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CASE REPORT / PRACA KAZUISTYCZNA

Wild at heart — multiple mechanisms of cocaine-induced myocardial infarction

Pułapki diagnostyczne zawału mięśnia sercowego typu drugiego w wyniku stosowania kokainy

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Abstract

Introduction. Results of cocaine intake can be such cardiovascular complications as hypertension, myocardial infarction, arrhythmia, and cardiomyopathy. Anabolic androgenic steroids use is related to hypertension, cardiomyopathy and lipid metabolism derangements.

Case report. On admission to the cardiology department a 37-year-old man presented prolonged pain at rest located in the middle part of the chest, which occurred 2 days earlier. He was after the use of cocaine, tetrahydrocannabinol, and alcohol. Smoking and using the growth hormone and anabolic androgenic steroids (AAS) in the past were present in the patient's medical history. Physical examination with no deviation, heart rate 72/min, and blood pressure 130/90 mm Hg. Features of anterior wall myocardial infarction (MI) were present in electrocardiogram — ST-elevation up to 3 mm in V2–V5. In laboratory tests, troponin T rise (1.99 ng/mL) and D-dimer (503 ng/mL) were observed. In echocardiography — apex hypokinesis and concentric left ventricular hypertrophy — max. thickness: 19 mm. No significant abnormalities were detected in an immediate coronary angiography. Heart damage of vascular etiology involving an apical region and partial interventricular septum were confirmed in magnetic resonance imaging. No significant arrhythmias are present in

electrocardiogram Holter monitoring. Conservative treatment was prescribed: beta blockers, atorvastatin, enoxaparin, captopril, acetyl salicylic acid (ASA), electrolytes *i.v.* and was released from hospital after 4 days. On discharge in a good general condition with a recommended further treatment.
Conclusions. The presented case is an example of ST-elevation myocardial infarction (STEMI) in a young patient with left ventricular hypertrophy of multifactorial aetiology (hypertrophic cardiomyopathy, athlete's heart, hypertrophy due to anabolic steroids, and growth hormone intake). Immediate coronary angiography showed no significant abnormalities. The patient during STEMI was after ingestion of cocaine and other stimulants which, apart from pulmonary embolism, vasospastic angina, and Takotsubo cardiomyopathy, can be an aetiology factor. Elimination of all modifiable risk factors is the key factor influencing successful therapy in this case.
Key words: acute coronary syndrome, cocaine, growth hormone, anabolic steroids, hypertrophic cardiomyopathy

Introduction

A 37-years-old patient was admitted to the hospital to the cardiology department 48 hours after an occurrence of prolonged retrosternal resting chest pain. The patient was after ingestion of cocaine, alcohol, and tetrahydrocannabinol. Medical history embraced smoking, using growth hormone (GH) and anabolic androgenic steroids (AAS). The man suffered from celiac disease and underwent cervical neck surgery. In physical examination, no significant abnormalities were found. Blood pressure was 130/90 mm Hg and heart rate was 72/min.

Initial diagnosis

Because of the presented symptoms and history of cocaine, GH and AAS intake, myocardial infarction (MI) type II was suspected.

Differential diagnosis

The differential diagnosis that should be taken into consideration in the presented patient is in Table 1 [1].

Table 1. Differential diagnosis

Probable causes of LV hypertrophy	**For**	**Against**
Hypertrophic cardiomyopathy	LV concentric hypertrophy	Diagnosis has to be confirmed by genetic test
Athlete's heart	Medical history	Diagnosis has to be confirmed by imaging test 3 months after a detraining
Hypertrophy due to anabolic steroids, GH intake	Medical history of presented substances intake	
Probable causes of ACS	**For**	**Against**
I type of MI	Majority of ST-elevation MI are associated with atherosclerotic plaque rupture, MRI results support vascular aetiology of heart damage	no significant changes in coronary angiography
MINOCA — myocardial infarction with non-obstructive coronary arteries in presented case — no detection of coronary occlusion due to atherosclerosis — non-obstructive coronary arteries as per angiographic guidelines, with no lesions ≥ 50% in a major epicardial vessel detection of elevated cardiac biomarkers (cardiac troponin > 99th percentile of the upper reference level), clinical symptoms of myocardial ischaemia, new ECG changes, evidence of new regional wall motion abnormality, identification of new loss of viable myocardium on MRI		
Coronary causes		
Takotsubo cardiomyopathy	Transient LV apical ballooning Left ventricular apical hypokinesis detected using echocardiography	Significant rise in troponin level In presented case MRI results support vascular aetiology of heart damage
Variant angina (Prinzmetal angina) — vasospastic angina	Probable coronary artery spasm due to cocaine intake	Transient changes in ECG during variant angina episode the lack of vasoactive (provocative) test that could confirm or exclude this diagnosis (acetylcholine or

