CASE REPORT

SUDDEN DEATH DUE TO VOLUNTARY LIGHTER FLUID INHALATION: A CASE REPORT

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ABSTRACT

The abuse of hydrocarbon by inhalation in adolescent ages has increased in last year due to the euphoric effect it provokes. Butane, which is a kind of hydrocarbon compound, may be abused by inhalation and thus lead to addiction. Inhaling lighter fluid containing butane can cause lung toxicity, brain damage, myocardial effects, and neurologic problems. We reported, in parallel to literature, a 19-year-old patient who inhaled lighter fluid and who presented with lung haemorrhage, hypoxia and acute coronary syndrome.

Key-Words: Butane Inhalation; Sudden Death; Adolescent Ages

Introduction

Hydrocarbons are primarily consisting of carbon and hydrogen molecules.1 Employees of petrochemistry, plastic industry, furniture and printing house may be exposed to these substances. At home, people may be exposed to products containing hydrocarbons accidentally or voluntarily. Butane, which is a kind of hydrocarbon compound, may be abused by inhalation and thus lead to addiction.[1] In this study, we aimed to present and discuss, in parallel to literature, a 19-year-old patient who inhaled lighter fluid and who presented with lung haemorrhage, hypoxia and acute coronary syndrome.

Case Report

A 19-year-old male was found unconscious in his room with a 270 ml lighter fluid tube containing "butane". First aid to the patient was provided by emergency assistance team who reached the place within 3.5 minutes. The team started the resuscitation on the patient who was gasping and in cardiopulmonary arrest. Following the intervention that lasted 45 minutes, the rhythm of the patient returned. After hospitalization, his history obtained from the relatives revealed that he was addicted to synthetic glue inhalation for approximately 4 years, and that he was always hindering the treatment. After intubation at hospital, the vital signs were: arterial blood pressure: 124/46 mmHg, heart rate: 94 beats/ minute and respiratory rate: 28 breaths/minute. In the neurological examination, pupils were isochoric with no light reflex bilaterally, and the Glasgow Coma Scale Score was 4 ($E_1M_2V_1$) intubated. Auscultation of the lungs was positive for bilateral coarse rales, and haemorrhagic fluid was aspirated from the endotracheal tube with frequent intervals. Cranial computed tomography (CT) showed a diffuse parenchymal oedema with no acute parenchymal bleeding or space occupying lesion (figure 1). Thorax CT showed ground glass appearance especially at the basal regions of bilateral lungs corresponding to an alveolar pattern. Laboratory values obtained from the arterial blood samples were: pH: 7.49; PaO_2: 51 mmHg; $PaCO_2$: 24 mmHg; HCO_3: 19,8 mEq/L and the oxygen saturation was 89%. Electrocardiography (ECG) showed sinus rhythm and an elevation at the ST segments of the V3-V4 derivations (figure 2). Bedside echocardiography revealed 55% ejection fraction with no segmental wall motion abnormalities or remarkable valvular problems. Other laboratory test results were as: leucocytes: 19.3 thousand/L (4-10); aspartate aminotransferase: 376 U/L (15-40); alanine aminotransferase: 89 U/L (15-40); creatine kinase

(CK): 1091 U/L (0-190); CK-MB: 274 U/L (0-24); Troponin I: 10, 38 ng/ml (0-0,16).

