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Hyperbaric oxygen therapy in carbon monoxide intoxication – case descriptions

Tlenoterapia hiperbaryczna w zatruciu tlenkiem węgla – opis przypadków

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S u m m a r y

Carbon monoxide (CO) is a toxic gas which combines with hemoglobin over 250 times stronger than oxygen thus disabling hemoglobin to transport oxygen in the organism. Secondary hypoxia negatively affects the functioning of the circulatory system and the central nervous system. The symptoms of intoxication start occurring together with the rise of the level of carboxyhemoglobin (COHb). In case of carbon monoxide intoxication the treatment consists in the administration of 100% oxygen in normal conditions or under increased pressure in the hyperbaric chamber.

The present work presents the cases of eight persons who experienced accidental CO intoxication. The authors have described the specificity of the intoxications, the principles of pre-hospital procedure and the application of hyperbaric oxygen therapy in the treatment of severe carbon monoxide intoxications. Attention has been drawn to the existing possibilities of determining the level of carboxyhemoglobin in pre-hospital conditions which – together with unitary criteria qualifying for hyperbaric therapy – would allow for the shortening of the time in which the patient could be transported to a facility possessing specialized treatment equipment.

S t r e s z c z e n i e

Tlenek węgla (CO) jest toksycznym gazem, który łączy się z hemoglobiną przeszło 250 razy silniej niż tlen, uniemożliwiając jej tym samym transport tlenu w organizmie. Wtórna hipoksja negatywnie wpływa na funkcjonowanie układu krążenia oraz ośrodkowego układu nerwowego. Objawy zatrucia pojawiają się wraz ze wzrostem poziomu karboksyhemoglobiny (COHb). W przypadku zatrucia tlenkiem węgla leczenie polega na podawaniu 100% tlenu w warunkach normalnych lub pod zwiększonym ciśnieniem w komorze hiperbarycznej.

Niniejsza praca przedstawia przypadki ośmiu osób, u których doszło do przypadkowego zatrucia CO. Autorzy opisują specyfikę zatruć, zasady postępowania przedszpitalnego oraz zastosowanie tlenoterapii hiperbarycznej w leczeniu ciężkich zatruć tlenkiem węgla. Zwrócono uwagę na istniejące możliwości oznaczenia poziomu karboksyhemoglobiny w warunkach przedszpitalnych, co wraz z jednolitymi kryteriami kwalifikującymi do terapii hiperbarycznej pozwoliłoby skrócić czas, w jakim pacjent mógłby dotrzeć do ośrodka dysponującego sprzętem pozwalającym na leczenie specjalistyczne.

Conflict of interest
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None
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INTRODUCTION

The proper functioning of the human organism depends on the appropriate supply of cells with energy which is necessary for them to maintain their function. The generation of energy which is the product of food conversion occurs in the presence of oxygen taken from air in breathing processes. The oxygen atmospheric pressure equal 160 mmHg is gradually lowered and in the lungs the oxygen is combined with the hemoglobin of the erythrocytes present in the capillaries of the pulmonary vesicles. Further exchange occurs through the walls of the pulmonary vesicles in accordance with the pressure gradient. Oxygen molecules diffuse from the lumen of the vesicles to blood in accordance with the pressure gradient. The oxygen molecules diffuse into blood due to the fact that in the pulmonary vesicles in the vesicle air the oxygen pressure is PO_2 = 100 mmHg and it is higher than the oxygen pressure in the blood reaching the vesicle which is equal PO_2 = 40 mmHg (1). A significant role in oxygen transportation is played by hemoglobin (Hb) which is a protein composed of two polypeptide α chains and two β chains as well as four iron (Fe) subunits. During the blood circulation in the body hemoglobin releases oxygen which diffuses into the cells and the oxygen pressure slowly decreases. Another important role off hemoglobin is the transportation of carbon dioxide CO_2 from tissues to the lungs (2).

