Subacute myocardial infarction due to long-term paint thinner and ecstasy abuse

To the Editor,

Substance abuse can cause death, as well as negative effects, on social life and is becoming more common among young people.

Ecstasy (3,4-methylenedioxymethamphetamine, MDMA) and paint thinner usage may lead myocardial infarction (MI). Hereby, we discussed a subacute anterior MI patient (28-year-old male) without a previous medical history, abusing paint thinner and ecstasy.

Our patient, flooring parquetry, used to inhale approximately 250 mL paint thinner with a towel a day for 15 years, take 2 pills of ecstasy a day for 5 years, and smoke 20 cigarettes a day for 15 years. On admission, he had crushing chest pain, and the physical examination was normal. Electrocardiography (ECG) revealed mild sinus tachycardia with poor R wave progression, ≥ 1 mm ST-segment elevation in leads V1-V4, and T wave inversion in leads V1-V6, DI-aVL. Following a loading dose of ticagrelor (180 mg) and acetylsalicylic acid (300 mg), he was taken to the catheterization laboratory. Coronary angiography revealed 95% stenosis at the proximal segment of the left anterior descending artery (LAD) and 80% stenosis at the mid-segment of the LAD. The right coronary artery and left circumflex artery were normal. After percutaneous transluminal coronary angioplasty, a drug-eluting stent was implanted into LAD lesions successfully. Tirofiban infusion was administered intravenously for 24 hours. Echocardiography showed a mild anterior wall motion abnormality, and the left ventricular ejection fraction was 50%. The consulting psychiatrist diagnosed him with substance abuse and antisocial personality disorder. His in-hospital course was uneventful, and he was discharged after 5 days without any complaint.

Ecstasy (MDMA) is a psychostimulant amphetamine derivative and increases the release of serotonin, dopamine, and noradrenaline by blocking reuptake transporters in neurons. Ecstasy makes people energetic, euphoric, sociable, and extroverted and can cause cardiac toxicity, including rhythm disturbances, MI, and sudden death. Coronary vasospasm, catecholamine-mediated platelet aggregation, increase in shear stress with subsequent rupture of asymptomatic atherosclerotic plaques, and increased myocardial oxygen demand can lead to amphetamine-induced myocardial ischemia (1). Sadeghian et al. (2) described a 24-year-old male using ecstasy who had two MIs in a 3-month period. While the coronary angiography was normal in the first episode, in the second one, a thrombus totally occluding the proximal segment of the LAD was shown. It was revealed that amphetamines increase endothelial tissue factor expression and activity and also inhibit tissue factor pathway inhibitor expression (3). These can lead to thrombus formation, causing acute coronary syndromes.

Paint thinner is a solvent and contains hydrocarbons, like toluene, xylene, N-hexane, and benzene. It is easy to access, especially in Turkey. The paint thinner that our patient used contained 74%-78% methylbenzene (toluene, phenylmethane). Toluene is oxidized by mono-oxidase enzymes in target tissues; then, extensive free radicals are released, causing tissue damage (4). Acute coronary syndrome due to paint thinner inhalation is not well known. Possible mechanisms include coronary vasospasm, arrhythmias, and thrombus formation (5).

As a conclusion, in patients with chest pain abusing ecstasy or paint thinner, acute MI should be considered. People should be informed that ecstasy and paint thinner abuse can be fatal.

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Available Online Date: 21.01.2015

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DOI:10.5152/akd.2015.5975