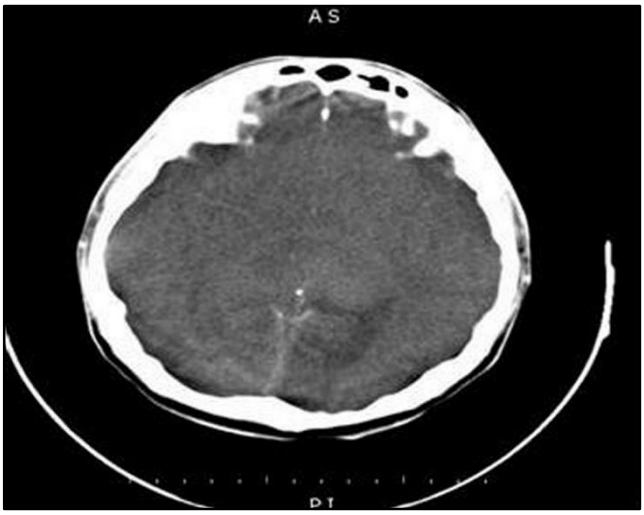

Figure-1: Diffuse Cerebral Oedema

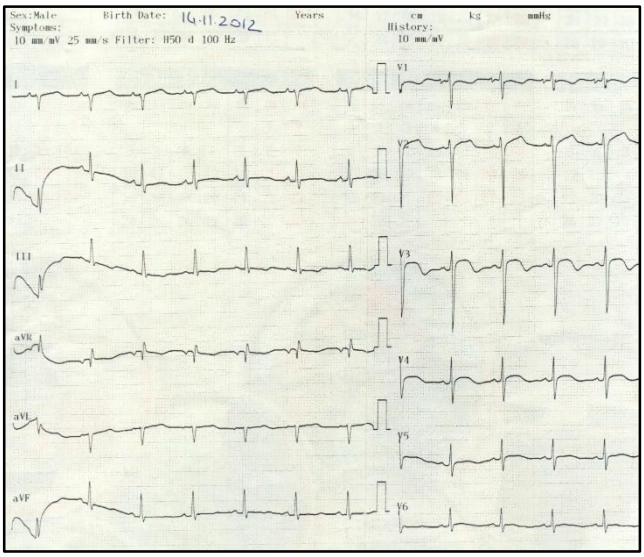

Figure-2: ST Segment Elevation in Anterior Derivations

Repeat cranial CT on the 4th day of hospitalization demonstrated signs of cerebral edema with diffuse effacement of the cerebral sulci (figure 2). 150 ml of 20% mannitol infusion 6 times/day was started as anti-oedema treatment. To maintain the patency of the vascular access, 100 mL/hr i.v. normal saline was infused. Synchronized Intermittent Mandatory Ventilation mode of the mechanical ventilator was used for volume focused respiratory support. At a fraction of inspired oxygen of 60%, blood gas values were as: pH: 7.28; PaO_2: 94 mmHg; $PaCO_2$: 35 mmHg. Since the clinical and radiological findings of the patient necessitated a prolonged duration of mechanical ventilation, percutaneous tracheostomy was performed on the 8th day of hospitalization. On the 2nd day of the monitoring, in addition to 20 kcal/hr enteral feeding via a tube, parenteral nutrition including 20 mL/hr 20% dextrose and 30 ml/hr 8% aminoacid solution (Hepatamine, Eczacıbaşı-Baxter) was started. Central venous pressure monitoring was performed to control the liquid electrolyte balance. The patient died at the 27th day of the hospitalization due to multi organ failure.

Discussion

The abuse of hydrocarbon by inhalation in adolescent ages has increased in last year due to the euphoric effect it provokes. According to the data of American Association of Poison Control Centers for 2006, the hydrocarbon poisoning rate is 2% among all the poisoning cases.[1] Half of these patients include ≤19 year old individuals. In Turkey, this rate is around 5.4%.[2] These substances are abused by either sniffing, inhalation using a rag (huffing) or inhalation using a bag (bagging).[3] In our case, the patient is from the adolescent age group and used lighter fluid by bagging.

Butane, an aliphatic hydrocarbon, is used as lighter fluid. It is a volatile substance that transforms from liquid to gaseous phase. Like many other hydrocarbons, it passes through the alveoli into the blood stream by passive diffusion. It has a high dissolution rate in blood and tissues and is easily absorbed. It evaporates and takes the place of oxygen, thus leading to a hypoxic state and lung toxicity. Lungs are the organs that are affected most following exposure.[4]

Brain may be directly damaged with the systemic absorption of hydrocarbon substances. Simple asphyxia and anoxia resulting from severe hypoxia or bagging may lead to indirect damage. Moreover, with bagging, re-inhalation of the exhaled air may lead to hypercarbia and thus provoke severe brain oedema.[3,5] The above mechanisms and the hypoxia during cardiopulmonary arrest may have contributed to the cerebral oedema in our patient (figure 1).

The systemic absorption of halogenated hydrocarbons such as butane may lead to atrial fibrillation, ventricular tachycardia, ventricular fibrillation, cardiomegaly and myocardial

infarction.[6] Tachyarrhythmias and coronary vasospasm occur due to sensitization of the myocardium to the effects of catecholamine. [3] In our patient, ST elevation on ECG, and elevated CK, CK-MB and Troponin I levels on biochemical analyses were observed (figure 2). Because of the normalization of the cardiac biomarkers and ECG findings on repeat tests, and the bad general situation of the patient, coronary angiography was not performed.

Conclusion

Inhalation of lighter fluid whose incidence in the adolescent age group has increased nowadays, may lead to fatal results by affecting vital organs such as lungs, brain and heart. Our case exemplifies the presentation and the severity of the events that can be seen in such a case.

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