

Clinical Spectrum of Acute Chlorine Poisoning in Children

Çocuklarda Akut Klor Zehirlenmesinin Klinik Spektrumu

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ABSTRACT

Objective: Chlorine gas (Cl₂) is a common substance used in industry, which causes toxic inhalation as a potent pulmonary irritant. Herein, we aimed to investigate the findings of pediatric cases accidentally exposed to Cl₂ gas.

Material and Methods: In October 2017, an accident involving Cl₂ gas exposure occurred in a school where 650 students were trained.

Results: Fifty students breathed in the steam generated in the school hallway as a result of an accident during cleaning. The mean age of the patients was 11.2±1.5 years (range 2–18 years); 62% of the children were male. Among patients evaluated at the emergency department, 21 (42%) patients were discharged within 4-6 hours after the initial examination and symptomatic treatment. The remaining 29 patients were hospitalized. The presenting symptoms were mostly associated with one another, which included cough and dyspnea (n=30, 60%), nausea and vomiting (n=6, 12%), headache (n=7, 14%), and sore throat (n=3, 6%). Thirty patients had elevated creatine kinase-MB (CK-MB) (mean 54.9±50.1 U/L). Five patients had sinus tachycardia on electrocardiogram. During the follow-up period, cardiac enzymes of all patients returned to normal levels. Seven patients were treated with steroids, bronchodilators, and humidified oxygen; 11 patients were treated with oxygen and bronchodilators; the remainders took oxygen alone.

Conclusion: We suggest that this study will contribute to raising awareness about chemicals that can produce toxic substances, as in our cases.

Key Words: Bleach, Chlorine gas, Hydrochloric acid, Pediatric

ÖZ

Amaç: Klor gazı endüstride yaygın olarak kullanılan ve akciğerlerde güçlü bir tahriş edici olarak inhalasyon toksisitesine neden olan bir maddedir. Bu çalışmada kaza ile klor gazına maruz kalan çocuk olgularının bulgularını incelemeyi amaçladık.

Gereç ve Yöntemler: Ekim 2017'de, 650 öğrencinin eğitim aldığı bir okulda Cl₂ gazına maruz kalmayı içeren bir kaza meydana geldi.

Bulgular: Elli öğrenci temizlik sırasında meydana gelen bir kaza sonucu okul koridorunda oluşan buharı teneffüs etti. Ortalama yaşları 11.2±1.5 yıl (dağılım 2-18 yıl) olan çocukların %62'si erkekti. Acil serviste değerlendirilen hastalardan 21'i (%42) ilk muayene ve semptomatik tedaviden sonra 4-6 saat içinde taburcu edildi. Kalan 29 hasta hastaneye yatırıldı. Çoğunlukla birbirleriyle benzer olan başvuru semptomları; öksürük ve nefes darlığı (n=30, %60), bulantı ve kusma (n=6, %12), baş ağrısı (n=7, %14) ve boğaz ağrısı (n=3, %6)'di. Otuz hastada yüksek kreatin kinaz-MB (CK-MB) vardı (ortalama 54.9±50.1 U/L); beş hastanın elektrokardiografisinde sinüs taşikardisi vardı. Takip süresi boyunca tüm hastaların kardiyak enzimleri normale döndü. Yedi hasta steroid, bronkodilatör ve nemlendirilmiş oksijen ile tedavi edildi; 11 hasta oksijen ve bronkodilatör ile tedavi edildi; kalanlar yalnızca oksijen aldı.



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Sonuç: Bu çalışmanın, bizim vakalarımızdaki gibi toksik maddeler üretebilecek kimyasallar hakkında farkındalığının artırılmasına katkı sağlayacağını düşünüyoruz.

Anahtar Sözcükler: Çamaşır suyu, Klor gazı, Hidroklorik asit, Pediatrik

INTRODUCTION

Chlorine gas (Cl_2) is a common substance used in society and industry. It is potentially toxic, particularly in children. Swimming pools, school chemistry experiments, and chemical vehicle accidents are the sources of pediatric toxicities, resulting in respiratory and cardiovascular morbidity (1). The respiratory system is the most commonly affected organ system by chlorine gas exposure (2). Accidental mixing of sodium hypochlorite and hypochlorous acid is one of the reasons for Cl_2 intoxication (3). To date, several accidental exposures have been reported. Herein, we aimed to investigate the findings of pediatric cases accidentally exposed to Cl_2 gas.

