Fatal butane inhalation from gas cartridges: a case report and literature review

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Abstract: Volatile substances are commonly misused with easy-to-obtain commercial products, such as glue, shoe polish, nail polish remover, butane lighter fluid, gasoline, and computer duster spray. This report describes a case of sudden death of a 29-year-old woman after presumably inhaling gas cartridge butane from a plastic bag. Autopsy, pathological and toxicological analyses were performed in order to determine the cause of death. Pulmonary edema was observed pathologically, and the toxicological study revealed 2.1μL/mL of butane from the blood. The causes of death from inhalation of volatile substances have been explained by four mechanisms; cardiac arrhythmia, anoxia, respiratory depression, and vagal inhibition. In this case, the cause of death was determined to be asphyxia from anoxia. Additionally, we have gathered fatal butane inhalation cases with quantitative analyses of butane concentrations, and reviewed other reports describing volatile substance abuse worldwide.

Key Words: forensic science, butane, inhalation, volatile substance, autopsy, toxicology.

Volatile substances are those that vaporize at ambient temperatures, and they are commonly misused with easy-to-obtain commercial products, such as glue, shoe polish, nail polish remover, butane lighter fluid, gasoline, and computer duster spray [1]. There are several methods by which these volatile substances can be inhaled: (1) bagging, whereby volatile substances are placed into a plastic bag and inhaled; (2) sniffing, whereby volatile substances are inhaled directly from the container; (3) spraying directly into the oral or nasal cavities; and (4) huffing, whereby volatile substances are inhaled from a substance-soaked cloth placed over the nose or mouth [1].

Butane, one of the most common volatile substances, has the chemical formula C₄H₁₀, with a molecular weight of 58.12. It is highly flammable, colourless, and odourless. Butane and many other volatile substances cross the blood–brain barrier rapidly due to their nonpolar, hydrophobic, lipophilic characteristics, where the concentration peaks are observed within 1 to 3 minutes after inhalation [2, 3].

The most common effects of volatile inhalant use are euphoria, relaxation, vertigo, and headache [4]. According to Garland, 63.7% and 62.9% of adolescent inhalant users experienced euphoria and relaxation, respectively, and volatile substances are presumably inhaled for these positive effects.

This report describes a case of sudden death, presumably after inhaling gas-cartridge butane from a plastic bag. An autopsy and pathological and toxicological analyses were performed in order to determine the cause of death. Furthermore, we have gathered fatal butane inhalation cases with quantitative analyses of butane concentrations and reviewed other reports describing volatile substance abuse worldwide.

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CASE

The night before her 30th birthday, a 29-year-old woman was found dead, face down, with her lower body under a kotatsu table (Fig. 1). She was found with a plastic bag attached to her mouth. Butane gas cartridges (liquefied butane; presumably greater than 70% butane) were discovered inside the house: 1 on top of the kotatsu table was almost full, 10 near the table were empty, 1 near the table was half full, and 9 in the kitchen were full. Additionally, there were about 50 more empty gas cartridges in the house (Fig. 2). She had learned how to inhale gas from gas cartridges from her ex-boyfriend and had continued the practice ever since.

Moreover, blister packages located on the table had 2 empty spaces for Paxil® (paroxetine, antidepressant), 1 empty space for Desyrel® (trazodone, antidepressant), 1 empty space for Lendormin® (brotizolam, sedative-hypnotic thienodiazepine), and 1 empty space for Seroquel® (quetiapine, antipsychotic). The woman had been visiting a hospital for mental instability on a regular basis.

A forensic autopsy was performed 3 days after the discovery of her death in order to specify the cause of death.

Autopsy findings

The body was 147.0 cm in height and weighed 52.0 kg. Bright red liver mortis was present on the anterior surface of the body. Numerous petechiae were observed on the palpebral conjunctiva, lungs, anterior neck, upper chest, and axillary regions. There were several subcutaneous haemorrhages on the lower extremities ranging from 1.0 cm×1.0 cm to 3.0 cm×2.5 cm. There were 6 circular scars of 1.0 cm to 1.5 cm in diameter, which resemble scars from cigarette burns. No apparent traumatic lesions related to the woman’s death were discovered. The heart weighed 255 g, with no apparent arteriosclerosis of coronary arteries. The colour of cardiac blood was dark red and did not contain any coagulation. Viscous fluid with fine foam was observed in the trachea and bronchi. Organ weights were as follows: right lung 345 g, left lung 375 g, liver 1,260 g, spleen 94.1 g, pancreas 95 g, right adrenal gland 4.3 g, left adrenal gland 4.1 g, right kidney 110.1 g, left kidney 105.0 g, and brain 1,330 g.

Pathological findings

Moderate to severe congestion was evident in most of the organs. Severe fat deposition and mild to moderate adipose infiltration in the heart, pulmonary edema (Fig. 3), and sinusoidal dilation of the liver were observed. No apparent inflammatory lesions or natural diseases related to the woman’s death were found in any organ.

