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Characteristic manifestations of acute paint thinner-intoxicated children

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ABSTRACT

In this study, we evaluated the acute clinical and para-clinical abnormalities arising from paint thinner (PT) poisoning in children. Data were collected from 2008 to 2013, and sourced from the medical records of PT-intoxicated children and through questionnaires. A total of 42 children were enrolled in the study. The mean age was 37.2 ± 2.4 months. The participants ranged from10 to 96 months of age; with a median age of 3 months; and a modal age of 24 months. The sex ratio in the study was 22 males (64.9%) to 20 females (35.1%). Clinical biochemistry results of participants revealed the significant presence of alkaline phosphatase (ALP, 569.25 ± 151.58 U/L), and lactate dehydrogenase (LDH, 576.14 ± 164.97 IU/L). Arterial blood gas (ABG) analysis was also carried out. Chest X-ray predominantly revealed right side alveolar air space consolidation. These results confirmed hepatotoxicity and pneumonia in PT-intoxicated children. The study also revealed that positive outcomes were achieved in patients with early treatment and management. In addition, the current finding confirmed the timely transfer of the victim to the poisoning center.

1. Introduction

Paint thinners (PTs) are a mixture of various aromatic hydrocarbons, halogenated hydrocarbons and naphtha such as toluene, xylene, and N-hexane. They are widely utilized in industry for the production of plastics, varnish, paint and glue; and are commonly utilized as solvent in painting and decorating applications such as thinning oil-based paint, cleaning brushes and removing household paints (Singh et al., 2012; Verma and Gomber, 2009; Solak et al., 2006). Due to the nature of its applications, PT is often found in domestic households and may be readily accessible to children if not properly stored. PTs may be accidentally ingested by children, who are then transferred to toxicology centers for poisoning treatment.

PT-intoxication among children is one of the most serious problems worldwide. Methemoglobinemia may be acquired or congenital, and is characterized by higher than normal levels of methemoglobin (MetHb) in the blood which results in decreased oxygen release ability to tissues and tissue hypoxia if MetHb is in high concentration. In the former case, methemoglobia may be caused by ingestion of compounds containing nitrates. Cytochrome (CYP)-b5 reductase enzyme, the key MetHb reduction enzyme, is presents at lower levels in infants (under 6 months) than in adults, thereby making children more susceptible to methemoglobinemia from PT exposure than adults (Verma and Gomber, 2009). The problem is further aggravated because of the promoted growth of nitrite-producing gram-negative bacteria with increased alkalinity in the gastrointestinal tract of infants. Nevertheless, children manifested marked clinical symptoms of intoxication such as cyanosis, mild tachypnea and less or no presentation of central nervous system (CNS) or gastrointestinal problems (Verma and Gomber, 2009).

Chronic PT intoxication is caused by the long-term abuse or misuse of solvents. The routes of exposure are inhalation, injection and consumption (Per Oral) (Shinya et al., 2003; Rahimi et al., 2015), and are associated with elevated MetHb levels, hepatotoxi-

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Table 1
Demographic characteristics of paint thinner (PT)-intoxicatedchildren.

Demographic data	N	Range	Mean ± SD	Percent
Sum of patients (child) involved	42			
Age (months)		10–96	37.19 ± 24.38	
Sex				
Male	22			64.86
Women	20			35.14
Amount of dose (mL) ingested		3–125	22.36 ± 32.42	
Time of ingestion (min)		30–2880	209.33 ± 451.65	
Mean duration of stay in the poisoning section (day)			1–7	

city, neurotoxicity, myotoxicity, nephrotoxicity and cardiotoxicity (Singh et al., 2012; Verma and Gomber, 2009; Solak et al., 2006).

PT can generate reactive oxygen species (ROS), thereby causing oxidative stress (OS). Neurotoxic effect is related to current reactants. Moreover, exposures to PT cause a significant increase in lipid peroxidation (LPO) products such as malondialdehyde (MDA) and 4-hydroxylamines in all brain regions (Baydas et al., 2003). PT poisoning leads to unfavorable manifestations such as cyanosis with minimal tachypnea and chocolate-brown-colored blood (Solak et al., 2006).

