Analysis of the features of acute carbon monoxide poisoning and hyperbaric oxygen therapy in children

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The aim of this study was to make a retrospective descriptive analysis of the features of children with acute carbon monoxide poisoning (COP). We evaluated 74 children (43 girls, 31 boys; age range 1 to 17.8 years) who were consecutively admitted to our emergency unit and hospitalized with accidental acute COP between June 2003 and June 2005. All patients received normobaric oxygen therapy until their carboxyhemoglobin (COHb) levels were decreased below 2% and their symptoms resolved. Thirty-eight of 74 patients (51.4%) also received hyperbaric oxygen (HBO) therapy as indicated by signs and symptoms or COHb levels. COHb levels were significantly higher and hospitalization period was longer in the children who had abnormal neurological findings ($p<0.05$ for both). All patients showed complete recovery without neurological sequelae except one who had visual impairment at discharge, and antiepileptic therapy was started because of epilepsy after seven months. Acute COP is an important health problem in our country, especially in winter, because of poorly functioning heating systems. The clinical spectrum including neurological findings varies during childhood. We suggest that HBO therapy could be used safely in children.

Key words: carbon monoxide poisoning, children, hyperbaric oxygen therapy, delayed neurologic sequelae.

Carbon monoxide poisoning (COP) is one of the most common causes of mortality and morbidity due to poisoning[1-4]. It accounts for approximately 3,800 deaths in the United States per annum[1,2]. Although the incidence of COP has not been well investigated in the pediatric population, almost one-third of CO exposures occurred in children[5,6]. The frequency of COP has been reported as between 3.6-9.4% among childhood poisoning[7,8] and between 58.2-75% among fatal childhood poisonings[4,9].

The sources of exogenous CO that cause poisoning include motor vehicle exhaust fumes, poorly functioning heating systems (gas heaters, catalytic gas ovens or stoves), improper use of coal or wood stoves and inhaled smoke[10]. The manifestations of acute COP are nonspecific and severity of symptoms ranges from mild to severe, such as coma, respiratory depression and hypotension[10]. Coma, confusion, seizures, syncope and death can occur in patients with prolonged or severe CO exposure[10]. Initial symptoms such as headache, dizziness, nausea, vomiting, and malaise may mimic a nonspecific viral illness[10,11]. In younger children, these effects may be more difficult to recognize[11]. Children may be at greater risk of injury after CO exposure as a result of their high respiration rate, high oxygen metabolism, and immature central nervous system[6,12]. After apparent recovery from the acute CO intoxication, delayed neurologic and/or psychiatric symptoms are more frequently reported in adults than children[3,13-15]. In some cases, neuroimaging studies may demonstrate different abnormalities after severe poisoning, particularly in the basal ganglia and the white matter[10,16-18].

The treatment of COP is essentially 100% normobaric oxygen (NBO).[10] However, hyperbaric oxygen (HBO) therapy is often

recommended for both adult[14,19] and pediatric[15,20-22] patients with severe COP. However, it is unclear whether HBO therapy influences the rate of delayed neurologic sequelae of COP[23]. On the other hand, there are some debated data about definitive indications for HBO therapy in both adults and children[16]. We aimed in this study to conduct a retrospective descriptive analysis of the features of children with acute COP.

