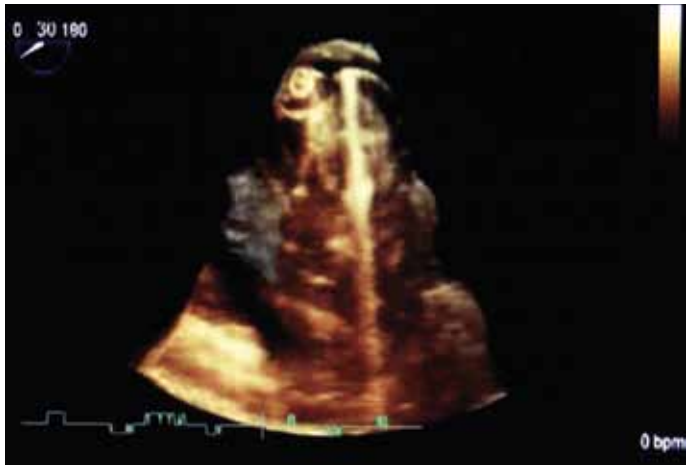

Figure 4. Three-dimensional transesophageal echocardiogram shows closure of paravalvular defect with use of the Amplatzer duct occluder

been increased relatively rapidly over for last two decades (5, 6). Variety of devices actually developed for other indications have been used for PVL percutaneous closure. Therefore, mismatch between defect and occluder shapes may cause persistence of residual leaks. This limitation may be overcome by three-dimensional evaluation of the defect by 3D TEE as in our case. Meticulous care should be given to proper delineation of the defect size and shape in every case; therefore, most appropriate device can be selected with resulting best-fitting device deployment. In this respect, 3D TEE provides invaluable information about both defect properties and successful device deployment

Conclusion

With the increasing numbers of aortic valve replacements, need for PVL intervention will increase. Percutaneous PVL closure should be considered in patients who are deemed to be poor surgical candidates. Real-time 3D TEE is an important modality for diagnosing paravalvular leaks and with the use of this technique procedure results will improve.

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Acute myocardial infarction associated with Captagon use

Kaptagon kullanımı ile ilişkili akut miyokart enfarktüsü

Introduction

Although acute myocardial infarction (AMI) is usually a disease of older ages, it may be seen in younger ages. In young cases of AMI non-atherosclerotic coronary artery disease (CAD), thrombophilia, illicit drug abuse, premature atherosclerosis must be considered. Illicit drug abuse such as amphetamines and cocaine has been widely recognized as a causative agent in AMI. Abuse of fenethylline as the brand Captagon may also cause AMI. Today, most of the counterfeit Captagon tablets do not contain fenethylline, but combination of substances that mimic the effects of the original molecule.

This report describes a young male who presented with an acute anterior MI after taking a tablet of Captagon and discuss cardiovascular effects of amphetamines and substances that mimic the effects of the amphetamines.

Case Report

A 21-year-old male was admitted to the emergency department of local State Hospital with chest pain. He collapsed during initial examination, and cardiac monitorization showed ventricular fibrillation, and immediate, successful defibrillation was carried out. An electrocardiogram (ECG) showed (Fig. 1) widespread ST segment elevation in anterolateral leads with reciprocal changes in inferior leads confirming AMI. He was given 300 mg acetyl salicylic acid, oxygen, unfractionated heparin bolus (5000 units) and an infusion of heparin was initiated. Then, he was transferred to our center for an emergency Percutaneous Coronary Intervention (PCI). On admission, he was confused with having strange behaviors and acting aggressively. Information about the patient's past medical history was taken from his relatives and revealed no any cardiovascular risk factors except smoking. On physical examination, his blood pressure was 115/70 mmHg, heart rate was 90 beats per minute. Cardiac and respiratory examinations were unremarkable. There were some old self-mutilation scars on his arms and chest wall. A repeated ECG showed normalization of the ST segment changes in anterolateral leads. To evaluate changes in his thinking and behavior, a neurological consultation and cranial computed tomography (CT) scan was performed. Neurological consultation revealed no objective evidence of any disease, and cranial CT was normal. A primary PCI was deferred, so he was agitated and had normalization of ST changes on ECG (Fig. 2).

The patient's agitation was controlled with haloperidol. Initial laboratory tests revealed elevated creatinine kinase (CK) of 344 U/L (reference range 24-195 U/L), troponin I (TnI) of 0.616 ng/mL (cut-off value in our laboratory is 0.4 ng/mL).

