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Case Report

A case of reversible cardiomyopathy associated with acute toluene exposure

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ABSTRACT

Inhalation of toluene-based products is popular among young adults. It has been shown to have a variety of adverse effects on several organs and systems. Although the heart seems to be a sensitive target organ to toluene, cardiotoxicity has often been ignored, especially in cases of acute toluene abuse, with relatively low concentrations. Thereby, routine cardiac examination and echocardiography for cardiotoxicity should be performed in cases of acute toluene exposure, even though there is no cardiovascular sign or symptoms.

1. Introduction

In recent years, as a result of development of petro chemistry industry, abuse of organic volatile chemicals especially sniffing of adhesives has become a public health problem in adults in the worldwide. Toluene is an aromatic hydrocarbon that is generally used in industrial products. Because of its joyful effect, inhalation of toluene-based products is popular among young adults. However, it has been shown to have a variety of adverse effects on several organs and systems. Since toluene is lipophilic aromatic hydrocarbon, it is easily absorbed by lipid-rich tissues. Acute effects of toluene is mainly neurological (euphoria followed by depression, altered mental state, cerebellar abnormalities, and peripheral neuropathy),[1] but as well as central nervous system involvement, it also affects gastrointestinal, respiratory, urinary and hematopoietic systems.[2,3] Although the heart seems to be a sensitive target organ to toluene,[4] its cardiotoxicity has often been ignored, especially in cases of acute toluene abuse.[4] Data on acute toluene-induced cardiomyopathy is sparse. We herein report a case of a toxic cardiomyopathy after acute exposure to a toluene-based product.

2. Case report

A 21-year-old man without a history of any cardiovascular disease, presented to our emergency department with a complaint of dyspnea, confusion and constitutional symptoms. In his history, he had acute exposure to intensive toluene sniffing. However, previous history of toluene abuse, frequency and duration of toluene exposure were not clear. On arrival, the patient was appeared intoxicated, unconscious and partially responsive to verbal and painful stimuli. Vital signs included blood pressure of 76/43 mmHg, pulse rate of 138 bpm, respiratory rate of 20 breaths/min, and a body temperature of 37.1 °C. His neurological examination was unremarkable A 12-lead ECG showed sinus tachycardia with a rate of 154 bpm and ST segment depression in all leads. QTc interval and QRS duration was 495 msec and 128 msec, respectively (Fig. 1). Transthoracic echocardiography revealed left ventricular global hypokinesis with an ejection fraction of 30%. The arterial blood gas analysis revealed the following results: pH 7.210, $PaCO_2$ 27.6 mmHg, PaO_2 80.1 mmHg, bicarbonate 12.5 mmol/L, K + 3.1 mmol/L, cCl- 110 mmol/L, cLac 13.3 mmol/L and an anion gap of 18. The diagnosis of increased anion gap hyperchloremic metabolic acidosis was made and potassium chloride and intravenous fluid replacement therapy was administered to the patient.

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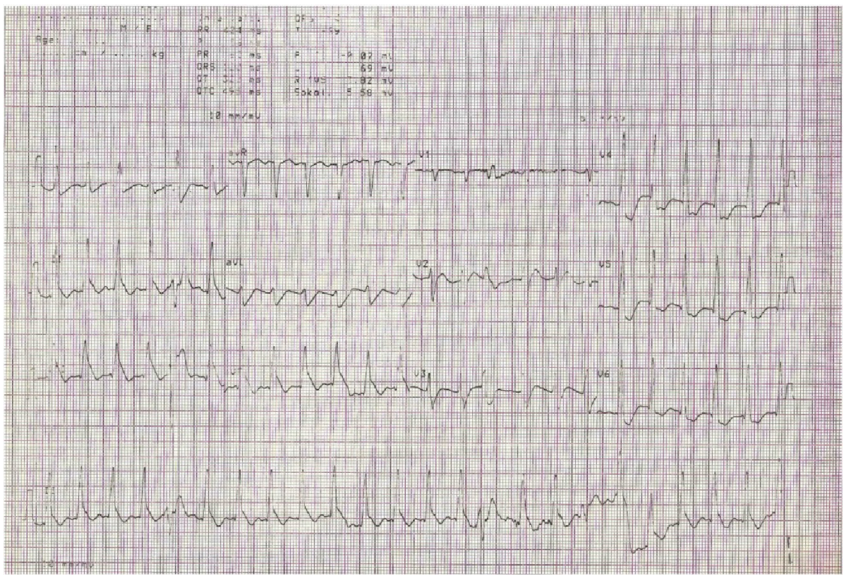

Fig. 1. ECG on admission shows sinus tachycardia and ST segment depression.

Laboratory examination revealed that: complete blood count is normal, peak aspartate transaminase 245 U/L, peak alanine transaminase 143 U/L, peak troponine I 9.3 ng/ml, peak creatine kinase-myocardial band 263 U/L, peak creatine kinase 10,200 U/L, peak lactate dehydrogenase 795 U/L. Coronary angiography of the patient showed neither structural nor functional lesions in the coronary arteries. In the light of these findings, cardiomyopathy was diagnosed. After withdrawal of toluene exposure and replacement of potassium, treatment consisting of metoprolol and ramipril was administered. He had a dramatic clinical recovery throughout the hospital course. Control ECG was completely normal without ST segment changes. QTc interval and QRS duration was 438 msec and 76 msec, respectively (Fig. 2). In his second echocardiography, left ventricular ejection fraction was measured as 60%. Eight days after his hospitalization, the patient was discharged from the hospital. Although he did not obey the suggested treatments, no relapse was seen during the follow-up period.