Material and Methods

We retrospectively evaluated 74 consecutive children (43 girls, 31 boys) who were exposed to CO accidentally and hospitalized at Eskişehir Osmangazi University Pediatric Department between 1 June 2003 and 1 June 2005. The median age was 9.5 years (range 1.0-17.8). The girl/boy ratio was 1.4. Detailed clinical history including tobacco smoke exposure and type of home heating system was taken and physical and neurological examinations were performed in each patient. Peripheral blood samples for blood gas analysis were taken as soon as possible on admission and if needed for other investigations such as hemogram, biochemical analysis, creatine phosphokinase (CPK), creatine kinase with muscle-brain subunits (CK-MB), and troponin T. Chest roentgenogram, cranial computerized tomography (CT) scan, and cranial magnetic resonance imaging (MRI) were studied. Blood gas analysis was performed with Radiometer (Copenhagen) ABL 735 blood gas testing analyzer. Because all patients were nonsmokers, significant level of carboxyhemoglobin (COHb) for COP was accepted as above 2%. We provided NBO therapy to all patients with oxygen at a rate of 10 liters per minute by face mask that prevented rebreathing at ambient pressure until their COHb levels were decreased below 2% or their symptoms resolved. One intubated patient was mechanically ventilated with 100% oxygen. An additional 38 out of 74 patients received NBO treatment plus HBO therapy, as indicated by signs and symptoms or COHb levels. In our study, indications of HBO therapy included neurologic symptoms on presentation (seizure, coma, lethargy, syncope), continued neurologic findings after NBO therapy (headache, confusion, visual disturbances, ataxia), abnormal neuropsychiatric findings, or elevated COHb level (>20%), which were also recommended by other investigators[6]. We consulted the military-based HBO center in our city and decided to give HBO therapy based on the established criteria of 15 October 2004. Some patients who fulfilled these criteria prior to that date and an additional two patients (1 with pulmonary edema and severe respiratory failure and 1 with ear pain during HBO therapy) did not receive HBO therapy.

Hyperbaric oxygen therapy was initiated within 24 hours (median 3.5; range 1-24 hours) after exposure to CO in a multiplace chamber (Bara-Med®, Model HTC 4/2/6, Environmental Tectonics Corporation (ETC), USA). This chamber is pressurized with compressed air while the patients breathe 100% oxygen through special masks or oxygen hoods. The patients were observed by treatment control panel during the session. Each chamber session lasted about 140 minutes, which included 20 minutes for compression, 100 minutes at a pressure of 2.4 atmospheres absolute (ATA) with two 5-minute intervals between 30 minutes of oxygen administration, and 20 minutes for decompression.

Results were expressed as median or mean and standard deviation. Statistical analysis comparing the two groups was made using the independent samples t test, chi-square test and Mann-Whitney U test depending on the type of data. Correlation analysis was performed using Spearman's rho correlation method. For all statistical analyses, a value of $p<0.05$ was accepted as statistically significant. All analyses were two-tailed and performed with the SPSS software version 11.5 for Windows.

Results

Between June 2003 and June 2005, a total of 16,485 patients were admitted to Eskişehir Osmangazi University Pediatric Emergency Unit. Eight hundred and nine patients were admitted with intoxication, and 107 (13.2%) of them were diagnosed as COP. Three patients were admitted with cardiopulmonary arrest and died, 30 patients were followed up in the Pediatric Emergency Department and discharged based on improvement in clinical findings and COHb, and 74 patients were hospitalized. The sources of CO exposure of the patients are given in Table I. In the case of 60 patients (81.1%), additional family

Table I. Sources of Carbon Monoxide (n=74)

Source of exposure	Number of patients (%)
Coal stoves (via fume and/or leak of CO)	40 (54.1)
Natural gas appliances	20 (27.0)
Gas water heaters and LPG*	14 (18.9)

* Two patients were exposed to CO by leak of gases from car fueled by liquefied petroleum gas (LPG).

members were also poisoned. The signs and symptoms of the patients on admission are shown in Table II. The clinical history of 4 patients (5.4%) was unremarkable for CO intoxication and diagnosis was confirmed with routine blood gas analysis. According to the clinical history, the median duration of exposure period to CO was 3.8 hours (range 0.2-8.0). The median interval between end of the exposure to CO and initial COHb measurement was 1.2 hours (range 0.5-3.0). On neurologic examination, 21.6% of patients had abnormal findings including positive Babinski sign, hyperactive deep tendon reflex (DTR) or hypertonic posture, or loss of consciousness. In our study, 13 (17.6%) patients with more severe symptoms underwent neuroimaging, 11 of whom were found to be normal; only 2 patients (2.7%) had brain edema on cranial CT and in 1 of them, cranial CT on admission also showed bilateral hypodensity in the watershed areas.