He was then transferred to the coronary intensive care unit and maintained on the following medications: Haloperidol (3 mg/day), acetyl salicylic acid (300 mg/day), clopidogrel (75 mg/day), metoprolol succinate 25 (mg/day), atorvastatin calcium (10 mg/day), intravenous nitroglycerin infusion (5 mcg/minute, titrated doses on his blood pressure response), unfractionated heparin infusion (under activated partial thromboplastin time control). The following day, his mental status was considered as normal, and haloperidol therapy was stopped. Investigations including lipid profile, homocysteine, thyroid and kidney function tests, and blood sugar levels revealed normal results. Search for connective tissue disorders and vasculitic syndromes were unremarkable. Abnormal laboratory results showed leukocytosis, elevated levels of serum aspartate aminotransferase and cardiac enzymes. His cardiac enzyme markers peak levels were CK of 1513 U/L, TnI of 18.7 ng/mL.

Because of recurrent chest discomforts on the third day of his admission, a coronary angiography was planned and performed. It showed a significant narrowing in the proximal portion of the left anterior descending (LAD) artery (Fig. 3, Video 1. See corresponding video/movie at www.anakarder.com). The circumflex and the right coronary arteries were normal. Significant narrowing in the proximal portion of the LAD persisted despite of the repeated doses of intracoronary nitroglycerin (total of 200 micrograms). A bare metal direct stenting was performed on this lesion, with a residual 10% narrowing (Fig. 4, Video 2. See corresponding video/movie at www.anakarder.com). A high pressure, nominally sized balloon post dilatation was performed for optimizing stent expansion and resulted in rupture of the balloon. Neither vessel perforation nor dissection was occurred on or near the stented segment. Final angiography showed a satisfactory result (Fig. 5, Video 3. See corresponding video/movie at www.anakarder.com). The following day, to clarify the past medical history of the patient, a further attempt was performed. He confessed us that he had taken a pill of Captagon before the chest discomfort. Screening of urine for illicit drugs metabolites was not performed, because he admitted that he had taken Captagon just before discharging him from the hospital.

A transthoracic echocardiographic examination was performed before the patient was discharged from the hospital. The left ventricular ejection fraction was estimated to be about 50%, and there was only mild hypokinesia of the anterior segments. He was discharged on the fifth day on 300 mg of oral acetyl salicylic acid, 75 mg of clopidogrel, 2.5 mg of perindopril daily with instruction of avoiding usage of any illicit drug.

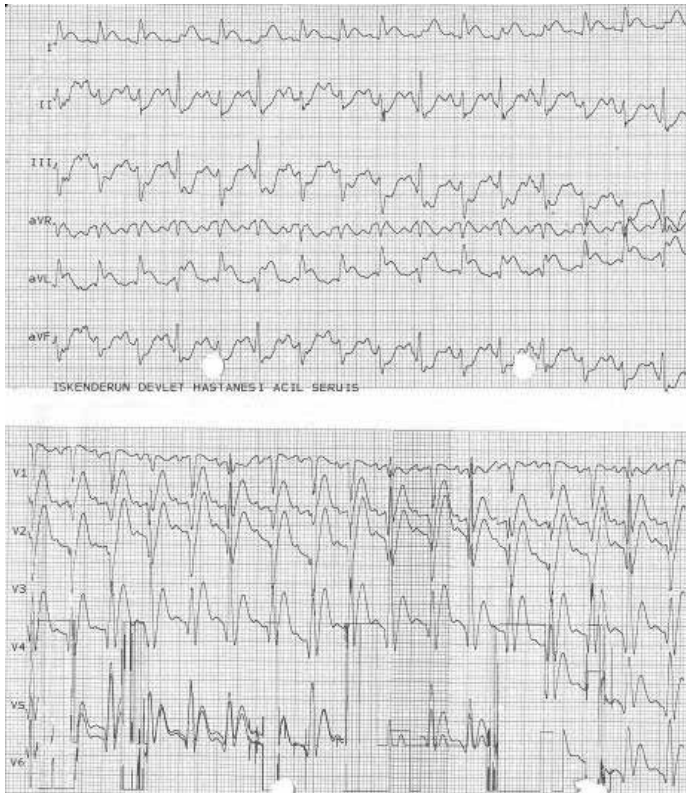

Figure 1. A 12-lead electrocardiogram obtained at presentation shows widespread ST segment elevation in anterolateral leads

