



Acute Myocardial Infarction with Normal Coronary Angiography after Butane Inhalation

Bütan İnhalasyonu Sonrası Gelişen Normal Koroner Anjiyografili Akut Myokard İnfarktüsü

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ABSTRACT

Introduction: Butane gas, which is known lighter gas, is used especially by teenagers and young people with the aim of getting "high." When these gases are inhaled or sniffed for recreational purposes, they can lead to acute cardiac events such as acute myocardial infarction (MI), ventricular fibrillation, or sudden death.

Case Report: A 19-year-old male patient with cardiopulmonary arrest after sniffing lighter gas was brought to our emergency department by the 112 emergency ambulance services. He was diagnosed with high-lateral MI based on ST elevation in the electrocardiogram after successful resuscitation. However, his epicardial coronary arteries were normal based on coronary angiography. Our patient is one of rare cases who was diagnosed MI after butane inhalation and could perform emergency cardiac catheterization. Our patient underwent emergency cardiac catheterization.

Conclusion: Butane gas inhalation can cause ST-elevation MI. MI due to butane gas inhalation is caused not by mechanical obstruction but by coronary vasospasm.

Keywords: Butane gas, vasospasm, myocardial infarction, angiography

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ÖZET

Giriş: Çakmak gazı olarak bilinen bütan gazı genellikle gençler arasında keyif yapıcı bir madde olarak kullanılmaktadır. Bu gazlar eğlence ve keyif alma amaçlı soluma veya koklama yoluyla kullanıldığında akut myokard infarktüsü (MI), ventriküler fibrilasyon veya ani ölüm gibi ciddi kardiyak yan etkilere neden olabilmektedir.

Olgu Sunumu: 19 yaşında erkek hasta 112 tarafından çakmak gazı koklama sonrasında kardiyak arrest olarak acil servise getirildi. Başarılı bir resüsitasyon sonrası çekilen elektrokardiyografisinde yüksek lateral MI tanısı alan hastanın yapılan koroner anjiyografisinde normal koroner damarlar saptandı. Bizim vakamız bütan gazı inhalasyonu sonrası MI tanısı alan ve koroner anjiyografisi yapılabilen nadir vakalardan birisidir.

Sonuç: Bütan gazı inhalasyonu ST segment elevasyonlu MI nedeni olabilmektedir. Bu bütan gazı ilişkili MI nedeni ise mekanik obstrüksiyondan daha çok koroner vazospazma bağlıdır.

Anahtar Kelimeler: Bütan gazı, vazospazm, myokard infarktüsü, anjiyografi

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Introduction

Butane is a colorless, volatile, and flammable gas with a boiling point of -0.5°C . It is used in the form of liquid petroleum gas and lighter fuel. The lighter fuel, which can be inhaled using nylon bags held over the mouth, is being more gradually used as a pleasure-inducing substance by teenagers and young people (1, 2).

Following its inhalation, butane is absorbed by the lungs and mixes with the blood circulation, which rapidly leads to central nervous system depression symptoms, such as a state of extreme cheerfulness, drunkenness, dizziness, headache, changes

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Figure 1. ECG of patient after returned spontaneous circulation

in consciousness, visual and auditory hallucinations, drowsiness, disrupted orientation, palpitation, blurred sight, agitation, seizures, delirium, speech disorder, and muscle coordination loss. This can cause myocardial infarction (MI), ventricular fibrillation, and cardiorespiratory arrest (2-5).

In our report, we present a respiratory and cardiac arrest condition in a 19-year-old patient who was brought to our emergency department. The patient was diagnosed with high-lateral MI based on the electrocardiogram (ECG) after successful resuscitation. However, the patient had normal coronary results in the coronary angiogram. In this case report, we aim to draw attention to the cause of the cardiac arrest that was butane (lighter fuel) inhalation.

Case Report

A 19-year-old male patient with cardiopulmonary arrest after sniffing lighter gas was brought to our emergency department by the 112 emergency ambulance services. We immediately admitted him to the critical care room, performed cardiopulmonary resuscitation (CPR), and intubation. Fifteen minutes after CPR, the spontaneous circulation of the patient had returned. After the return of spontaneous circulation, the patient's vital signs were as follows: blood pressure 70/40 mmHg, heart rate 110 beats/per min, respiratory rate: he was intubated, temperature 36.6°C, and oxygen saturation 97%. The Glasgow coma score was 3. There were ST-segment elevations in leads D1-aVL and V4-V6 T on his ECG (Figure 1). In his complete blood count test, the white blood cell count was 11.900/mm³, and in the biochemical tests, the following results were obtained: glucose: 219 mg/dL, creatinine: 1.52 mg/dL, ALT: 135 U/L, AST: 140 U/L, LDH: 466 U/L, CPK: 1239 U/L, troponin: 0.007 ng/ml, and CK-MB: 13.8 U/L. We thought that the reason for MI was a coronary vasospasm. To ease the vasospasm, we could have infused nitroglycerin, but we did not do that because of refractory hypotension. To perform emergency cardiac catheterization and intensive care follow-up, the patient was transferred to another tertiary hospital. The coronary angiogram revealed that the patient had normal epicardial coronary arteries and akinetic anterior and



Figure 2. Emergency coronary angiography views of patient after returned spontaneous circulation

lateral walls (Figure 2). On the second day, the patient had another cardiopulmonary arrest, and this resulted in his death.

Discussion

Because of the convenience and easy access to butane found in cigarette lighters, room sprays, and deodorants, butane gas is used as a pleasure-inducing substance by 15–20 year-olds through the inhalation of lighter fuel in cigarette lighters (1, 2). In literature, there are reported acute cardiac events such as cases of MI, ventricular fibrillation, and asystole because of butane inhalation (5, 6). The suggested mechanisms of MI due to butane inhalation are coronary artery spasms and hypoxia (6). The occurrence of ventricular fibrillation is explained by the increased sensitivity of the myocardium to catecholamines, decreased arrhythmia threshold, and direct toxic effects on the myocardium (7). El-Menyar et al. (6) reported that they performed coronary angiography on a patient with MI after butane intoxication and that they found normal coronary arteries. According to the authors, the reason for MI was a coronary vasospasm because ST-segment elevations were resolved after preangiographic treatment with nitroglycerin. To the best of our knowledge, although cases of MI after inhaling butane have been reported in literature, there are very few cases who underwent emergency cardiac catheterization and demonstrated normal coronary arteries (5, 6). Our patient is one of the cases who underwent emergency cardiac catheterization. Similar to the other cases, we thought that the cause of MI was coronary vasospasm because our patient had a normal coronary angiogram, and his ST-segment elevations were still present during angiography.

Conclusion

In conclusion, physicians should be aware that butane gas inhalation can cause ST-elevation myocardial infarction. These two cases in the literature and our case demonstrated that the cause of myocardial infarction in this kind of cases is not mechanic obstruction but is coronary vasospasm.

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