The mean initial COHb level (%) was 26.4±10.7 (range 6-56) in all patients. The frequency of symptoms according to COHb levels is demonstrated in Table III. Some blood gas analysis and biochemical parameters at admission are presented in Table IV. As initial treatment, all patients received NBO therapy with non-rebreather mask and 38 (51.4%) patients received NBO plus HBO therapy (Table V). A chest X-ray was performed in all patients before HBO therapy. The median number of HBO chamber sessions was 1 (range 1-19) (Table VI). In 1 patient, HBO therapy was discontinued because of ear pain. During the hospitalization, 6 (9.5%) patients had hyperglycemia, and 1 (1.4%) of them also had pulmonary edema, which resolved with diuretic treatment. The median hospitalization period was 3.0 (range 1-25) days. The median duration of recovery of COHb levels (below 2%) was 14.5 (range 4-52) hours in patients who received HBO therapy and 17.5

Table II. Symptoms and Signs of the Patients on Admission (n=74)

Symptoms and signs	n (%)
Altered mental state	39 (52.7)
Dizziness	28 (37.8)
Headache	24 (32.4)
Nausea	23 (31.1)
Vomiting	19 (25.7)
Syncope	17 (23.0)
Weakness	12 (16.2)
Upper respiratory tract	10 (13.5)
Loss of consciousness	7 (9.5)
Convulsion	7 (9.5)
Without symptom	6 (8.1)
Positive Babinski sign	4 (5.4)
Tremor	4 (5.4)
Ataxia	4 (5.4)
Abdominal pain	2 (2.7)
Gastrointestinal	2 (2.7)
Cardiopulmonary arrest	1 (1.6)

Table III. Frequency of Symptoms According to COHb Levels of the Patients (n=74)

	Initial COHb level (%)					
	6-20 (n=20)		>20-40 (n=48)		>40-56 (n=6)	
Symptom	n	%	n	%	n	%
Dizziness	4	20.0	21	43.8	1	16.7
Headache	7	35.0	12	25.0	0	
Nausea	4	20.0	17	35.4	0	
Vomiting	2	10.0	11	22.9	3	50.0
Syncope	0		15	31.3	1	16.7
Weakness	3	15.0	9	18.8	0	
Altered mental state	6	30.0	26	54.2	4	66.7
Loss of consciousness	2	10.0	4	8.3	1	16.7
Convulsion	3	15.0	3	6.3	0	
Abdominal pain	0		2	4.2	0	
Cardiopulmonary arrest	1	5.0	0		0	
Upper respiratory tract	2	10.0	6	12.5	2	33.3
Gastrointestinal	0		2	4.2	0	
No symptom	2	10.0	4	8.3	0	

Table IV. Some Blood Gas Analysis and Biochemical Parameters at Admission

Parameters	n (total)	Median (Range)
COHb level (%)	74	26.3 (6.0-56.0)
paCO2 (mmHg)	74	37.6 (9.1-51.6)
pH	74	7.35 (7.05-7.53)
HCO3 (mmol/L)	74	21.0 (9.9-25.0)
Lactate (mg/dl)	74	22.5 (6.0-93.0)
AST (U/L)	74	22.0 (10.0-121.0)
ALT (U/L)	74	14.0 (6.0-42.0)
*CPK (U/L)	26	95.0 (44.0-236.0)
*CK-MB (U/L)	35	21.0 (8.3-83.0)
*Troponin T (ng/ml)	39	0.01 (0.01-0.18)

COHb: Carboxyhemoglobin. AST: Aspartate aminotransferase.
ALT: Alanine aminotransferase. CPK: Creatine phosphokinase.
CK-MB: Creatine kinase with muscle and brain subunits.
*: Studied in some patients.

Table V. Types of Oxygen Therapy Compared to Initial COHb Levels

	Type of oxygen therapy		
Initial COHb level	NBO	NBO and HBO	Total
≤20%	10	10	20
>20%	26	28	54
Total	36	38	74

COHb: Carboxyhemoglobin. NBO: Normobaric oxygen. HBO: Hyperbaric oxygen.

Table VI. Total Number of Sessions of Hyperbaric Oxygen Group (n=38)

Number of HBO sessions	No. of patients (%)
1	31 (81.6)
2	2 (5.3)
4	2 (5.3)
5	1 (2.6)
10	1 (2.6)
19	1 (2.6)

HBO: Hyperbaric oxygen.