3. Discussion

Toluene is the most frequently inhaled organic volatile chemical. It is used in various ubiquitous products (glues, adhesives, paints, paint thinners, organic solvents and cleaning products) and its low price makes toluene easily reachable and difficult to control. After inhalation, toluene rapidly diffuses into the blood and easily penetrates lipid-rich tissues, and monooxidase enzymes that are connected to cytochrome P450 oxidize toluene in tissues, lipid peroxidation products and free oxygen radicals are released.[5] Toluene abuse also causes electrolyte and acid base disturbances. Hyperchloremic metabolic acidosis was observed in most of cases of toluene intoxication and is indicative of distal tubular acidosis.[6] Hypokalemia in toluene intoxication is caused primarily by increased renal potassium loss secondary to increased excretion of hippurate, a major metabolite of toluene.[6]

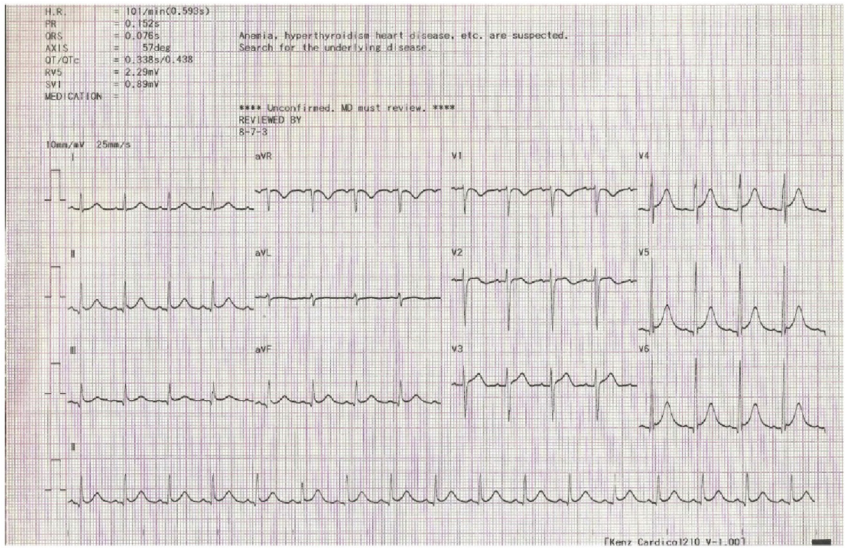

Fig. 2. Control ECG shows completely normal recording without ST segment changes.

Experimental studies revealed that toluene inhalation causes morphological changes in the lung, liver, kidney, adrenal gland, central nervous system and heart of mice in the short and long term. Cardiac involvement of toluene intoxication should be of primary clinical importance. Exposure to toluene can lead to cardiac arrhythmias and sudden sniffing death syndrome. Tachyarrhythmia is the classical manifestation of toluene cardiotoxicity. Recurrent ventricular fibrillation resistant to defibrillation and antiarrhythmic treatment associated with toluene inhalation was reported.[7] Previous reports indicated toluene might cause bradyarrhythmia, heart block and AV dissociation.[8] This patient presented with shock and tachycardia. It is hard to differentiate toluene-induced cardiomyopathy from shock-related multiple organ dysfunction syndromes and myocardial stunning. Nonetheless, hypokalemia and inhibition of cardiac sodium currents by toluene exposure might be responsible for its arrhythmogenic effect.[8] However, some authors proposed that arrhythmia is associated with toluene's direct arrhythmogenic effects on the cardiac tissue, rather than hypokalemia.[9] It has been suggested that myocardial sensitization to endogenous catecholamines or ischemia related vasospasm might also play an important role in the development of cardiomyopathy and fatal arrhythmias.[10]

Development of cardiomyopathy is associated with dose and exposure time of toluene, environmental and genetic factors. Cardiomyopathy associated with toluene is commonly occurred after chronic exposure of toluene.[11] Vural and Ogel[12] reported a case of toxic dilated cardiomyopathy in a 21-year-old patient with chronic toluene abuse, and demonstrated that the function of the myocardium and the patient's functional capacity had been largely improved following absence of toluene abuse and medical treatment. Correspondingly, they argued that the toxic effect of toluene on the heart muscle might be reversible. In another case, a patient working in the furniture sector was diagnosed as dilated cardiomyopathy and ventricular tachycardia, and his symptoms and left ventricle ejection fraction were improved, after leaving work.[13] In our case, toluene exposure is acute and relatively at low concentrations. We proposed that acutely toluene exposure might also cause left ventricular dysfunction. Although metabolites of toluene are substantially eliminated by kidney in 12 h, it might take 72 h to completely eliminate some toluene stored in fat tissues.[1] In addition, electrolyte and acid base disturbances improve rapidly with repletion of potassium and extracellular volume. Correspondingly, in our case, we suggested that toluene inhalation was responsible for the etiopathogenesis of toxic cardiomyopathy and the adverse cardiovascular effects of toluene had been disappeared after 72 h of discontinuing of toluene, and repletion of potassium and extracellular volume.

In conclusion, it should be mentioned that toluene might cause cardiac toxicity in some cases throughout its direct or indirect cardiotoxic effects, even in acute exposure with relatively low concentrations. Fortunately, this situation is often reversible with toluene discontinuation. Considering this fact, routine cardiac examination and echocardiography for cardiomyopathy should be performed in cases of acute toluene exposure, even though there is no cardiovascular sign or symptom.

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