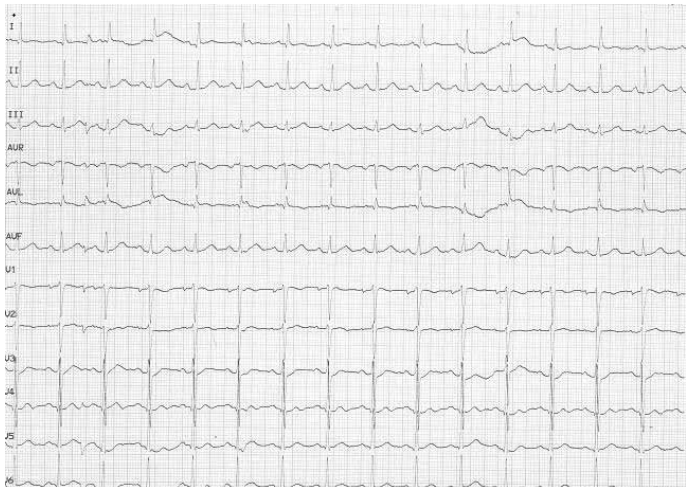

Figure 2. A repeated electrocardiogram obtained at our center shows normalized ST segments in anterolateral leads

Discussion

Drug abuse is a global problem seems to increase in all over the world. Abuse of fenethylline as the brand Captagon is most common in

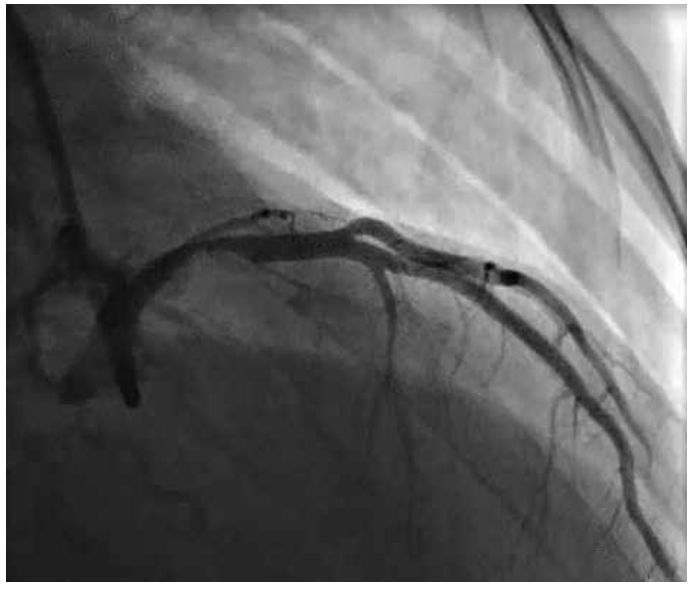

Figure 3. Coronary angiogram in the right cranial view shows significant narrowing of proximal left anterior descending artery

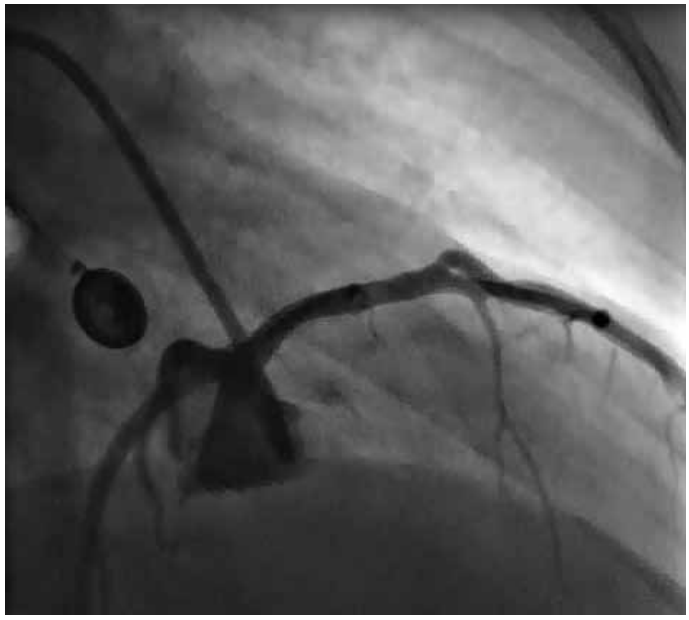

Figure 4. Post-stenting coronary angiogram in the right cranial view shows residual stenosis in the stent site