(range 8.8-39.3) hours in patients who received NBO treatment. The hospitalization period of patients who had abnormal neurologic signs was significantly longer than in the others ($p<0.05$); however, no correlation was found between initial COHb levels and this period ($r=0.047$, $p>0.05$). Initial COHb levels were significantly higher in patients with abnormal neurologic signs ($p<0.05$). Among those who received HBO therapy, hospitalization period was not significantly different between the study group and the patients whose initial COHb levels were above 20% ($p>0.05$). Fifty-seven (77.0%) patients were followed up for 6.1 to 12.5 months (median, 7.3 months) after discharge from hospital. One of them was discharged with visual impairment, and seven months later she developed epilepsy and was put on antiepileptic drug therapy. Visual impairment had gradually improved at the one-year follow-up examination. One month later, follow-up cranial MRI demonstrated bilateral cerebral infarction in the basal ganglia and occipitoparietal regions, and seven months later it showed addition of cerebral atrophy. Her IQ level was determined as 68 using Stanford Binet test. None of the patients had serious cardiac signs or symptoms.

Discussion

In this study, the frequency of COP among the overall childhood poisoning cases who were diagnosed in our emergency department was found as 13.2%. This is higher than several investigations (3.6-9.4%)[7,8].In our study, 66.2% of patients were admitted in the winter, which we suggest is related with the malfunctioning heating systems.

Carbon monoxide poisoning has no pathognomonic signs or symptoms, and a high level of suspicion is essential for making the diagnosis[10]. The most common symptoms in our patients were altered mental state, dizziness, headache, syncope, convulsion, and loss of consciousness (Table II). One of our patients had cardiopulmonary arrest, while 6 cases (8.1%) had no symptoms of CO intoxication but their COHb levels were unexpectedly elevated. It has been reported that COP has been commonly misdiagnosed as flu-like viral illness in both adults and children[11,24,25]. Vomiting may be the only presenting symptom of COP in infants, suggesting gastroenteritis[16]. In our study, 10 patients presented flu-like symptoms on admission. It may be coexistence or COP may produce symptoms similar to upper respiratory tract infection. The levels of COHb do not correlate well with the severity of symptoms because once the patient is removed from the CO source, levels fall rapidly with time, and duration of exposure is an important factor mediating toxicity[10]. On the other hand, we detected that COHb level and hospitalization period were significantly higher in the patients who had abnormal neurologic sign on admission ($p<0.05$ for both). We hypothesize that the extended duration of hospitalization in patients with abnormal neurologic signs is probably due to high vulnerability of the central nervous system to CO[16].

Hyperglycemia and glycosuria are commonly encountered during COP[26]. CO has probable contributory effects on the development of cerebral injury in patients at risk for cerebral ischemia[27]. In our study, hyperglycemia was found in 9.5% of patients and was regulated with appropriate insulin and fluid treatment. Meert et al.[28] reported that 34% of patients had hyperglycemia and 24.4% of patients demonstrated pulmonary edema or infiltration on chest radiograph. In our study, only one patient had pulmonary edema.

The most common pathological findings on MRI in patients with COP include bilateral necrosis in the globus pallidus and bilateral hyperintensities in periventricular white matter[10,16-18]. In our study, only one patient demonstrated bilateral cerebral infarction in the basal ganglia and occipitoparietal regions on MRI, and this patient had a history of cardiac arrest on admission.

Carbon monoxide poisoning is one of the principal indications for HBO therapy. Crush injury, traumatic ischemia, compartment syndrome, gas gangrene, necrotizing fasciitis, refractory osteomyelitis, massive air embolism, purpura fulminans and decompression sickness constitute other accepted indications for HBO therapy[22]. The beneficial effects of HBO therapy in children have been rarely reported[22]. Some studies have supported the effectiveness of HBO therapy in children with COP[15,20-22] while another reported effectiveness of NBO therapy[28]. Generally accepted indications

of HBO therapy in children with COP are as follows: severe neurologic symptoms on presentation, syncope, continued neurologic symptoms and findings after several hours of NBO therapy, myocardial ischemia and cardiac dysrhythmias, abnormal neuropsychiatric findings, high COHb level, and infants under six months of age with symptoms of lethargy, irritability, and poor feeding. During pregnancy, high COHb levels (>15-20%), and symptoms of COP or evidence of fetal distress were determined as accepted or recommended criteria[6,12]. Untreated pneumothorax represents an absolute contraindication[22].