		ergonovine testing)
Microvascular angina — coronary microvascular dysfunction	No evidence of coronary occlusion	The lack of intracoronary provocative testing (acetylcholine or ergonovine tests)
Non-coronary causes		
Myocarditis	Clinical manifestations include prolonged retrosternal chest pain	No significant elevation of inflammation parameters No evident clinical symptoms of inflammation
Alternative diagnosis		
Pulmonary embolism	Co-occurrence of risk factors	No significant elevation of D-dimer concentration
Hypertensive crisis	Cocaine intake according to medical history	No evidence of hypertension in physical examination
Tachyarrhythmias	Cocaine intake according to medical history	No detection of specific supraventricular and ventricular arrhythmias in ECG Holter monitoring

AAS — anabolic androgenic steroids; ECG — electrocardiogram; GH — growth hormone; LV — left ventricular; MI — myocardial infarction; MRI — magnetic resonance imaging

Diagnostic process

Electrocardiogram revealed ST-elevation up to 3 mm in V2–V5 and negative T-wave in V3 and aVF — features of anterior wall MI (Figure 1). Laboratory tests demonstrated microcytic anaemia (erythrocytes — 4.32 × 10^6/uL, haemoglobin — 11.50 g/dL, hematocrit — 34.20%, mean corpuscular volume — 79.2 fl), lowered serum creatinine level (0.58 mg/dL) and high sensitivity troponin T rise (1.840 ng/mL). Other parameters were normal. Transthoracic echocardiography showed concentric left ventricular (LV) hypertrophy (interventricular septum thickness up to 19 mm and posterior wall thickness up to 12 mm), apex hypokinesis and presence of LV false tendon. LV ejection fraction was 58% (Figure 2).

The patient underwent immediate coronary angiography that showed no significant abnormalities in both right and left coronary arteries (Figure 3.) Magnetic resonance imaging (results available after the patient's discharge) revealed abnormalities (Figure 4.). No significant supraventricular and ventricular arrhythmias were present during electrocardiogram Holter monitoring.