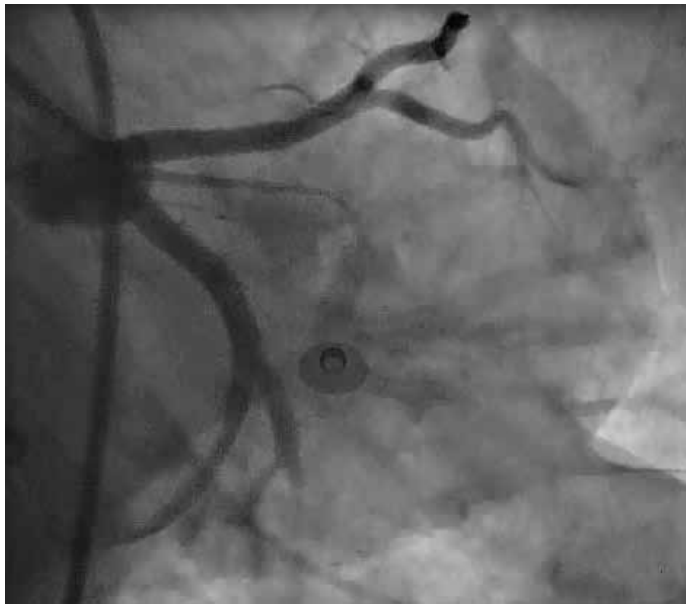

Figure 5. Coronary angiogram after postdilatation in anteroposterior caudal view shows satisfactory result

the Near and Middle East region. As a central stimulant, fenethylline was used in patients with Attention Deficit Hyperactivity Disorder (ADHD), narcolepsy and depression. Fenethylline is an N-alkylated amphetamine derivative and metabolized to amphetamine and theophylline both of which are active stimulants (1, 2). To our knowledge, legal Captagon tablets are not available today. Fake products may contain fenethylline or combinations of substances that mimics the effects of the fenethylline. Some fake products seem to have the original physical appearance of the original product. Today, most of the Captagon mimic and counterfeit tablets contain amphetamine, caffeine, ephedrine, quinine, theophylline acetaminophen, diphenhydramine and lactose or a combination of these substances. In the literature, several cases of AMI associated with amphetamine and ephedrine abuse have been reported previously (3, 4). A population-based epidemiologic study of hospital patients to examine the risk of AMI with amphetamine abuse has been indicated a modest, but significant, association between amphetamine abuse and AMI (5).

Ephedrine is a direct agonist and an indirect adrenoreceptor agonist, causing release of noradrenaline from presynaptic sympathetic nerve terminals. Its cardiovascular effects include positive chronotropic and inotropic effects, arterial vasoconstriction and hypertension (3). The amphetamines cause the release of catecholamines from presynaptic sympathetic nerve terminals stimulating peripheral alpha and beta adrenergic receptors. The most common effects of usage of amphetamines are hypertension and tachycardia (6). The true pathophysiology of myocardial infarction following amphetamine use seems to remain unclear. Some pathophysiological mechanisms has been suggested to clarify this state. Catecholamines can cause myocardial damage by increasing myocardial oxygen demand and inducing platelet aggregation and coronary arterial vasospasm. The increased catecholamine levels could trigger the rupture of an atherosclerotic plaque. The rupture of an atherosclerotic plaque, coronary arterial vasospasm and platelet aggregation can cause an AMI. However, coronary artery spasm followed by thrombus formation is considered the most likely pathophysiologic mechanism. There may be other mechanisms at the endothelial level. Similar to cocaine, amphetamines and amphetamine like substances may accelerate atherosclerosis by causing structural defects in the endothelial cell barrier (7).

The management of an AMI associated amphetamines or amphetamine-like substances abuse is not clear at present. In patients presenting with ST elevation AMI, first of all, the infarct-related artery (IRA) patency should be taken into consideration. If there is no contraindication, vasodilators seem logical in addition to antiaggregants and anticoagulants for the treatment of illicit substance-induced AMI. Guidelines recommend not to use beta-adrenergic receptor blockers for patients with an AMI caused by cocaine, because of risk of exacerbating coronary spasm (8). This state could be true for patients with an AMI caused by amphetamines or amphetamine-like substances. Since, these substances may trigger vasospasm; beta-adrenergic blocking agents should be avoided in such cases. Calcium channel blockers and nitrates are effective in the management of vasospastic angina, these agents may be also effective in the treatment of AMI secondary to amphetamines or amphetamine-like substances.

As a complementary to angiography, intravascular ultrasound (IVUS) could provide information of tissue components of coronary artery and detailed quantification of coronary atherosclerosis burden. We did not perform an IVUS, so it was not available. But, residual stenosis on coronary angiography after implantation of a coronary stent to the lesion site, no morphologic change of the coronary lesion despite of repeating doses of intracoronary nitroglycerin, rupture of the post-dilatation balloon were indirect findings of a atherosclerotic lesion. In our case, treatment with a beta blocking agent might have been responsible for persistent coronary spasm. But, there was a residual stenosis on implanted stent site which was regressed after post dilatation. So, in that case, we hypothesize that premature atherosclerosis and Captagon consumption were the main triggering factors for an AMI. In addition to smoking, consumption of counterfeit Captagon pills might be responsible for premature atherosclerosis in this young man.