CARBON MONOXIDE INTOXICATION

One of the reasons for the disruption of the proper process of oxygen transportation to the cells are the toxic effects of carbon monoxide (CO). In 1857 the physiologist Claude Bernard for the first time described the influence of CO on tissue hypoxia which occurred due to the reducing of the capacity of oxygen transportation in blood. Carbon monoxide is able to form stable bonds with metalloproteins, especially hemoglobin, myoglobin, hydroperoxidase, cytochrome oxidase and cytochrome p-450. Carbon monoxide possesses 200-300 times higher binding affinity to hemoglobin than oxygen and it competes with oxygen for the four heme groups of hemoglobin with which it forms carboxyhemoglobin (COHb). The formation of carboxyhemoglobin blocks the possibility of oxygen transportation to tissues and as a result leads to the occurrence of tissue hypoxia (3-5). This process contributes to the shifts of the oxyhemoglobin dissociation curve to the left which causes difficulties in diffusing into cells. The decreased amount of oxygen stimulates the respiratory function thus increasing the minute ventilation and causing further increase of the COHb concentration (5, 6). Besides competing for the bonds to hemoglobin carbon monoxide also possesses 40 times higher affinity to myoglobin which is a hemoprotein present in the skeletal muscles and in the myocardium in which it acts as a temporary oxygen reservoir. The bonding of myoglobin with CO molecules results in the creation of a non-active form – carboxymyoglobin (COMb) which is of particular significance for cardiologically ill patients. The limitation of oxygen consumption in the muscles may in a short time lead to lowering the cardiac output and next to circulatory failure (5, 7, 8). Acute carbon monoxide intoxication equally strongly affects the functioning of the central nervous system as that of the circulatory system. The increasing if the concentration of COHb in the central nervous system (CNS) results in the deceleration of the processes of the a-a3 cytochrome system and next in the lowering of the intracellular ATP level, the effect of which is the occurrence of convulsions in the intoxicated person. The consequence is the degeneration of neurons in particularly sensitive areas of the CNS. The conducted anatomopathological studies have demonstrated a significant level of neuron damage especially in the cerebral cortex and in the medulla oblongata. CO intoxication also causes metabolic disorders resulting in the increasing of the amount of lactates and pyruvates (5, 7). The population which is particularly vulnerable to CO are pregnant women. Fetal hemoglobin has got higher affinity for CO than the mother's hemoglobin and also the fetal elimination coefficient is slower, due to which even if the mother does not suffer from severe intoxication symptoms, she may experience the consequences of the intoxication after the period of initial latency which lasts from 1 to 6 weeks. The delayed symptoms may include memory disorders, personality changes, euphoria, impaired judgment and impaired abstract thinking, weakening of concentration (5, 8).

INTOXICATION SYMPTOMS

The symptoms of carbon monoxide intoxication are differentiated and they depend on the blood level of carboxyhemoglobin. With a COHb concentration of 4% the ability to visually distinguish between small lighting differences becomes deteriorated. A symptom which is also observed is the deterioration of the results of certain psychological tests, e.g. the selection of the appropriate letters, appropriate colors or supplementing missing letters. A carboxyhemoglobin concentration of 8-10% results in much more serious errors in test examinations i.e. arithmetic errors, errors in finding plural words etc. As the COHb concentration in the blood increases, the intoxication symptoms become more serious. A very dangerous symptom is increasing weakness, even paralysis of limb muscles usually occurring when the HbCO concentration reaches 50%. This state is very dangerous because it makes it impossible to seek rescue by escaping from the place of exposure and only external help may save the person who is at risk. The clinical course of carbon monoxide intoxication correlates with the baseline HbCO level and with the time of exposure. The COHb concentration which is considered critical is ca. 60% (8). The level of carbon monoxide intoxication in persons exposed to it depends on the CO concentration in the room in which one is staying, on the time of exposure and on the physical activity affecting minute ventilation (tab. 1).