Toxicology

The concentration of butane in blood samples was determined in triplicate by gas chromatography (GC) using a modified method of Puke [5]. Blood samples (0.5 mL) were placed into a sealed vial with an internal standard solution containing 0.5 mL of 0.15% acetone. The vial was heated at 60°C for 30 minutes and then cooled down to room temperature. A 1.0 mL headspace sample was injected into the GC. Controls were prepared by adding standard gases (butane, GL sciences; balance...
gas, nitrogen) after the fortification of 0.5 mL of distilled water and an internal standard solution containing 0.5 mL of 0.15% acetone. Controls were conditioned in the same manner as previously described. The horizontal axis was designated as the amount of standard gas added and the longitudinal axis as the butane and internal standard area ratio. An approximation formula was calculated from the data, and the concentration of butane in the blood was determined.

Samples were analysed by GC/MS using an Agilent 6890N with a flame ionization detector. Separation of components was achieved using a 30 m GS-Q capillary column (J&W, 0.53 mm i.d.). The column temperature was maintained at 120°C. The injector and detector temperatures were set at 200 and 250°C, respectively. Helium was used as a carrier gas at a flow rate of 5.8 mL/min.

The blood alcohol concentrations were below the detection limit. The qualitative toxicological analysis revealed the presence of paroxetine and trazodone, which are main ingredients of antidepressant drugs. The concentration of butane in the blood was determined to be 2.1 μL/mL.

**DISCUSSION**

The causes of death from inhalation of volatile substances have been determined to be cardiac arrhythmia, anoxia, respiratory depression, and vagal inhibition [6].

Cardiac arrhythmia is considered to be the most probable cause of death by inhalation of volatile substances. The association between inhalation of volatile anesthetics and cardiac arrhythmias has been well documented within the long history of anaesthesiology [7–9]. Adrenaline, also known as epinephrine, is a hormone secreted in the medulla of adrenal glands and also at the end of sympathetic nerves in response to fight-or-flight situations, such as stress and fear. It is a powerful vasoconstrictor and stimulates the heart [10]. Adrenaline is used to treat cardiac arrest and cardiac dysrhythmia. However, the higher the adrenaline level, the greater the cardiovascular effect and the greater the likelihood of triggering arrhythmias [6, 9]. Shepherd suggests that hallucinations caused by inhalant intoxication provoke disturbing fear and increase the level of adrenaline. Indeed, auditory and visual hallucinations are experienced by adolescent inhalant abusers [4].

Anoxia is a total depletion of oxygen. There must be occlusion of the airways or a decrease in the oxygen concentration of the inhaled air in order to induce anoxia. Anoxia is predominantly associated with inhalation of volatile substances from a plastic bag. As an individual inhales the volatile substance, the consciousness gradually declines, and it becomes difficult for that individual to remove the bag. This situation would result in anoxia and thus causes death [6].

Volatile substances easily migrate to the central nervous system (CNS). Inhalation of high concentrations of volatile substances would cause respiratory depression and, ultimately, respiratory arrest [3, 6]. Clark exhibited experimentally that exposures to either a CNS depressant or a CNS stimulant resulted in narcosis, shallow respiration, and eventually death from respiratory depression [11]. Although it is often thought that the major causes of death from inhaling volatile substances are cardiac arrhythmia and anoxia, Cronk et al. reported a case of a patient admitted to a hospital due to inhaling a volatile substance [12]. His electrocardiogram only exhibited sinus tachycardia, while he repeatedly suffered respiratory arrest after numerous resuscitation attempts. This case report illustrated that respiratory arrest alone could be a significant cause of sudden death in volatile substance inhalation.

Vagal inhibition of the heart is the over activity of the parasympathetic nervous system from stimulation of the laryngeal or pharyngeal mucosa. Such inhibition may occur when swallowing a bolus of food or upon sudden entry of cold water into the pharynx and larynx [13], which resembles the rapid cooling of the mouth or airways by spraying volatile substance gases into those orifices [14].

As previously mentioned, volatile substances can be found in commercial products, such as glue, shoe polish, nail polish remover, lighter fluid, gasoline, and computer duster spray. Depending on the product, gas components could be 100% butane or mixtures of butane, isobutane, propane, and other gases. Table 1 shows 15 fatal butane inhalation case reports, including this case, displaying the blood butane concentration, cause of death, and age and gender of the deceased. All but one case are reports from Japan. This might be because commercially available volatile substances consist mainly of butane in Japan, and the major contents of gas products in other countries may be different.