Conclusion

In the literature no cases of AMI associated with Captagon abuse have been reported yet. Toxicologic screening of blood and urine for illicit drug abuse may be helpful in suspected cases, especially in young

ones. In suspected cases of illicit drug abuse, beta-adrenergic blocking agents should be avoided. Patency of infarct-related artery can be achieved by PCI in patients with AMI associated with amphetamines or amphetamine-like substances.

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Video 1, 2, 3. Pre-stenting, post-stenting and final coronary angiographic video/movie images in different views.

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A case of iatrogenic hypothyroidism presented with cardio-inhibitory syncope and resolved by thyroxine supplementation

Tiroksin tedavisi ile düzelen kardiyoinhibitör senkop ile gelen iatrojenik hipotiroidili bir vaka

Introduction

Thyroid hormones have inotropic and chronotropic effects on heart functions (1). In hypothyroid situation, life-threatening dysrhythmias may occur (2).

We report a 15-year-old girl who presented with syncope and sinus bradycardia, low voltage, prolonged QT interval and first degree atrioventricular block on electrocardiogram due to iatrogenic hypothyroidism.

Case Report

A 15-year-old girl presented with syncope. She lost consciousness for approximately five minutes when she has been sitting, before her admission. No feces or urine incontinence or tonic contractions had been observed.

On physical examination she was conscious, her body weight was 42.5 kg (5-10 percentiles), height 158 cm (25-50 percentiles). The pulse rate was 52 beats/min, the blood pressure 85/40 mmHg. The auscultation of heart was normal except for bradycardia; neurological examination was normal. There was a transverse incision scar on the anteromedial neck; thyroid gland was nonpalpable. The blood testing showed macrocytic anemia (Hb: 9.6 gr/dl, Htc: 26.2%, MCV: 100.8 fl). Blood glucose, electrolytes telecardiogram and electroencephalogram were normal. Electrocardiography showed sinus bradycardia, low voltage, first degree atrioventricular block (PR interval was 0.20 s) (Fig. 1A). Echocardiographic examination was normal. Holter monitoring showed sinus bradycardia (minimum heart rate was 45 beats/min in sleepiness, 47 beats/min in wakefulness), first degree atrioventricular block, and prolonged QT interval (0.45 s). Head-up tilt testing was normal. Her exercise capacity was low for modified Bruce protocol. Her pulse rate maximally increased up to 109 beats/min with exercise.

Serum vitamin B12 and folic acid levels were normal. Thyroid functions were found as: free triiodthyronine 3 (T3) <0.26 pg/ml, free thyroxine (T4): 0.06 ng/dl, TSH: 604.6 µIU/ml. Ultrasonographic examination and thyroid scanning did not show any thyroid tissue. When her past history was detailed, it was learned that she had easy fatigability, dry skin, constipations, reduced exercise capacity and academic performance since one year. One year ago she had been operated for an anteromedial neck mass in another hospital. Since histopathological examination of the mass reported thyroglossal duct cyst, her operator had not prescribed any drug therapy and had said that follow-up was not required. However, the pathologic specimens of the patient were investigated again by Pathology Department of our hospital and ectopic thyroid tissue and thyroid adenoma was seen. Levothyroxine therapy was started with a dose of 25 µg/day and was raised weekly up to 150 µg/day. At the end of first month of treatment skin dryness, constipations, anemia and effort intolerance improved, academic performance increased. The electrocardiographic findings returned to normal (Fig. 1B).

Discussion

Thyroid hormones have inotropic and chronotropic effects on heart function and enhance overall total protein synthesis in the heart (1). Adenosine-triphosphatase activity of myosin heavy chain α is markedly higher than myosin heavy chain β. T3 stimulates the expression of myosin heavy chain α but decreases the expression of myosin heavy chain β. This regulation may modulate myocardial contractility. Thyroid hormones increase cardiac actin and troponin I. Release of calcium and its reuptake into the sarcoplasmic reticulum are important for systolic and diastolic functions. The gene encoding calcium pump of the sarcoplasmic reticulum is markedly T3 responsive (2). Thyroid hormones can regulate β-adrenergic receptor number in the heart and may enhance sensitivity to catecholamines (1).

Atrioventricular blocks, sinus bradycardia, prolongation of the QT and QRS intervals and Torsades de Pointes were reported in hypothy-