Together with the increasing of the blood concentration of carboxyhemoglobin symptoms typical for particular systems occur. The most common symptoms are CNS disorders which include headache and dizziness, disorders of memory and of attention concentration, visual disturbance, agitation, confusion, fainting, convulsions and coma. Symptoms typical for the disorders of the cardiovascular system are discomfort in the chest during minimal effort and rest pain. Simultaneously the examination may show tachycardia, hypo- or hypertension, supraventricular and ventricular arrhythmias, conduction blocks, pulmonary edema, acute coronary syndrome, sudden cardiac arrest. Symptoms related to the respiratory system include tachypnoe and the feeling of dyspnea. Moreover the present symptoms may be nausea and vomiting – related to the disorders within the digestive system as well as weakening of the muscle strength (9). The intoxicated patient's skin is usually pale blue or grayish in color. The "Cherry Red" symptom described in literature in which the skin becomes rosy occurs very rarely and it is present on the corpses of intoxicated persons after long exposure to the toxic atmosphere. In patients who are alive it may occur in case of acute intoxication during the first phase with a high concentration of CO in the surrounding air (3, 7).

Tab. 1. Carbon monoxide intoxication symptoms depending on the concentration of COHb (7, 8)

% COHb in the blood	Intoxication symptoms	Intoxication severity
0-10%	asymptomatic or with non-specific symptoms	mild intoxication
10-20%	headaches, a feeling of pressure around the temples and the forehead, pulsating in the temples, widening of cutaneous blood vessels, weakness, nausea	moderate intoxication
20-30%		
30-40%	as above, vomiting, dizziness, visual disturbance, fainting collapse, redness of skin	severe intoxication/ death
40-50%	as above, deepening of consciousness disorders, acceleration of heart rate and breath, possible death	
50-60%	tachycardia, tachypnoe, Cheyne-Stokes respiration, coma, convulsions, possible death	
60-80%	coma, convulsions, respiratory failure (bradypnoe) and circulatory failure (bradycardia), possible death	
> 80%	severe depression of the circulatory-respiratory system, death after a few breaths	

The non-specific symptoms of carbon monoxide intoxication are very differentiated and may be associated with the symptoms of stroke, hypoglycemia, acute psychosis, alcohol intoxication or influenza, the morbidity peak of which occurs in the autumn-winter period, similarly as in case of carbon monoxide intoxications. Appropriate diagnosing may therefore be difficult, especially when the taken history does not obtain the information about the possible exposure to CO. The possibility of carbon monoxide intoxication should be particularly taken into consideration when similar symptoms have occurred simultaneously in a few persons staying in one room or when the intoxicated person has recently stayed in a room where there was a device which could have been a potential source of carbon monoxide.

THE PROCEDURE IN CARBON MONOXIDE INTOXICATION

In case of carbon monoxide intoxication an issue which is of very high importance is the safety of the persons providing help because even short exposure to the harmful gas may result in the increasing of the number of intoxicated persons. In cases of justified suspicion of carbon monoxide intoxication already at the stage of accepting the report the emergency dispatcher may advise the reporting person to open the doors and windows (in order to allow for better ventilation of the rooms and for the inflow of uncontaminated air) and to leave the place where the intoxication occurred. After the Medical Rescue Team has arrived at the call point the rescue operations should begin from moving the intoxicated patient from the contaminated atmosphere as soon as possible. The Medical Rescue Team should above all assure their own safety and if there is a real threat for their health and life they should leave the evacuation of the intoxicated persons to the Fire Brigade. After the intoxicated person has been moved to a safe location it is necessary to perform the assessment of the vital functions, assessing sequentially: A – the patency of the airways, B – breathing, C – blood circulation, D – consciousness (10).

The basic treatment method for persons intoxicated with carbon monoxide is oxygen therapy. Oxygen in 100% concentration should be administered through a strictly adherent facial mask. In case of diagnosing respiratory failure the patient should be intubated and ventilated with positive end-expiratory pressure. The duration of oxygen therapy depends on the severity of the intoxication. The half-life of carbon monoxide elimination with breathing atmospheric air is ~320 minutes and with breathing 100% oxygen it reduces to 30-90 minutes. Further shortening of this time is possible thanks to applying treatment in a hyperbaric chamber. Simultaneously with applying oxygen therapy it is necessary to introduce symptomatic treatment aimed at the prevention or the treatment of lung edema and brain edema through the administration of Maitol and steroid drugs e.g. hydrocortisone. In case of the occurrence of hypotension caused by the increasing of the size of the venous bed liquid therapy should be introduced and convulsions should be treated by administering anticonvulsants e.g. Relanium (10-12).