The common biochemical markers of PT poisoning are elevated ALP and LDH serum levels above the normal range. These markers are associated with tissue damage and could indicate the development of hepato- or pulmonary toxicity in intoxicated patients (Rahimi et al., 2015). Toluene inhalation has also been reported to cause a substantial rise in hepatic activity enzymes, including serum AST and ALT. During thinner exposure, increased levels of MDA occur due to histopathological changes including massive hepatocyte degeneration, ballooning degeneration and mild pericentral fibrosis which indicate tissue damage and the risk of hepatotoxicity (Tas et al., 2011). To the best of our knowledge, little information is available in published literature on the characteristics of PT poisoning in children. This study therefore aims to evaluate the acute clinical and para-clinical abnormalities among juvenile-age PT poisoning with a view to improving treatment and management.

2. Methods

2.1. Subjects

The study design was retrospective, descriptive. It was carried out from June 2008 to September 2013 at Shahid Beheshti University of Medical Sciences (SBMUS), Loghman-Hakim General Teaching Hospital, Poisoning Center, Tehran, Iran. The exclusion criteria included all patients with co-ingestion of PT and other hydrocarbons or drugs, and/or incomplete medical records. A selection criterion including lower age limit of 12.79 and upper age limit of 61.55 was set. A total of 42 patients were selected and participated in the study.

2.2. Data collection

The data of patients were collected through questionnaire. Demographic and vital signs were recorded at the emergency department (ED) of hospital. Instructive toxicology information and Glasgow Coma Scale (GCS) were completed in post-emergency ward, and included time of toxicities (minutes), ingested dose (mL), cause of intoxication, patients' outcome and any management received by the subjects. The laboratory profile comprised kidney function test, liver function testing, complete blood count and differentiation, arterial blood gas analysis and electrolytes. A chest X-ray was performed in the emergency ward. All patients received standard supportive and symptomatic treatment. Fluids containing essential electrolytes were infused under the supervision of a clinical toxicologist.

Table 2
Vital signs of PT-intoxicatedchildren.

Patients' vital signsin theED	Range	Mean ± SD
RR (numbers/min)	15–80	30.69 ± 13.46
PR (beats/min)	76–144	106.74 ± 15.07
SBP (mmHg)	75–150	94.16 ± 11.94
DBP (mmHg)	50–85	63.33 ± 8.09
Temperature (°C)	35.8–38.8	36.86 ± 0.65

2.3. Statistical analysis

All data were statistically analyzed using SPSS software (SPSS version 13, USA) to produce values for mean ± standard deviation (SD). Microsoft Excel 2011 software was also utilized for presenting the frequency data. The sample studied had normal distribution with Kolmogorov-Smirnov, Shapiro-Wilk normality test at $P > 0.05$. A comparison of means was performed with independent sample *t*-test. A two-tailed p-value *p*-value of $P < 0.05$ was utilized in the study.

3. Results

3.1. General findings

A total of 42 patients comprising 22 male (64.9%) and 20 (35.1%) female patients participated in the study. The mean age was 37.17 ± 24.38 months; and the age range was 10–96 months. Table 1 illustrates the demographic characteristics of PT intoxicated-children. The largest proportion of PT-intoxicated patients mistakenly consumed thinner as a liquid. The average time between ingestion and presenting at the poison center was 209.36 min (ranging from 30 to 2880 min). The duration of hospitalization was up to seven days. Nevertheless, patients were followed up for several days after they have been discharged from the hospital.

Table 2 shows the vital signs in the PT-intoxicated children. Most of the victims were conscious on their arrival at the hospital 41 (97.6%) and had a Glasgow Coma Scale (GCS) of 15/15 and was in line with the fact that the parents had full awareness of what had occurred. Patients made a full recovery while in hospital or were followed up as outpatients after they have been discharged.

3.2. Chest-X-ray results

Fig. 1 illustrates the chest X-ray alveolar pattern distribution in the respiratory system of PT-intoxicated children. The chest-X-ray interpretations were normal for 61.9% of the study participants (designated 'Group A'). Alveolar consolidation was detected in the right pulmonary in the region of the paracardiac and parahilar, and was diagnosed as pneumonia for 28% of patients (Group B). Group C patients, which comprised 4.76% of participants, had left sided diffuse alveolar pattern in the region of paracardiac opacity, and