Hyperbaric oxygen therapy is only available at a few centers, making distance and transport of unstable patients major concerns with respect to HBO therapy[19,28]. We have the advantage of having an HBO therapy center in Eskişehir. It has been reported that HBO therapy is more effective within 4-6 hours of the initial exposure to the CO[6] and that optimal frequency is at least two sessions[19,29]. On the other hand, HBO therapy may be effective in CO-induced brain injury as late as one month after CO insult[30]. In our patients, HBO therapy was given initially within 24 hours. We provided a minimum of two HBO sessions to our seven patients who had relatively severe neurologic findings such as coma or convulsion. Neurologic findings of our patients gradually recovered with HBO therapy. In one patient who had been resuscitated for cardiopulmonary arrest before admission, neurologic findings gradually recovered with HBO therapy (total 19 chamber sessions within 25 days). She was discharged with visual impairment and epilepsy developed later.

The side effects of HBO are related to pressure/volume changes and to oxygen toxicity. Middle ear, sinuses, and lung may be commonly affected by pressure changes, and central nervous system and lung by oxygen toxicity. Waisman et al.[22] reported that two out of 139 children had side effects due to HBO therapy including generalized convulsion and pulmonary oxygen toxicity. In our study, 38 (51.4%) patients received HBO therapy within 24 hours without complication. In one patient, HBO was discontinued because of aural barotrauma. To prevent middle ear and pulmonary barotrauma, our patients underwent otoscopic examination and chest radiography before treatment was initiated.

After apparent recovery from the acute CO intoxication, delayed neurologic and/or psychiatric symptoms may be seen in 2.8-10.7%[15,21,28] of children after a lucid interval of 2 to 51 days[21,28]. In children, delayed neurologic sequelae include transient deterioration, memory difficulties, decline in school performance, mental retardation, mutism, urinary and fecal incontinence, various motor abnormalities, facial palsy, psychosis, chronic headaches, seizure, and epilepsy[15,21,28]. Kim and Coe[21] mentioned that neurologic sequelae occurred most frequently in comatose patients and more frequently in patients exposed to CO gas for more than eight hours. In our one patient who had a subsequent comatose period, she developed epilepsy seven months after CO exposure. We suggest that it might be related to asphyxia due to the neurotoxic effect of CO and/or cardiopulmonary arrest. Other patients completely recovered without any complication. Detailed neurologic examination was done on admission and follow-up, but we did not perform the neuropsychological testing because of the difficulties in application in children and its limited value in COP[16].

In conclusion, especially in winter because of the poorly functioning heating systems, acute COP has continued as an important health problem in adults and children in our country. The spectrum of COP symptoms may be wide and variable in children. Detailed history, physical examination, and suspicion are important for the diagnosis of COP. We suggest that HBO therapy can be used safely in children with COP. It might also be effective in preventing the delayed neurologic sequelae. We also recommend that further multi-center randomized controlled investigations be undertaken to determine the effectiveness of and indications for HBO therapy in children with COP.

REFERENCES

1. Cobb N, Etzel RA. Unintentional carbon monoxide-related deaths in the United States, 1979 through 1988. JAMA 1991; 266: 659-663.

2. National Center for Health Statistics. Vital statistics of the United States 1990, II. Mortality, Part A. Washington, D.C.: Government Printing Office; 1994: publication no. (PHS) 95-1101.