Another group of problems which may be faced by the medical rescue team in case of carbon monoxide intoxications are i.a. injuries caused by the falling of the intoxicated person. The procedure in such a case depends on the needs e.g. stopping hemorrhages or

orthopedic stabilization. In case of persons intoxicated with carbon monoxide as a result of a fire it is necessary to remember about treating the possible burns with particular regard to the burns of the upper respiratory tract which may in a short time cause the complete obstruction of the airways and result in the necessity to perform intubation (13). While taking rescue actions at the scene of the incident it is important to remember about the necessity for evacuation and to observe all the persons present in the location where the intoxication took place. It often occurs that carbon monoxide incidents are multiple because whole families become intoxicated. It is then necessary to guarantee the presence of more Medical Rescue Teams so as to ensure effective treatment. As an alternative it is possible to perform a screening examination of the persons potentially present in the zone of exposure to the gas toxicity using non-invasive methods of determining the carboxyhemoglobin level.

HYPERBARIC OXYGEN THERAPY IN THE TREATMENT OF CARBON MONOXIDE INTOXICATIONS

First descriptions related to attempts of applying high oxygen pressures come from the XVII century. An Anglican clergyman Henshaw claimed that high pressures may be helpful in the treatment of acute diseases and pressures lower than that of the atmosphere in the treatment of the chronic ones. In the 1960s the application of hyperbaric oxygen therapy became common due to publications proving the significant reduction of mortality occurring when this therapy was applied in the treatment of gas gangrene and carbon monoxide intoxications (14).

Hyperbaric oxygenation (HBO) is a method consisting in the application of passive oxygen therapy for breathing oxygen of increased pressure in a special pressure chamber (15). The pressure exerted on the patient during the hyperbaric therapy is the sum of the atmospheric pressure and the pressure present in the chamber. The treatment should be carried out in the conditions of applying 100% oxygen under the pressure of minimum 2 atmosphere absolutes (ATA) for the duration of at least 1 hour. Typically the patient remains in the hyperbaric chamber for 1.5-3 hours and the applied pressure is 2.0-2.8 ATA (14). The treatment involves the application of monoplace chambers designed for one patient or multiplace chambers which can accommodate up to a dozen persons (fig. 1a, b) (16).

Irrespectively of the type of the chamber, remaining in the atmosphere of 100% oxygen with pressure higher than atmospheric leads to the increase of the partial pressure of oxygen in the lungs and to a significant increase of oxygen concentration in the blood plasma taking place on the basis of phyical dissolution. With the pressure of 3 ATA the amount of oxygen dissolved in the blood plasma is 6 mm/dl where for

