ORIGINAL ARTICLE

Carbon Monoxide Poisoning in Children

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Background: Carbon monoxide (CO) is an important source of poisoning in children. We performed this study in order to clarify the clinical characteristics of pediatric patients with CO poisoning.
Methods: We identified and reviewed the medical records of pediatric patients diagnosed with CO intoxication and hospitalized in our department during a 10-year period. Epidemiologic and clinical data were collected and analyzed.
Results: A total of 30 children with CO poisoning were identified. Their ages ranged from 2 to 17 years, with a mean of 9.5±4.5 years. Improperly vented exhaust produced by gas hot water heaters was the most common cause of intoxication (53.3%), followed by house fires (26.7%). Intentional poisoning by charcoal burning by parents occurred in six patients (20.0%). The majority of events occurred during cool seasons (76.7%). The most common presenting symptom was consciousness disturbance (86.7%). Only one child died and the mortality was 0.03%. Five (16.7%) children developed delayed neurologic sequelae (DNS), but they all recovered completely within 2 months. Seizures during the acute stage, severe metabolic acidosis, significant hypotension, longer times for recovery to full consciousness, and longer hospital stays were observed in children with DNS.
Conclusion: Children with CO poisoning had good outcomes in this series. Although improperly vented exhaust from water heaters and house fires were the most common causes, intentional poisoning by parents through charcoal burning was also an important factor. Early identification of DNS risk factors might help to provide better care.

1. Introduction

Carbon monoxide (CO) is an important source of poisoning worldwide. About 50,000 emergency department visits each year in the USA are attributed to CO poisoning.[1] Most cases are caused by the incomplete combustion of carbon-containing materials. CO intoxication causes tissue hypoxia and direct damage at the cellular level,[2,3] 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.

The annual mortality due to CO poisoning in Taiwan has increased during the last decade.[4] This may be partially due to a rise in suicides associated

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with charcoal burning.[5] Charcoal burning in an enclosed space is a unique method of suicide used by Chinese people. Children may be intoxicated intentionally when their parents commit suicide, and this is now considered to be a new means of child abuse.[6]

There have been fewer studies on CO poisoning in children than in adults, and reports depicting the characteristics of children with CO poisoning in Taiwan are still lacking. The purpose of this study was to describe and analyze the causes, clinical characteristics, treatments and outcomes in this patient group.

2. Patients and Methods

We identified all pediatric patients (birth to 18 years old) with a diagnosis of CO poisoning hospitalized between January 1996 and March 2007. Each individual medical chart was reviewed. The age and sex of the patient, cause of CO exposure, date and scene, presenting signs and symptoms, vital signs and Glasgow Coma Scale (GCS) on admission, first arterial blood gas and carboxyhemoglobin (HbCO) level, clinical management (including the use of normobaric or hyperbaric oxygen, intubation, mechanical ventilation and inotropic agents), time lapse between admission and full recovery of consciousness, intensive care unit (ICU) stay, total hospital stay, results of computed tomography (CT) and magnetic resonance imaging (MRI), and occurrence of associated injuries and delayed neurologic deficits were all recorded. Records of subsequent outpatient visits were also reviewed for clinical findings that might be associated with CO poisoning.

Severe metabolic acidosis was defined as arterial blood pH <7.15. The time lapse for full consciousness recovery was defined as the period between admission and the moment when the patient's GCS equaled 15. The diagnostic criteria for delayed neurologic sequelae (DNS) were: (1) history of acute CO intoxication, (2) presence of a period of days or weeks when the patient had clear consciousness and was without obvious neurologic deficits after improvement of acute symptoms (lucid period), and (3) recurrence of original symptoms or development of new neuropsychologic symptoms after a lucid period.[7,8]

Categorical data were expressed as absolute counts and percentages. Continuous data were expressed as medians with standard deviations and ranges. Clinical characteristics were compared between (1) groups with different causes, and (2) groups with and without DNS. Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) for Windows software (version 12.0; SPSS Inc., Chicago, IL, USA). Fisher's exact test was used for analysis of categorical data. Continuous data were analyzed using a two-tailed t test or Mann-Whitney test. Statistical significance was defined as a p value less than 0.05.

3. Results

A total of 30 children with CO poisoning were identified. There were 11 males and 19 females. Their ages ranged from 2 to 17 years (mean, 9.5±4.5 years). The mean GCS was 11.6±3.9 points (range, 3–15 points). Mean initial HbCO level was 35.3±13.9% (range, 7.4–60.3). Twenty-five patients (83.3%) had initial HbCO levels >25%. Mean hospital stay was 2.5±5.0 days (range, 1–19 days). Thirteen patients (43.3%) needed intensive care during their acute phases. Six of them received mechanical ventilation. Two patients received inotropic agents because of significant hypotension. Mean ICU stay was 1.8±1.4 days (range, 1–5 days).