MATERIAL and METHODS

In October 2017, an accident involving Cl_2 gas exposure occurred in an elementary school housing 650 students. Fifty students inhaled the vapor formed in the school corridor after a cleaning staff mixed bleach and hydrochloric acid. We retrospectively reviewed the medical records of those who were admitted to our hospital. All patients admitted with acute chlorine poisoning after accidental exposure to the agent were included in the study. Hospitalization decisions were made by the pediatrician on duty. Criteria for admission included: potentially lethal exposure, respiratory distress (low oxygen saturation), persistent cough affecting the quality of life, and symptoms persisting for more than 6 hours after exposure. All medical records including signs and symptoms, physical examination, and laboratory results ECG, chest radiography and blood gas were evaluated.

Ethics committee approval of the study was obtained from Kırıkkale University Clinical Research Ethics Committee (Date: 08.07.2020, No: 2020/09).

Statistical Analyses

The IBM SPSS 25.0 software package was used for all statistical analyses. Independent samples t-test, Mann Whitney-U test, and Chi-square test were performed for comparison of two independent groups. The significance level was taken as $p < 0.05$ in all statistical analyses.

RESULTS

The mean age of the patients was 11.2 ± 1.5 years (range 2–18 years); 62% of the children were male. Among patients evaluated at the emergency department, 21 (42%) patients

were discharged within 4-6 hours after the initial examination; symptomatic treatment was administered to relieve upper respiratory tract symptoms mimicking a mild croup attack. These patients were treated with humidified oxygen via a mask. The remaining 29 patients were hospitalized. The presenting symptoms of the participant were only cough ($n=23$, 46%), cough plus dyspnea ($n=30$, 60%), nausea plus vomiting ($n=6$, 12%), headache ($n=7$, 14%), and sore throat ($n=3$, 6%). None of the patients was diagnosed with respiratory failure or acute pulmonary edema. Invasive or non-invasive airway support was not required. Seven patients were discharged on day 1, 21 patients on day 2, and 1 patient on day 3.

Regarding investigations performed at the hospital, 19 (38%) patients with respiratory distress were evaluated with a chest X-ray (CXR) which showed no pathology. They were also evaluated with an electrocardiogram (ECG) and arterial blood gas (ABG) analysis. Five patients (M/F = 1/4) had sinus tachycardia on ECG. CK-MB levels of these patients were above normal range (mean: 78 U/L, range: 46-278). The presenting symptoms of these patients were only cough ($n=1$), cough plus dyspnea ($n=3$) and sore throat ($n=2$). During the follow-up period, CK-MB levels returned to normal levels.

Six patients had hypercarbia and respiratory acidosis; 2 had metabolic acidosis; 2 had metabolic alkalosis; and 2 had respiratory alkalosis. Troponin levels were analysed at time of admission. All of the patients levels were in normal range. Thirty patients had elevated creatine kinase-MB (CK-MB) (mean: 54.9 ± 50.1 U/L) (Normal range: 0-25 U/L). During the follow-up period, sinus tachycardia disappeared and the CK-MB levels normalized. There was no abnormal result in complete blood count (CBC), transaminases, urea, and creatinine testing. During the follow-up period, cardiac enzymes of all patients returned to normal levels.

The proportion of asymptomatic female patients was significantly greater than that of the males (52.6% vs 25.8%; $p=0.04$). There was no significant difference between the mean ages (10.9 ± 1.4 vs 11.5 ± 1.6 years; $p=0.289$, for males and females, respectively) and CK MB levels (63.6 ± 16.5 vs 47.1 ± 29.8 U/L; $p=0.369$, for males and females, respectively) of the two sexes.

Seven patients who had signs and symptoms associated with upper or lower airway problems were treated with inhaled epinephrine, intravenous steroid (Dexametazon: 0.6 mg/kg), inhaled bronchodilators, and humidified oxygen; 11 patients were treated with oxygen and bronchodilators; the remainders took oxygen alone.

DISCUSSION

Sources of Cl₂ gas such as household disinfectants and swimming pool chlorinators make this agent potentially dangerous for children (3,4). There are some clinical reports in the pediatric age (5,6). Many reports have shown that the most prevalent complaints were pruritus, excessive lacrimation, rhinorrhea, conjunctival irritation, oropharyngeal pruritus, cough, sore throat, laryngeal stridor, and dyspnea (4,7,8). In our series, the most prevalent symptoms were cough and dyspnea.