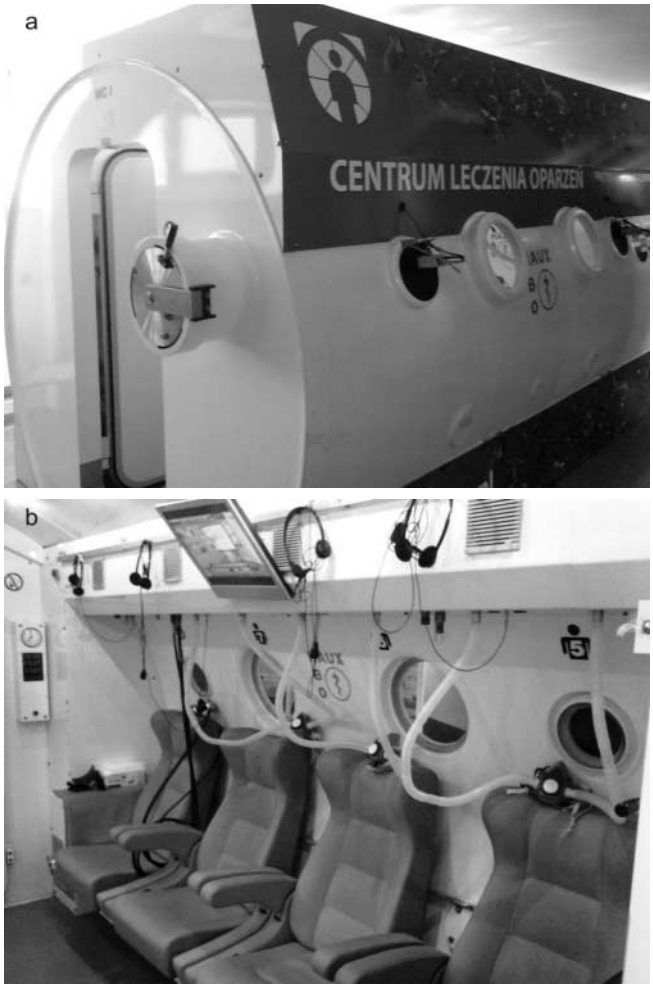

Fig. 1a, b. The hyperbaric chamber of The Burns Treatment Center in Siemianowice Śląskie

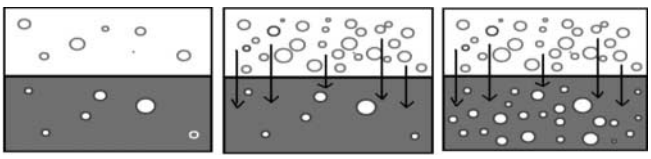

Fig. 2. The process of blood plasma saturation with oxygen resulting from the pressure increase in the hyperbaric chamber (7)

comparison in the conditions of 1 ATA this amount is 0.3 ml/dl (fig. 2) (7, 14).

Applying the hyperbaric oxygen therapy in carbon monoxide intoxication causes the significant reduction of the HbCO half-life. With breathing 100% oxygen with 3 ATA pressure the HbCO half-life is 20 minutes. The deactivation of carbon monoxide is also accelerated in these conditions. Moreover, the early application of hyperbaric oxygen therapy allows for avoiding late complications such as aphasia, memory disorders, balance disorders and disorders of muscle coordination. In case of carbon monoxide intoxication the issue which is of particular significance for the effectiveness of the applied treatment is the level of hemoglobin saturation with CO and the time between the exposure and the beginning of hyperbaric therapy. In CO intoxication the "golden hour" for applying hyperbaric therapy is 6 hours (10).

INDICATIONS FOR HBO TREATMENT IN CARBON MONOXIDE INTOXICATION

The applicable guidelines for applying HBO in Europe have been established during the ECHM (European Committee of Hyperbaric Medicine) conference in Lille in 2004. Taking into consideration various levels of recommendation for applying oxygen therapy, a division into three groups was made. Type I recommendation is applied when the patient's condition requires his or her immediate transfer to a hyperbaric facility due to the fact that only this method guarantees the positive effect of treatment. Type II recommendation provides applying hyperbaric oxygen therapy in situations in which this results in a positive therapeutic effect and type III recommendation assumes hyperbaric oxygen treatment as optional. The indications for conducting hyperbaric therapy have also been divided into several significance degrees depending on the state of advancement of clinical trials related to the application of HBO in particular disease entities. Considering the level of significance the application of hyperbaric treatment was included into group B – commonly accepted (7).

The data of the National Health Fund related to the years 2010-2015 indicate that in that period 4652 persons were hospitalized due to severe carbon monoxide intoxication. What is more, hyperbaric oxygenation was applied in 1954 patients out of all the cases of severe intoxications in that period. The visible disproportion may be the result of the lack of clear rules for qualifying patients for hyperbaric treatment (fig. 3).