The most common cause of intoxication was improperly vented exhaust produced by gas hot water heaters (16 patients, 53.3%). The second most common cause was house fires (eight patients, 26.7%). Intentional poisoning by parents using charcoal burning occurred in six patients (20.0%). Most events (23 patients, 76.7%) occurred during spring (11 patients, 36.7%) and winter (12 patients, 40%). Comorbidities, including inhalation pneumonitis and burns, occurred in five of eight house fire victims. One of these needed 3 days of intensive care in a burns unit in order to manage the skin lesions. There was no significant difference among the groups with different causes in terms of initial GCS, initial HbCO level, occurrence of seizures, time needed for full consciousness recovery, and ICU stay (Table 1). Patients intoxicated as a result of house fires were more likely than other groups to have severe metabolic acidosis, need endotracheal intubation, receive inotropic agents, and have longer hospital stays.

Presenting signs and symptoms included consciousness disturbance (86.7%), seizure (23.3%), headache and dizziness (10.0%), and vomiting (3.3%). Five children (16.7%) had severe metabolic acidosis. Almost all patients (29/30) received 100% normobaric oxygen therapy. Only one 17-year-old male with initial HbCO of 51.6% received hyperbaric oxygen therapy. He recovered rapidly after hyperbaric oxygen management, with no complications.

Brain imaging was performed in six patients (four CT and two MRI). Two patients had normal brain CT scans, while the other two showed brain edema. High signal intensity in the globus pallidus, hippocampus, thalamus and cerebellum were found on T2-weighted images in both of the patients who received brain MRI.

Table 1 Comparison of 30 children with carbon monoxide poisoning due to different causes

Characteristics	House fire ($n=8$)	Other causes* ($n=22$)	p
Age (yr)	7.8±3.8	10.2±4.7	0.200
Initial GCS (point)	9.0±5.6	12.2±3.2	0.238
Initial HbCO (%)	37.3±19.5	34.6±11.7	0.673
Severe metabolic acidosis (n)	5	1	0.0004[‡]
Seizure (n)	2	5	0.89
Mechanical ventilation (n)	5	1	0.0004[‡]
Inotropic agent use (n)	2	0	0.015[‡]
Consciousness recovery time (hr)	27.4±62.2[†]	8.1±20.6	0.829
Hospital stay (d)	8.1±5.4[†]	4.0±4.7	0.014[‡]
ICU stay (d)	2.5±2.0[†]	0.4±0.7	0.07

*Including improperly ventilated exhaust of hot water heater (16 children) and burning charcoal (6 children); [†]excluding one girl who had no detectable vital signs on arrival and died the next day; [‡]$p<0.05$. GCS = Galsgow Coma Scale; HbCO = carboxyhemoglobin; ICU = intensive care unit.

Five patients (16.7%) developed DNS after lucid periods of 3–14 days. Their symptoms included ataxia, dysmetria, personality change, dystonia, rigidity, focal weakness and enuresis. Complete resolution of the symptoms was noted within 2 months in all five patients (Table 2). Patients with DNS tended to have higher risks of seizures in the acute stage, more severe metabolic acidosis and significant hypotension, and they needed longer time to recover to full consciousness and had longer hospital stays (Table 3). Only one child died; she was a 9-year-old girl who was found unconscious at the scene of a house fire. Her vital signs were undetectable in our emergency room and her initial HbCO level was 60.3%. She was declared dead the following day.

4. Discussion

In this study, we found that acute neurologic symptoms, including various degrees of consciousness disturbance and seizures, were the predominant initial presenting features in CO-exposed children. In our series, 86.7% of patients had one of the above symptoms when they were brought to hospital. This observation differs significantly from previous studies, which reported such symptoms in about 65% of patients.[7–9] This suggests a higher severity of intoxication in our patients than in those in other studies. The difference may be due to different criteria used for patient selection: all the children in our study were hospitalized, while previous studies included many non-hospitalized patients. Intoxicated children may show a variety of nonspecific symptoms, such as vomiting, headache or dyspnea, and other symptoms, such as dizziness, weakness, difficulty in concentrating or confusion, visual changes, chest pain, abdominal pain, muscle cramping and ataxia, have also been reported in both adults and children.[2,7,10,11]

Improperly vented heater exhaust was the commonest cause of CO poisoning in this study, which is consistent with observations from several previous studies.[4,8,12–14] Our victims of house fires tended to have cardiac and respiratory failure in the acute stage, and to have higher risks of associated burns, inhalation pneumonitis, and to have suffered from inhalation of other toxic substances at the fire scene. The above factors may contribute to their increased risks of mortality and longer hospital stays.[15,16] Several reports have emphasized the rising incidence of suicide by burning charcoal in urban Taiwan and Hong Kong.[5,6,17,18] According to analysis, the rapid rise in this form of suicide is partially related to media publicity. Combined homicide-suicide cases involving children as victims are not uncommon, but are rarely reported in the medical literature.[6] In accordance with previous reports, all abusers in our series were one of the child's biological parents. Co-intoxication with hypnotics was also found in two of our patients. Although most children recovered without significant deficits, one had severe DNS. Intentional poisoning by burning charcoal should be considered as a means of child abuse with potential mental and physical consequences.[6] The incidence of such tragedies can be reduced by the early identification of risk factors and prompt intervention.

