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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An unknown side effect of isotretinoin: Pericardial effusion with atrial tachycardia

To the Editor,

Isotretinoin is a synthetic vitamin A derivative used in the treatment of acne vulgaris and other dermatologic disorders. Systemic isotretinoin therapy may cause some cardiac side effects, like atrial tachycardia (1), congenital heart disease, and cardiac remodeling (2), reported as case reports. A 26-year-old female presented to the emergency unit of with syncope after a long palpitation episode. Her physical examination was normal except for tachycardia. A 12-lead electrocardiogram revealed atrial tachycardia, and the heart rate was 149 beats/min. After a 25-mg intravenous injection of diltiazem hydrochloride bolus, the atrial tachycardia terminated and normal sinus rhythm was sustained. Her laboratory tests and chest X-ray were normal. Echocardiography revealed normal left ventricular function and pericardial effusion of 0.8 cm at posterior side, 0.9 cm at the right atrial side and 1.3 cm at the right ventricle side. Several atrial tachycardia episodes were detected on rhythm Holter. During the longest episode of atrial tachycardia, the heart rate was 149 beats/min. The patient had been on oral isotretinoin therapy of 0.5 mg/kg/day for the previous 4 months because of nodular acne and was not using any other medication. After consulting with a dermatology physician, isotretinoin was stopped. Holter analysis revealed whole-day sinus rhythm 2 months after the drug therapy was interrupted. Echocardiography revealed gradual regression of pericardial effusion at the follow-up.

Isotretinoin is the most effective treatment options for severe nodular acne, and it belongs to the first generation of synthetic 13-cis reti-

noic acid compounds. Although cheilitis, dry skin, and transient worsening of acne are the most common side effects, adverse effects associated with the nervous, musculoskeletal, ocular, gastrointestinal, hematological, psychiatric, and cardiac systems were also reported.

Cardiac side effects, such as congenital heart disease (3) and arrhythmias, which are frequently atrial arrhythmia, and a case right bundle branch block were reported due to isotretinoin use. Limited reports about adverse cardiac effects are controversial or insufficient to understand the underlying pathology. The therapeutic mechanism of retinoids or their side effects are not well defined. The most common adverse musculoskeletal system events associated with isotretinoin use are calcifications of the tendon and ligaments and myalgias. Isotretinoin was supposed to stimulate degeneration in the synovial cells by influencing the lysosomal membranes of the cells. Therefore, the cells were damaged by becoming vulnerable.

Large doses of retinol result in tissue damage due to the release of certain acid hydrolases and lysosomal enzymes into circulation (4). On the other hand, it was shown that with excess retinol, resting heart rate is increased and action potentials are affected (increased systole duration and decreased diastole duration) because of altered myocardial cell permeability (5). The cell membrane fragility for both myocardial and pericardial cells may be convincing to explain the mechanism of atrial tachycardia and pericardial effusion. Structural changes in the cell membrane affect action potentials, and this may cause atrial tachycardia. Alterations in pericardial cell permeability might also have led to extravasation of fluid to the pericardial space.

It might be useful to question isotretinoin use in cases of pericardial effusion or atrial tachycardia, especially in young patients with dermatological problems, in our daily practice.

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