After its inhalation, Cl₂ is transformed into hypochlorous acid (HOCl) and hydrochloric acid (HCl) through chemical reactions in human body (9). These products react with cellular proteins, nucleic acids, and lipids of the cells that line the airway epithelium, causing an inflammatory reaction in the alveolar space (10-11). This is why steroids are indicated in moderate-to-severe airway exposure. Seven of our patients with moderate exposure were treated with intravenous steroids. We did not know how much Cl₂ was released into the air. Evans reported that doses below 0.5 ppm can cause tickling of the nose and the throat, itching of the nose, coughing, burning of the eyes, and dryness of the throat (12). The fatal dose ranges from 50 to 2.000 ppm. So, it is obvious that a fatal dose was not released in our series.

There is no specific antidote for the treatment of Cl₂ exposure and the management is largely supportive (13). Guloglu et al. (4) recommended a combination of humidified O₂ and β agonist as supportive therapy in their study group because trachea-bronchitis and bronchoconstriction and/or pulmonary edema may develop in these cases. Moreover, Agabiti et al. (5) suggested the use of intravenous cortisone and humidified O₂ in hospitalized moderate and severe cases. Seven of our patients had signs and symptoms of tracheobronchitis. Thus, they were treated with intravenous steroids, humidified oxygen, and bronchodilator.

A baseline CXR should be obtained if a patient is symptomatic; additionally, respiratory functions should be monitored with ABG and pulse oximetry (14). Chest radiograms can show diffuse nodular opacities, patchy consolidation, pulmonary edema, and signs of vascular congestion. Radiologically, air

trapping can be seen in cases with persistent hyper reactivity and airflow obstruction. Our patients had no pathological sign on CXR. Cardiotoxicity is another important complication of Cl₂ toxicity. Chlorine inhalation attenuates myocardial contractile force, reduces systolic and diastolic blood pressures, and may cause cardiac failure and even death. The most common electrocardiographic finding is sinus tachycardia (15). Guloglu et al. (4) detected ST depression and sinus tachycardia in one patient and premature beats in another (only a total of 2 patients with ECG abnormalities were reported) among 18 cases. In our study, five patients had sinus tachycardia, however their troponin levels were normal at the time of admission. During the follow-up period, sinus tachycardia disappeared.

Girls were more severely affected than boys in our study. Considering that the severity of symptoms increase with the duration of exposure, it is thought that girls' departure from the accident area may be slower than boys and therefore they may have been exposed to Cl₂ for a longer time. In addition, considering that a significant part of the symptoms are related to anxiety (panic attack), it seems compatible with the high rate of these conditions in girls (16). The effect of a fear-panic state experienced collectively in mass incidents cannot be ignored, either. An observation and supportive approach should be applied for a final decision.

Twelve patients had different types of metabolic acid-base disturbances. Chlorine gas inhalation is usually accompanied by metabolic acidosis that has been attributed to lactic acidosis (17). On the other way, the presence of hypercarbia can lead to respiratory acidosis (18). Hyperventilation leads to respiratory alkalosis (19). However, two patients had metabolic alkalosis that we did not expect to encounter in chlorine intoxication. It may be a laboratory error.

Moreover we applied Haddon matrix to chlorine toxicity (Table I). A matrix looks at factors related to personal, agent and environmental attributes; before, during and after an injury (20). In this way, we aimed to ensure that such events do not recur.

The present study has a retrospective nature and is thus limited by several limitations. Firstly, the review of medical records mainly focused on common, recognizable symptoms. Subtle

Table I: Haddon matrix applied to acute chlorine poisoning.

	Victims	Agent Toxic substance	Environment Factors
Pre injury	Teach children not to play with unknown substances especially hazardous chemicals	Teach cleaning staff no to mix bleach and hydrochloric acid	Schools should establish a chemical storage and handling policy that addresses how chemicals should be properly stored, labeled, and secured.
Injury	Teach children how to respond to gas toxicity	Prevent gas contact with the students	Emptying the room, open the windows
Postinjury (after child injured)	Provide first aid	Put chemicals away from children	Put a warning note

toxicity was not evident in the review of medical records. Secondly, there was no measurable method for the correlation of the severity of Cl intoxication and symptoms.

In conclusion, serious undesirable situations including intoxication may develop after negligent and careless use of simple household chemicals. Accidental mass intoxication incidents, albeit rare, are important acute situations for which healthcare providers should be prepared. A good infrastructure should be available for rapid organization, medical treatment, and access to drugs and material. A similar one has been experienced during the more serious pandemic process. We are of the opinion that physicians should increase their interest in chemical reactions that may produce toxic substances such as in our cases.

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