy in children with CO poisoning is still small compared to the rich literature on the subject in the adult population[13,15,23]. Hyperbaric oxygen was adopted as a CO poisoning treatment method in adults already long ago. To date, there have been no clear guidelines regarding the use of HBO in paediatric patients[16,23]. Based on metanalyses, an advantage of hyperbaric oxygenation over normobaric oxygenation was demonstrated in preventing delayed neurological sequelae (i.e. headache, memory impairment, difficulty concentrating, sleep disorders) in patients with CO poisoning[16,24,25]. Research has also demonstrated high efficacy of hyperbaric oxygen therapy in patients with severe CO poisoning in whom symptoms completely subsided already after one HBO session. In the present study, this effect was confirmed. These data translate to measurable economic benefits associated with reduced costs of further medical care on patients with complications after CO exposure[16,18,24,26,27]. Studies confirm not only the high efficacy of the method used, but also its safety for both adults and children[22,24].

The results obtained in this study provide a clear indication of the efficacy of hyperbaric oxygen therapy. Apart from high efficacy, this method is characterised by high safety. In patients who underwent HBO therapy, apart from one case complicated with pneumonia, no delayed sequelae of CO poisoning were observed.

CONCLUSION

Regardless of the causes of CO poisoning in the paediatric population, hyperbaric therapy is a highly effective and safe method. Regardless of the severity of poisoning, one hyperbaric chamber session is sufficient to achieve complete recovery from symptoms, thus preventing delayed sequelae of poisoning or CO side effects.

The analysis of the number and severity of cases of children with CO poisoning hospitalised at the Department of Paediatrics, Paediatric Nephrology and Allergology of the Military Institute of Medicine and treated at the Department of Hyperbaric Medicine shows the need for close cooperation between paediatricians and hyperbaric medicine and intensive care specialists in order to closely monitor patients with CO poisoning and provide them with the best possible treatment.

Conflict of interest

The authors do not report any financial or personal affiliations to persons or organisations that could adversely affect the content of or claim to have rights to this publication.

DOI: 10.15557/PiMR.2019.0027 PEDIATR MED RODZ Vol. 15 No. 2, p. 157–163

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