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.[2] DNS was estimated to occur in 10–40% of adult survivors of CO intoxication, and was usually noted after lucid periods of

Table 2 Characteristics of patients with delayed neurologic sequelae

Patient	Age (yr)/sex	Cause	Initial presentation	Initial HbCO (%)	Lucid period/time for resolution (d)	Symptoms	Brain image
1	10/male	House fire	Coma	53.8	7/51	Ataxia, dysmetria	CT: normal
2	5/female	Heater	Seizure with consciousness disturbance	7.4	3/7	Disorientation, dystonia of arms, right wrist weakness	CT: brain edema
3	10/male	Heater	Consciousness disturbance	40.8	14/6	Urinary incontinence, dizziness	Not done
4	15/female	Heater	Seizure with consciousness disturbance	40	5/7	Personality change, rigidity, dystonia	MRI: high signal intensity in bilateral caudate nucleus, globus pallidus, thalamus and hippocampus on T2-weighted image
5	8/male	Burning charcoal	Consciousness disturbance	17.6	3/60	Personality change, dystonia, rigidity, ataxia	MRI: high signal intensity in bilateral globus pallidus, and hippocampus on T2-weighted image

HbCO = carboxyhemoglobin; CT = computed tomography; MRI = magnetic resonance imaging.

3–240 days.[2] Children have a much lower incidence of DNS than adults, with incidences of 2.8–10% reported in previously published pediatric studies.[7,11] The incidence of DNS in our series (17.8%) was slightly higher. 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.[19] The ratio of patients with and without consciousness disturbance in our series was higher than in other studies, which might help to explain our higher incidence of DNS. Previous studies indicated that clinical assessment of the severity of poisoning was unreliable in predicting the occurrence of DNS in mildly or moderately intoxicated patients.[20–22] However, in our study, we found that patients with DNS were more likely to have seizures, severe metabolic acidosis, and hypotension, and to require a longer time for recovery. The propensity for DNS may be affected by the above factors in more severely intoxicated patients.

Hyperbaric oxygen has long been considered to be an important modality for treating CO intoxication, but universal consensus on treatment is so far lacking. Two recent large-scale systemic reviews concluded that guidelines regarding the use of hyperbaric oxygen for reducing neurologic deficits in CO-intoxicated patients could not be established based on existing randomized trials.[23,24] Published evidence and guidelines for the use of hyperbaric oxygen therapy in pediatric CO intoxication are scant.[15,25–28] In our study, almost all children (28/29) were treated with normobaric oxygen, and although symptoms of DNS developed in five of them, they were transient and self-limited. Initial HbCO values in the DNS and non-DNS groups were not significantly different, suggesting that the effectiveness of hyperbaric oxygen in preventing apparent DNS in our patients was questionable. Large-scale, randomized controlled studies including comprehensive neuropsychiatric follow-up are needed to determine the indications for hyperbaric oxygen use in cases of pediatric CO intoxication.

Our study was limited by its retrospective nature, small patient number, and lack of a standard neuropsychiatric test for detecting subtle neuropsychiatric deficits. These factors could result in an underestimation of the incidence of DNS in these children.

In conclusion, pediatric CO poisoning in Taiwan is most commonly caused by improperly vented heater exhaust, and usually occurs in cool weather. Victims of house fires tend to have more severe clinical courses and need intensive care. Intentional CO poisoning by parents using charcoal burning is an important means of child abuse. Regular monitoring of high-risk groups and prompt intervention are

Table 3 Comparison of 29 children with carbon monoxide poisoning with or without delayed neurologic sequelae (DNS)*

Characteristics	With DNS ($n=5$)	Without DNS ($n=24$)	p
Age (yr)	9.6±3.3	9.5±5.0	0.611
Initial GCS (point)	6.6±3.8	12.3±3.6	0.115
Initial HbCO (%)	35.8±22.8	35.2±12.1	0.85
Severe metabolic acidosis (n)	3	3	0.017[†]
Seizure (n)	4	3	0.001[†]
Mechanical ventilation (n)	2	4	0.33
Inotropic agent use (n)	2	0	0.001[†]
Consciousness recovery time (hr)	75.0±72.2	2.8±4.3	0.008[†]
Hospital stay (d)	15.2±5.0	3.4±2.6	0.013[†]
ICU stay (d)	2.0±2.1	1.2±1.3	0.08

*Excluding one girl who had no detectable vital signs on arrival and died the next day; [†]$p<0.05$. GCS = Galsgow Coma Scale; HbCO = carboxyhemoglobin; ICU = intensive care unit.

mandatory. DNS in children is more likely to occur in patients with hypotension, seizures and severe metabolic acidosis. It is usually self-limited and resolves without the use of hyperbaric oxygen therapy.

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