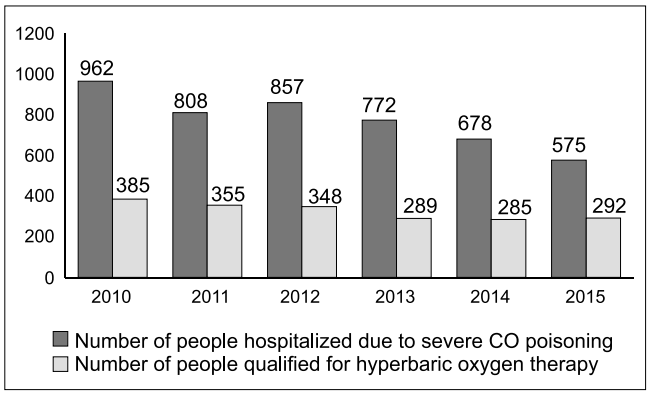

Fig. 3. The number of severe CO intoxications and the hyperbaric oxygenations carried out due to the intoxications in the years 2010-2015
Source: https://prog.nfz.gov.pl

In accordance with the position of the Clinical Toxicology Section of the Polish Medical Association transportation to a facility equipped with HBO should be taken into consideration in case of pregnant women with COHb concentration > 25%, in case of pregnant women with COHb concentration > 15% and accompanying neurological and/or cardiologic disorders and/or metabolic acidosis occurring despite of carrying out normobaric oxygen therapy treatment and also in patients who despite of having undergone normobaric oxygen therapy suffer from prolonged coma, persistent neurological and/or cardiological disorders and/or metabolic acidosis (9). According to other authors treatment applying hyperbaric oxygen therapy should be taken into consideration in patients in whom the COHb level was found to be higher than 25% together with the loss of consciousness in the taken history and with pathological neurological symptoms as well as signs of myocardial ischemia and heart arrhythmia. They should undergo at least one session in the hyperbaric chamber within 24 hours from the moment of intoxication and an additional session should be considered in persons with persistent neurological symptoms (5, 17, 18).

CASES DESCRIPTIONS

Case 1

Male patient aged 19, the taken history showed that the patient was treated for bronchial asthma. The Medical Rescue Team was summoned due to the patient's loss of consciousness in the bathroom (found by parents). The ABCDE examination: airways patent, 18 breaths, HR – 80/min. Arterial pressure 110/80, glycemic level 110 mg%. 7 points in the GCS scale (eye opening response to pain, incoherent, flexion to pain or decorticate). An examination carried out with a CO detector indicated the result of 50 ppm, an examination carried out with the Masimo Rainbow pulse oximeter (Lifepak 15) indicated 32% HbCO. The existing risk was communicated to the residents who were ordered to leave the flat. The Fire Brigade was informed and came to the scene of the event to take appropriate actions. The patient underwent passive oxygen therapy 12 l/min and was transferred to the Accident & Emergency Department (A&E) where the COHb blood level examination gave the result 28.60%. The decision was made to transfer the patient to a facility in which it was possible to carry out hyperbaric treatment.

Case 2

Male patient aged 89, the taken history demonstrated that the patient was treated for paroxysmal atrial fibrillation and prostatic hypertrophy. The Medical Rescue Team was called due to the patient's loss of consciousness which occurred while he was taking a bath. In the moment when the Medical Rescue Team arrived the patient was conscious, he was slowed down, oriented in the place and time, his circulatory and respiratory systems were efficient. ABCDE examination: airways patent, breath 16/min, HR – 90/min. Arterial pressure110/80, GCS 15 points. An examination carried out with a CO detector indicated the result of 130 ppm, an examination carried out with the Masimo Rainbow pulse oximeter (Lifepak 15) indicated 28% HbCO. The existing risk was communicated to the residents who were ordered to leave the flat. The Fire Brigade was informed and came to the scene of the event to take appropriate actions. The patient underwent passive oxygen therapy 15 l/min and was transferred to the A&E Department where the COHb blood

level examination gave the result 34.80%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Case 3