3. Choi IS. Delayed neurologic sequelae in carbon monoxide intoxication. Arch Neurol 1983; 40: 433-435.

4. Flanagan RJ, Rooney C, Griffiths C. Fatal poisoning in childhood, England & Wales 1968-2000. Forensic Sci Int 2005; 148: 121-129.
5. Litovitz TL, Klein-Schwartz W, Dyer KS, Shannon M, Lee S, Powers M. 1997 annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. Am J Emerg Med 1998; 16: 443-497.
6. Martin JD, Osterhoudt KC, Thom SR. Recognition and management of carbon monoxide poisoning in children. Clin Ped Emerg Med 2000; 1: 244-250.
7. Andiran N, Sarikayalar F. Pattern of acute poisonings in childhood in Ankara: what has changed in twenty years? Turk J Pediatr 2004; 46: 147-152.
8. Marchi AG, Renier S, Messi G, Barbone F. Childhood poisoning: a population study in Trieste, Italy, 1975-1994. J Clin Epidemiol 1998; 51: 687-695.
9. Asirdizer M, Yavuz MS, Albek E, Canturk G. Infant and adolescent deaths in Istanbul due to home accidents. Turk J Pediatr 2005; 47: 141-149.
10. Ernst A, Zibrak JD. Carbon monoxide poisoning. N Engl J Med 1998; 339: 1603-1608.
11. Baker MD, Henretig FM, Ludwig S. Carboxyhemoglobin levels in children with nonspecific flu-like symptoms. J Pediatr 1988; 113: 501-504.
12. Liebelt EL. Hyperbaric oxygen therapy in childhood carbon monoxide poisoning. Curr Opin Pediatr 1999; 11: 259-264.
13. Tibbles PM, Perrotta PL. Treatment of carbon monoxide poisoning: a critical review of human outcome studies comparing normobaric oxygen with hyperbaric oxygen. Ann Emerg Med 1994; 24: 269-276.
14. Thom SR, Taber RL, Mendiguren II, Clark JM, Hardy KR, Fisher AB. Delayed neuropsychologic sequelae after carbon monoxide poisoning: prevention by treatment with hyperbaric oxygen. Ann Emerg Med 1995; 25: 474-480.
15. Crocker PJ, Walker JS. Pediatric carbon monoxide toxicity. J Emerg Med 1985; 3: 443-448.
16. Tomaszewski C. Carbon monoxide poisoning. Early awareness and intervention can save lives. Postgrad Med 1999; 105: 39-40, 43-48, 50.
17. Pracyk JB, Stolp BW, Fife CE, Gray L, Piantadosi CA. Brain computerized tomography after hyperbaric oxygen therapy for carbon monoxide poisoning. Undersea Hyperb Med 1995; 22: 1-7.
18. Zagami AS, Lethlean AK, Mellick R. Delayed neurological deterioration following carbon monoxide poisoning: MRI findings. J Neurol 1993; 240: 113-116.
19. Weaver LK, Hopkins RO, Chan KJ, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002; 347: 1057-1067.
20. Rudge FW. Carbon monoxide poisoning in infants: treatment with hyperbaric oxygen. South Med J 1993; 86: 334-337.
21. Kim JK, Coe CJ. Clinical study on carbon monoxide intoxication in children. Yonsei Med J 1987; 28: 266-273.
22. Waisman D, Shupak A, Weisz G, Melamed Y. Hyperbaric oxygen therapy in the pediatric patient: the experience of the Israel Naval Medical Institute. Pediatrics 1998; 102: E53.
23. Buckley NA, Isbister GK, Stokes B, Juurlink DN. Hyperbaric oxygen for carbon monoxide poisoning: a systematic review and critical analysis of the evidence. Toxicol Rev 2005; 24: 75-92.
24. Dolan MC, Haltom TL, Barrows GH, Short CS, Ferriell KM. Carboxyhemoglobin levels in patients with flu-like symptoms. Ann Emerg Med 1987; 16: 782-786.
25. Grace TW, Platt FW. Subacute carbon monoxide poisoning. JAMA 1981; 246: 1698-1700.
26. Choi IS. Carbon monoxide poisoning: systemic manifestations and complications. J Korean Med Sci 2001; 16: 253-261.
27. Rovlias A, Kotsou S. The influence of hyperglycemia on neurological outcome in patients with severe head injury. Neurosurgery 2000; 46: 335-342; discussion 342-343.
28. Meert KL, Heidemann SM, Sarnaik AP. Outcome of children with carbon monoxide poisoning treated with normobaric oxygen. J Trauma 1998; 44: 149-154.
29. Gorman DF, Clayton D, Gilligan JE, Webb RK. A longitudinal study of 100 consecutive admissions for carbon monoxide poisoning to the Royal Adelaide Hospital. Anaesth Intensive Care 1992; 20: 311-316.
30. Coric V, Oren DA, Wolkenberg FA, Kravitz RE. Carbon monoxide poisoning and treatment with hyperbaric oxygen in the subacute phase. J Neurol Neurosurg Psychiatry 1998; 65: 245-247.