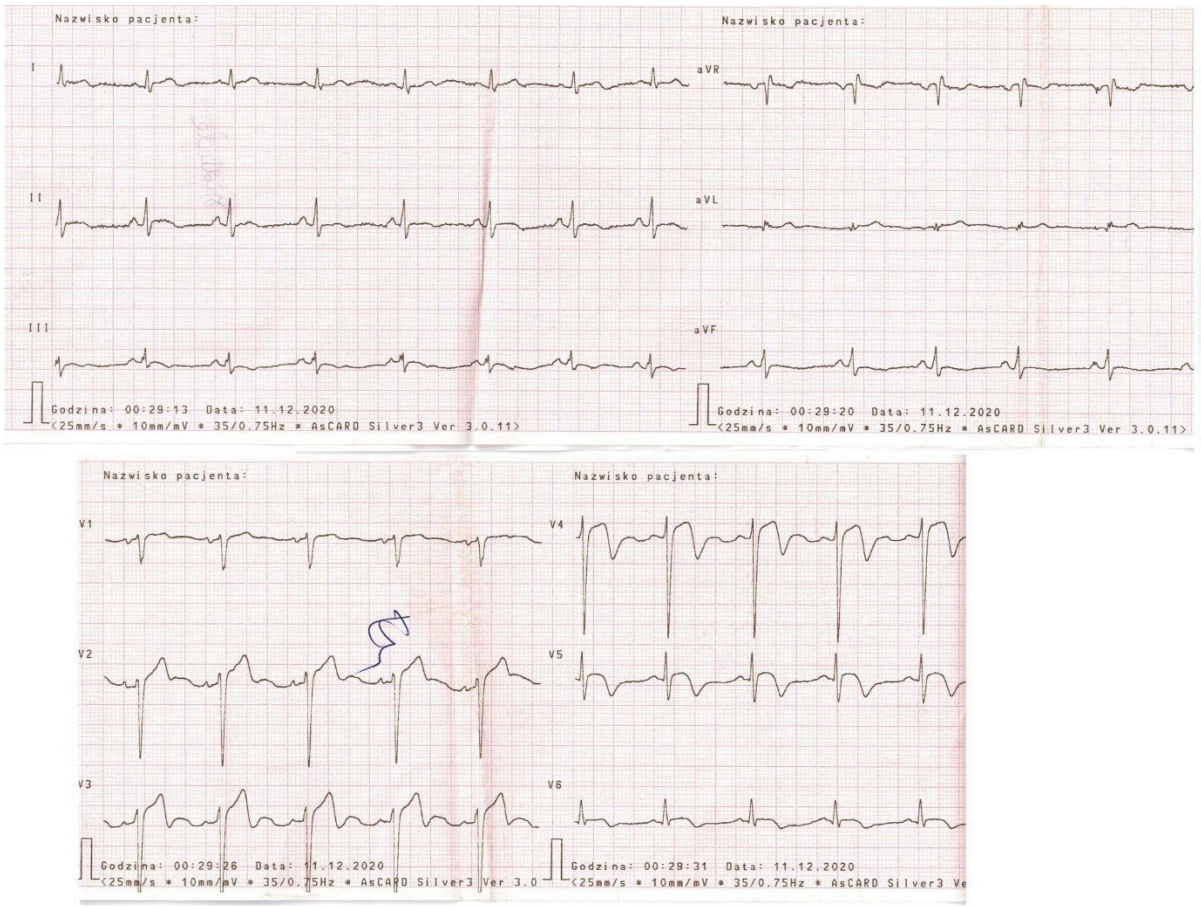

Figure 1. 12-lead electrocardiogram: Normal axis, sinus rhythm, heart rate about 70/min., J point and upward ST-segment elevation in V2–V4 (about 0.2 mV), horizontal ST elevation in V5 about 0.1 mV. "rS" morphology of QRS complex in V1–V4, and "RS" in V5. Biphasic T waves with initial deflection positive and terminal deflection negative in V3–V4, negative T waves in V5, and slightly negative T waves in III and V6. Interventricular conduction disturbances in III. PR interval — 180 ms, QT interval 420 ms, QTc interval 454 ms, QRS complex time about 90 ms