Female patient aged 62 without medical history, complaining of pain and dizziness. When the Medical Rescue Team arrived to the call point she was conscious and oriented auto- and allopsychically. ABCDE examination: airways patent, breath 18/min, HR – 100/min. Arterial pressure – 185/110, GCS 15 points. An examination carried out with a ToxCO sensor demonstrated 37% of HbCO in the exhaled air. The patient underwent passive oxygen therapy 12 l/min, next she was transferred to the A&E Department where CO intoxication was confirmed by performing a COHb blood level examination – result 34.80%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Case 4

Male patient aged 54 without medical history. He complained of dizziness, convulsions, disturbances of consciousness. The ABCDE examination: airways patent, breath 16/min, HR – 88/min. Arterial pressure – 140/80, GCS 15 points. The examination carried out using a CO detector gave the result 160 ppm. The Fire Brigade was notified. An examination carried out using the Masimo Rainbow pulse oximeter (Lifepak 15) indicated 40% HbCO. An examination carried out with a ToxCO sensor demonstrated 32% of HbCO in the exhaled air. The patient underwent passive oxygen therapy 12 l/min, next he was transferred to the A&E Department where CO intoxication was confirmed by performing a COHb blood level examination – result 37.20%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Case 5

Male patient aged 43 without medical history. Loss of consciousness in the bathroom, vomiting. Earlier he had had a few glasses of wine. In the moment of the arrival of the Medical Rescue Team the patient was slowed down. The ABCDE examination: airways patent, breath 10/min, HR – 85/min. Arterial pressure – 140/90, GCS 11 (4/3/4/) in the ambulance GCS 14 points. An examination carried out with a CO detector gave the result 250 ppm in the air. The Fire Brigade was notified. An examination carried out using the Masimo Rainbow pulse oximeter (Lifepak 15) indicated the level > 40% HbCO. The patient underwent passive oxygen therapy 15 l/min, next he was transferred to the A&E Department where CO intoxication was confirmed by performing a COHb blood level examination – result 49%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Case 6

Female patient aged 28 without medical history, after a loss of consciousness she reported headache and weakness. In the ABCDE examination: airways patent, breath 14/min, HR – 72/min. Arterial pressure – 100/60, GCS 15 points. An examination carried out with a CO detector gave the result 280 ppm in the air. The Fire Brigade was notified. An examination carried out using the Masimo Rainbow pulse oximeter (Lifepak 15) indicated the level 28% HbCO. The patient underwent passive oxygen therapy 15 l/min, next she was transferred to the A&E Department where CO intoxication was confirmed by performing a COHb blood level examination – result 26.50%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Case 7

Female patient aged 29 without medical history. Condition after fainting without losing consciousness, in the moment of the arrival of the Medical Rescue Team she reported tinnitus. In the ABCDE examination: airways patent, breath 18/min, HR – 90/min. Arterial pressure – 140/80, GCS 15 points. An examination carried out with a CO detector gave the result 180 ppm in the air. The Fire Brigade was notified. An examination carried out using the Masimo Rainbow pulse oximeter (Lifepak 15) indicated the level 21% HbCO. The patient underwent passive oxygen therapy 15 l/min, next she was transferred to the A&E Department where CO intoxication was confirmed by performing a COHb blood level examination – result 25%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Case 8

Female patient aged 29 without medical history. Condition after fainting in the bathroom, she reported tinnitus and numbness of hands. In the ABCDE examination: airways patent, breath 12/min, HR – 100/min. Arterial pressure – 140/80, GCS 15 points. An examination carried out with a CO detector gave the result 31 ppm in the air. The Fire Brigade was notified. An examination carried out using the Masimo Rainbow pulse oximeter (Lifepak 15) indicated the level 29% HbCO. The patient underwent passive oxygen therapy 15 l/min, next she was transferred to the A&E Department where CO intoxication was confirmed by performing a COHb blood level examination – result 27.60%. The decision was made to transfer the patient to a facility where it was possible to carry out hyperbaric treatment.

