Case report
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Rhabdomyolysis and thinner intoxication

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Abstract

We report a case of paint thinner intoxication by inhalation, with rhabdomyolysis, renal failure, skin lesions and severe sciatic nerve lesion at gluteal region.

Keywords: thinner intoxication, rhabdomyolysis, acute renal failure.

Introduction

During the battle of Britain in World War II, Bywaters and Beall described the first causative association of ARF with the release of muscle cell contents including myoglobin after a crush injury to muscle. Victims frequently had reddish brown urine, oliguria subsequently developed, and they died of ARF. Over the past 50 years it has become apparent that rhabdomyolysis with subsequent development of ARF may follow a host of other causes. Nonetheless, the majority of cases are attributable to either prodigious exercise, trauma, or alcohol abuse.

Rhabdomyolysis due to aromatic hydrocarbons such as toluene and thinner are rare [1,2].

Akisu [2] reported a case who had rhabdomyolysis, polyneuropathy and chemical pneumonia due to ingestion of 200 ml of thinner in 1996 .

To the best of our knowledge rhabdomyolysis due to inhalation of thinner has not been reported yet.

This is the first case reported in the literature to survive acute thinner intoxication by inhalation with rhabdomyolysis.

Case report

A 24-year-old male wall painter was admitted to the emergency department of this hospital on August 12, 2006 because of burns with thinner and dyspnea.

The patient was painting a wall and using thinner a day before when he developed inhalation poisoning and skin burns with thinner. He had been trapped in a confined space for more than 20 hours, had been unconscious for some hours and did not remember the minutes. He was brought to the emergency room by EMS.

On arrival, he was conscious and had a GCS of 15/15. The temperature was 37°C, blood pressure 110/70 mm Hg, pulse 100 beats per minute and respiratory rate 28 per minute.

On physical examination, he had tachypnea and tachycardia. There was a 2×2 cm first degree burn on right hand fingers, a 7×10 cm first degree burn on posterior lower chest, a 20× 20cm first degree burn over the right buttock and a first degree burns on the toes of both feet

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Creatintine (mg/dl)	3	Aspartate aminotransferase (U/I)	189
Blood-urea-nitrogen (mg/dl)	38	Alanine aminotransferase (U/I)	391
Sodium (meq/l)	135	Lactate dehydrogenase (U/l)	1501
Potassium (meq/l)	4	Creatine phosphokinase (U/l)	15000
Calcium (mg/dl)	7		
Phosphorus (mg/dl)	7.7	WBC count (per microl)	24000
Magnesium (mg/dl)	3.6	Hemoglobin (g/dl)	20
Total bilirubin (mg/dl)	0.8	Platelet count (per mm^3)	267000
Direct bilirubin (mg/dl)	0.5		
		ABG: pH	6.98
Urine analysis: SG	1010	PCO_2 mmHg	18.7
WBC	3-4	HCO_3 mmol/l	9.7
RBC	10-15	BE mmol/l	-22
Blood	3+	PO_2 mmHg	52.5
Albumin	2+	SO_2	90%
Granular cast(/hpf)	1		

Table 1. Results of laboratory tests.

(Figures 1-4). Physical examination was otherwise normal. The patient had no other medical problems and took no medications.

In laboratory tests performed upon arrival, creatinine was 3 mg per deciliter, blood-urea-nitrogen 38 mg per deciliter, potassium 4 mEq per liter, magnesium 3.6 mg per deciliter, phosphorus 7.7 mg per deciliter, and creatine phosphokinase 15000 units per liter.

Analysis of blood gases showed hypoxia as well as metabolic acidosis along with respiratory alkalosis. Kidney ultrasound revealed no hydronephrosis. Right kidney size was 133×56 mm and left kidney size 114×47 mm. Parenchymal echo pattern was augmented and corticomedullary differentiation was increased. The results of other laboratory tests are shown in Table 1.