According to Table 1 [15-26], the blood butane concentrations range from 0.11 to 15.3 μL/mL. The most common cause of death was arrhythmia (6 cases), followed by asphyxia due to anoxia (5 cases). Two cases involved a combination of multiple causes. The causes of death for the other 2 cases were determined to be butane poisoning and asphyxia by inhalation of gastric contents. In cases where it was evident that inhalation of gastric contents or vomit caused asphyxia, these were concluded to be the cause of death, regardless of the blood butane concentration. If it was evident that the deceased was using a plastic bag immediately before death to inhale butane, commonly called “bagging,” and the inhalation of butane was proved by toxicological analysis, the cause of death was determined to be asphyxia due to anoxia. On the other hand, the cause of death was decided to be arrhythmia in cases where bagging was denied or the
deceased was able to move for a short time after bagging. It seems there is congestion of internal organs and lung edema to some degree [27–29]; however, there are no pathological findings specific to butane inhalation. When relatively high butane concentrations were observed and no other possible causes of death were present, the cause of death was determined to be butane poisoning [25, 26]. In the present case, the woman was found with a plastic bag attached to her mouth, suggesting that she was inhaling gas-cartridge butane from this plastic bag. Autopsy findings, such as multiple petechiae, congestion of internal organs, and the absence of coagulated blood in the heart, indicated that the woman had an acute course from the beginning of the inhalation to the time of death. Pathological analysis also confirmed moderate to severe congestion of most organs. Pulmonary edema was observed microscopically; nevertheless, this finding was not specific to butane inhalation. The qualitative toxicological analysis revealed the presence of paroxetine and trazodone, which are main ingredients of antidepressant drugs. Trazodone is an antidepressant, known to depress the central nervous system. Although a quantitative analysis was not performed, trazodone might have had some competitive impact along with the respiratory depression effect of butane itself, similar to the case reported by Tanaka et al. [25]. The concentration of butane in the blood was determined to be 2.1 μL/mL, yet this value was not high compared to other cases (Table 1). Since the woman was supposedly inhaling butane from a plastic bag, the cause of death was determined to be asphyxia due to anoxia.

By comparing cases of fatal butane inhalation, it could be stated that butane abusers consist mainly of teenagers (Table 1). It is plausible that this is because butane is commercially available in the form of lighter fluid, aerosol spray, and gas cartridges, which could easily be purchased by teenagers [30]. According to Johnston et al., the lifetime prevalence of inhalants for 8th graders, 10th graders, 12th graders, college students, and young adults (ages 19 to 28) has been decreasing in the United States in the 12 years of their study [31]. However, the 2011 ESPAD report by the European Monitoring Centre for Drugs and Drug Addition reported increases in the percentage of lifetime use of inhalants in 15 out of 36 countries they have monitored [32]. Eleven countries displayed no change, and only 7 countries demonstrated some decrease. Another study in Australia has shown that about 17.3% of students from age 12 to 17 have ever used inhalants in their lifetime [33]. This report summarizes that the percentage of students (ages 12 to 15) who had used inhalants in their lifetime in 2011 was no different from that in 2008 and 2005.

<table>
<thead>
<tr>
<th>Butane concentration in blood (μL/mL)</th>
<th>Cause of death</th>
<th>Age Gender</th>
<th>Suspected Inhalation Method</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.65</td>
<td>Arrhythmia</td>
<td>16M</td>
<td>Bagging</td>
<td>Aoki T et al. Acta Crim Jpn 1982; 48: 202-204. (17)</td>
</tr>
<tr>
<td>0.94</td>
<td>Arrhythmia/lung edema</td>
<td>13M</td>
<td>Bagging</td>
<td>Nishi K et al. Jpn J Legal Med 1985; 39: 214-216. (18)</td>
</tr>
<tr>
<td>2.1</td>
<td>Arrhythmia</td>
<td>29F</td>
<td>Bagging</td>
<td>This report</td>
</tr>
<tr>
<td>2.4</td>
<td>Arrhythmia</td>
<td>14M</td>
<td>Bagging or sniffing</td>
<td>Fuke C et al. Legal Med 2002; 4; 134-138. (5)</td>
</tr>
<tr>
<td>6.8</td>
<td>Arrhythmia</td>
<td>23F</td>
<td>No bagging</td>
<td>Tanaka N et al. Soud Lek 2010; 4: 44-45. (22)</td>
</tr>
<tr>
<td>15.3</td>
<td>Arrhythmia</td>
<td>15M</td>
<td>Unknown</td>
<td>Ago M et al. Legal Med 2002; 4; 133-118. (26)</td>
</tr>
</tbody>
</table>
Moreover, the male-to-female ratio in Table 1 was 4:1 (80% male). A reasonable explanation for this phenomenon is uncertain, yet 74%, 84.6%, 92.3%, and 83.8% of inhalant users were male in other studies (34–37, respectively). However, in Australia, slightly greater percentages of female students (ages 12 to 15) who used inhalants over their lifetime were observed [33]. Some reports not included in Table 1 have illustrated fatal butane inhalation by male teenagers [28, 38]. Table 1 shows the cases of only 3 females, which were all reported after 2010. The inhalant abusers consist not only of male teenagers, the possibility an increase in the number of female abusers should be considered. More effective warnings should be given about the dangers and fatalities of inhaling volatile substances, including butane.

The present case described fatal butane inhalation, in which we concluded that the cause of death was asphyxia due to anoxia. Many studies over several years have reported on volatile substances and their fatal outcomes; yet there seem to be no specific remarks concluding that the cause of death is cardiac arrhythmia, anoxia, respiratory depression, or vagal inhibition. Forensic pathologists need to consider the possibility of volatile substance inhalation when sudden deaths are reported, especially in teenagers and young adults.

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References