Discussion

In the presented cases all the patients experienced symptoms typical for carbon monoxide intoxication such as loss of consciousness, fainting, headache and dizziness, nausea, vomiting, convulsions, tinnitus. The application of CO detectors allowed for confirming the

presence of the toxic gas; the lowest detected level was 31 and the highest one – 280 ppm. Non-invasive methods of determining COHb levels allowed for confirming the suspicion of the intoxication. In one case the level of carboxyhemoglobin exceeded the diagnostic scale of the device – the result was over 40% COHb whereas in other cases the level was determined in the range from 21 to 40%. After being transferred to the A&E Department the patients underwent laboratory examinations consisting in determining the level of carboxyhemoglobin in the blood samples taken from them. The results of the performed examinations confirmed the presence of COHb in the range from 25 to 49%. In case of five patients the result of the examination carried out in the A&E Department was lower than the result of the examination carried out by the Medical Rescue Team – it was by average 4.2% lower. In case of the patient with the COHb level exceeding 40% the result of the examination was higher by 9% and in case of two patients the level of carboxyhemoglobin in the laboratory examinations was higher by respectively 4 and 6.8%. Analyzes were also carried out in relation to the time which elapsed from the examination carried out by the Medical Rescue Team at the scene of the event until the moment of the examination confirming the intoxication performed in the A&E Department. The average time was 55 minutes; the shortest time was 27 minutes and the longest one – 84 minutes. All the patients were qualified and then transferred to an acute intoxication treatment center equipped with a hyperbaric chamber and located 70 kilometers from the A&E Department (tab. 2).

CONCLUSIONS

The symptoms of carbon monoxide intoxication are not characteristic. The key role in the correct diagnosing is played by a carefully taken history relating to the circumstances in which the symptoms started occurring, the confirmation or exclusion of the presence of CO and also determining the blood concentration of carboxyhemoglobin. The basic method of treatment for patients intoxicated with carbon monoxide is applying oxygen therapy until the moment of the reducing of the carboxyhemoglobin level to proper values and in case of severe intoxications, until the moment when hyperbaric therapy is possible. Medical Rescue Teams equipped with devices allowing for confirming the presence and measuring the concentration of carbon monoxide in the atmosphere as well as non-invasive determining of the blood level of COHb may make a diagnosis on the basis of the so called triad of symptoms required for diagnosing acute carbon monoxide intoxication. This triad includes the taken history indicating possible exposure to CO, the presence of symptoms indicating intoxication and the determining of the elevated level of carboxyhemoglobin (3). Developing uniform criteria indicating patients qualifying for therapy in the hyperbaric chamber and introducing equipment allowing for detecting carbon monoxide and for non-invasive determining of the carboxyhemoglobin level could allow for shortening the time in which a patient with symptoms of acute intoxication reaches a specialist facility.

Tab. 2. The characteristics of the described patients

No.	Sex/ age	Medical Rescue Activities Card (MCR)	COHb measurement – Medical Rescue Team	COHb measurement – A&E Department	Time of performing the examination – Medical Rescue Team	Time of performing the examination – A&E Department	Time between the Medical Rescue Team examination and the A&E Department examination
1	M 19	losses of consciousness GCS 2/2/3	32% (LifePak) CO 50 ppm	28.60%	06:22	07:54	84 min
2	M 89	after a loss of consciousness GCS 15	28% (LifePak) CO 130 ppm	34.80%	22:50	23:40	50 min
3	F 62	headache and dizziness GCS 15	37% (ToxCO)	25.10%	16:04	17:08	64 min
4	M 54	dizziness, convulsions during taking a bath GCS 15	40% (LifePak) CO 160 ppm	37.20%	14:58	16:04	66 min
5	M 43	loss of consciousness in the bathroom, vomiting GCS 4/3/4 in the ambulance GCS 14	> 40% (LifePak) CO 250 ppm	49%	23:09	23:55	44 min
6	F 28	after loss of consciousness, headache GCS 15	28% (LifePak) CO 280 ppm	26.50%	20:38	21:05	27 min
7	F 29	after loss of consciousness, tinnitus, GCS 15	21% (LifePak) CO 180 ppm	25%	16:21	17:13	52 min
8	F 28	fainting, tinnitus GCS 15	29% (LifePak) CO 31 ppm	27.60%	00:34	01:23	49 min

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