Two hours after arrival, when he was given sodium bicarbonate because of metabolic aci-

		Days					
		1	*2*	*8*	*13*	*16*	*22*
BUN	mg/dl	38	94	122	90	57	25
Cr	mg/dl	3	6	11.9	8	3.8	1.2
Na	meq/l	135	147	125	132		137
K	meq/l	4.5	5.6	5.1	5.2		4.5
Ca	mg/dl	7	7				8.7
P	mg/dl	7.7	7.7				
uric acid	mg/dl		9.3				
Ast	u/l	189		86			35
Alt	u/l	391		48			36
CPK	u/l	15000		9100			147
LDH	u/l	1501		870			253
Alb	g/dl	3.1					
Bilirubin.T	mg/dl			1.1			
Bilirubin.D	mg/dl			0.5			
pH		6.98	7.4				
PCO_2	mmHg	18.7	26				
PO_2	mmHg	525	86				
$\%SO_2$		90	97.5				
BE	mmol/l	-22	-36				
HCO_3	mmol/l	9.7	20				

Table 2. The patient's lab tests.

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dosis, he developed cardiopulmonary arrest, for which he was intubated and cardiopulmonary resuscitation was started. During CPR he repeatedly had ventricular fibrillation and ventricular tachycardia and was treated accordingly. The CPR lasted for 50 minutes and sinus rhythm was restored finally. Following extubation, the patient was admitted to the internal medicine floor after 24 hours.

On admission, the patient was oliguric. Urine volume was 50 to 250 milliliter per day for the subsequent week and the color was red brown.

After admission, levels of BUN and Cr raised and the patient was put on hemodialysis from August 15 to August 24 with a total of 7 hemodialysis sessions. Ten days later the patient's oliguria improved and levels of BUN and Cr started to reduce gradually.

Two weeks after admission, the patient developed right foot drop. EMG and NCV reported severe sciatic nerve lesion at the gluteal region on the right side with evidence of axonal degeneration.

For skin lesions, the patient received supportive treatment and wound cleansing twice daily. For foot drop, physiotherapy was prescribed.

The patient was discharged home on September 3 with prescription of vitamin E 400 units per day, vitamin B_1 300 mg per day and multivitamin tablet once daily. Creatinine was 1.2 and BUN 25.

Two months later on November 5, creatinine was 0.8 and BUN 21. Skin lesions had healed but the neurologic deficit had remained unchanged.

Discussion

Holtz [3] reported a case of paint thinner intoxication by oral intake, with loss of consciousness, upper gastrointestinal injuries, renal failure, rhabdomyolysis and cervical plexus injury, in 1992.

Chronic exposure to aromatic hydrocarbons can result in liver, kidney and bone marrow injury, myopathy, rhabdomyolysis, metabolic acidosis, and electrolyte abnormalities [4].

Fig. 1. Skin burns on the fingers.

Similar to ethanol and inhalational anesthetic agents, inhalants of abuse are highly lipid soluble. They enter the bloodstream through the lungs and rapidly diffuse throughout the body and into the nervous system [5]. Neurons, which have a high lipid content, are particularly susceptible to the solvent properties of inhalant compounds [6].

Metabolism and elimination proceed along various routes. Aliphatic hydrocarbons are likely to be exhaled unchanged, whereas aromatic hydrocarbons are metabolized via the hepatic microsomal system, and still other inhalants are excreted by the kidneys [5].

The desired effects of inhalant abuse include euphoria, lightheadedness, and a general state

Fig. 2. Skin burns on the fingers.

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Fig. 3. Skin burns on the toes.

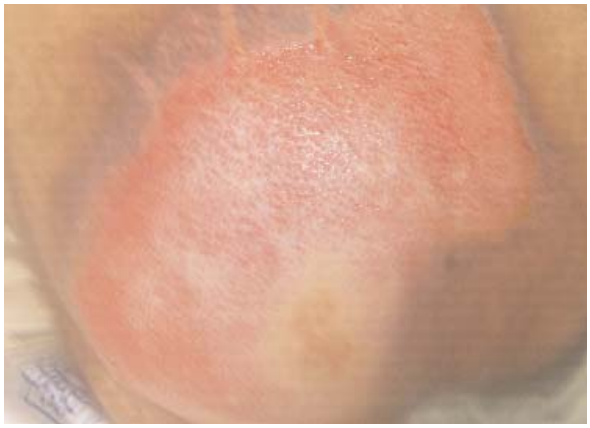

Fig. 4. Skin burns on the right buttock.

of intoxication similar to that produced by alcohol or marijuana. The effect usually lasts for only 15 to 30 minutes, but they can be sustained by continuous repeated use [7].

The most severe consequence from abuse of these substances is hypoxia or anoxia, which may cause death. Chemical pneumonitis with surfactant dysfunction, bronchospasm or noncardiogenic or hemorrhagic pulmonary edema may occur [9].

Dysrhythmias, myocarditis, or myocardial infarction may occur with acute or chronic use [9].

The CNS effects of inhalants include slurred speech, ataxia, disorientation, headache, hallucinations, agitation, coma and peripheral neuropathies [8].

Volatile substance use may cause metabolic acidosis, urinary calculi, and glomerulonephritis. Toluene, in particular, causes metabolic acidosis and sometimes rhabdomyolysis, with profound potassium and phosphate wasting [10].

Glue-sniffer's rash is an eczematoid dermatitis with erythema, inflammatory changes, and pruritus. Burns may occur when a flammable inhalant ignites [11].

Hydrocarbons are substances derived from petroleum distillation. They have a wide range of volatility. The high-volatility compounds are more likely to be abused as inhalants.

Aliphatic hydrocarbons are straight-chain compounds and include solvents and paint thinners. Abuse of these solvents may result in acid-base disturbances and derangements of calcium, potassium and phosphate [12]. Persistent peripheral neuropathy and myopathy with myoglobinuria and creatine kinase elevation have been reported [13].

Aromatic hydrocarbons and toluene are cyclic compounds containing a benzene ring and are used as industrial solvents. Benzene, toluene, and xylene are encountered most commonly. Aromatic hydrocarbons are highly volatile. When inhaled, they replace alveolar air and result in hypoxia [12]. Benzene causes renal and hepatic damage, as well as leukemia, aplastic anemia and multiple myeloma [14].

Toluene is a solvent used in paint thinners [15]. Toluene intoxication can cause cranial neuropathies, and myocardial depression. Chronic exposure to toluene can result in a variety of syndromes, including: generalized muscular weakness, with metabolic acidosis, hyperkalemia, hypophosphatemia, rhabdomyolysis, elevated creatine kinase [16] and neuropsychiatric symptoms, including peripheral and optic neuropathy [7].

Management of acute inhalant intoxication is supportive. Maintenance of cardiorespiratory function and removal of the patient from the source of the toxin are of primary importance. Supplemental oxygen is administered to enhance clearance from the respiratory tract and

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to treat hypoxia. Arrhythmias should be treated according to standard protocols; however, epinephrine and other catecholamines should be used with caution, because they can precipitate or worsen arrhythmias in the irritable myocardium [9]. Amiodarone has been successfully used to treat fluorocarbon - induced ventricular fibrillation [17].

The diagnosis of rhabdomyolysis is established by a marked elevation in serum creatine phosphokinase (typically greater than 10000 IU/L) and other muscle enzymes.

Another characteristic sign of rhabdomyolysis is myoglobinuria, which is suggested by persistent red to reddish-brown urine that tests positive for heme by dipstick after centrifugation while the plasma has a normal color and tests negative for heme [18].

It has also been suggested that the plasma creatinine concentration rises more rapidly with rhabdomyolysis (up to 2.5 mg/dl) than with other causes of acute renal failure.

Release of preformed creatinine from injured muscle and/or release of creatine that is then converted into creatinine in the extracellular fluid have been proposed to explain this finding [18].

ARF due to rhabdomyolysis in our case improved, but his sciatic nerve lesion did not improve.

Peripheral neuropathy caused by hydrocarbons present as distal weakness and muscle atrophy, mainly in the hands and feet.

Sensory symptoms are minimal and consist of numbness. After elimination of the exposure, recovery is slow, and may be incomplete [18], such as our patient that ARF due to rhabdomyosis improved, but sciatic nerve lesion did not recover.

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