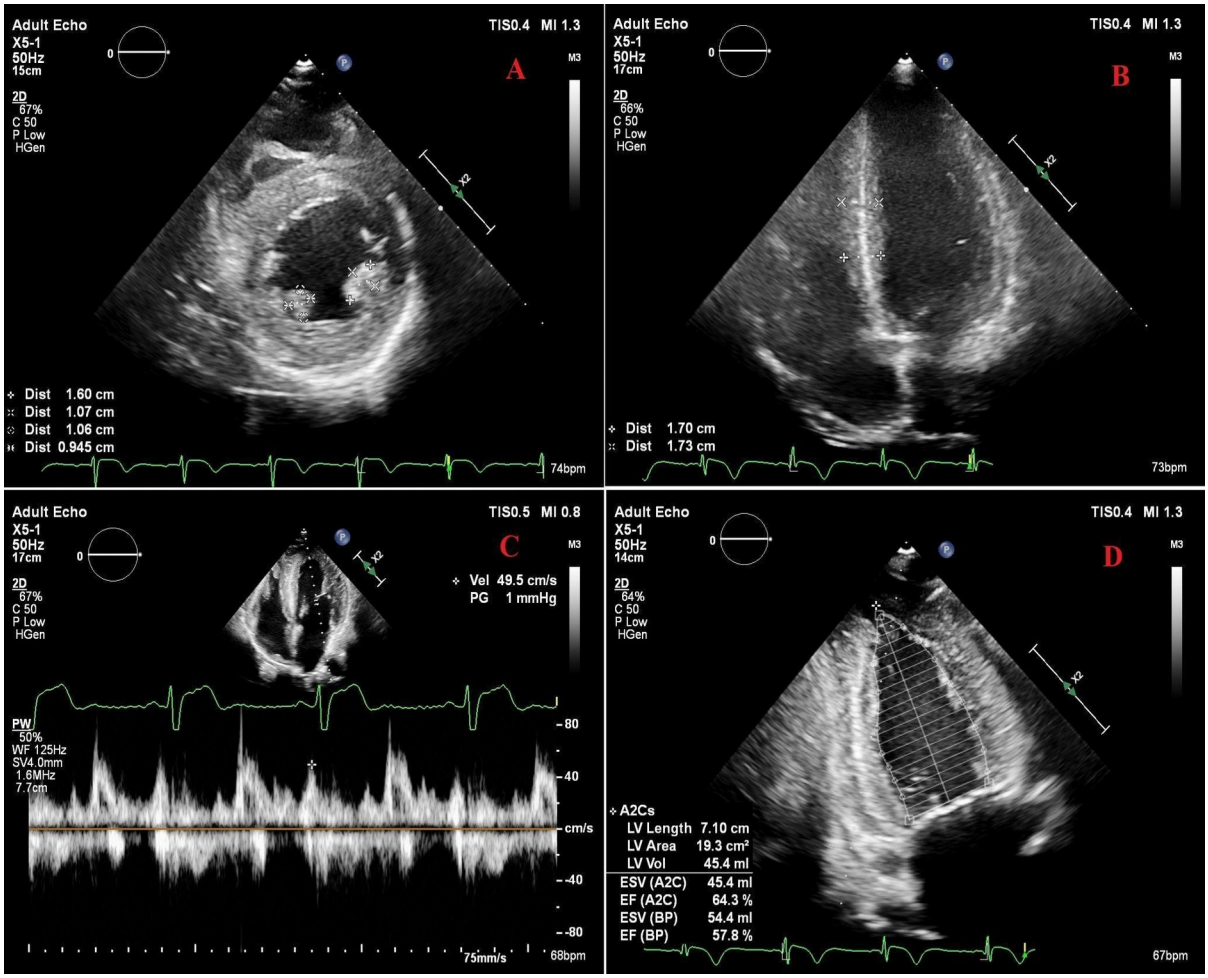

Figure 2. Transthoracic echocardiography: **A**. Parasternal short axis, thickening of the papillary muscles; **B.** Apical 4 chamber view, increased thickness and brightness of IVS myocardium; **C.** Pulse doppler imaging: mitral flow; **D**. Apical 2-chamber view, assessment of LV ejection fraction, concentric LV hypertrophy; IVS — interventricular septum; LV — left ventricular

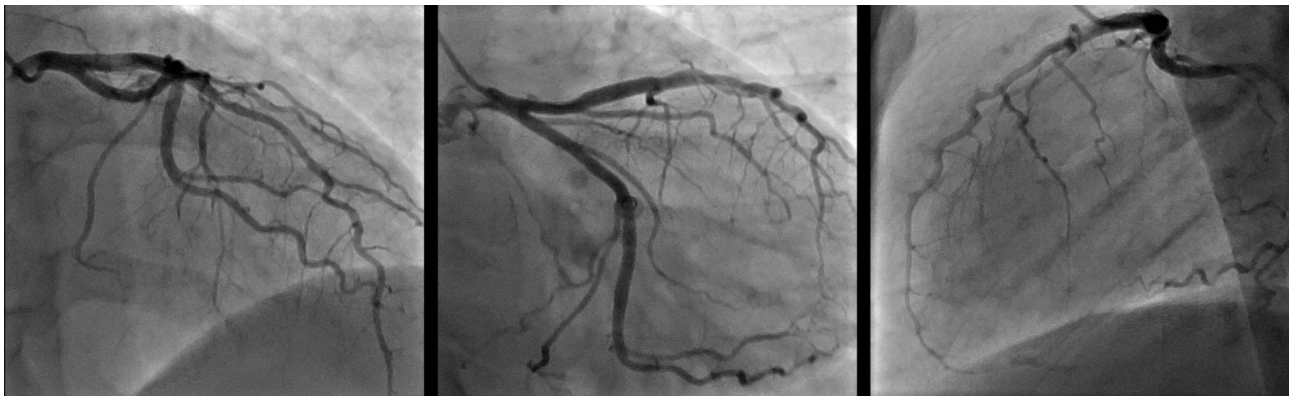

Figure 3. Coronary angiography; the dominance of the left coronary artery (LCA), narrowing and irregular outlines of left anterior descending artery (LAD), suspected intramuscular course of LAD

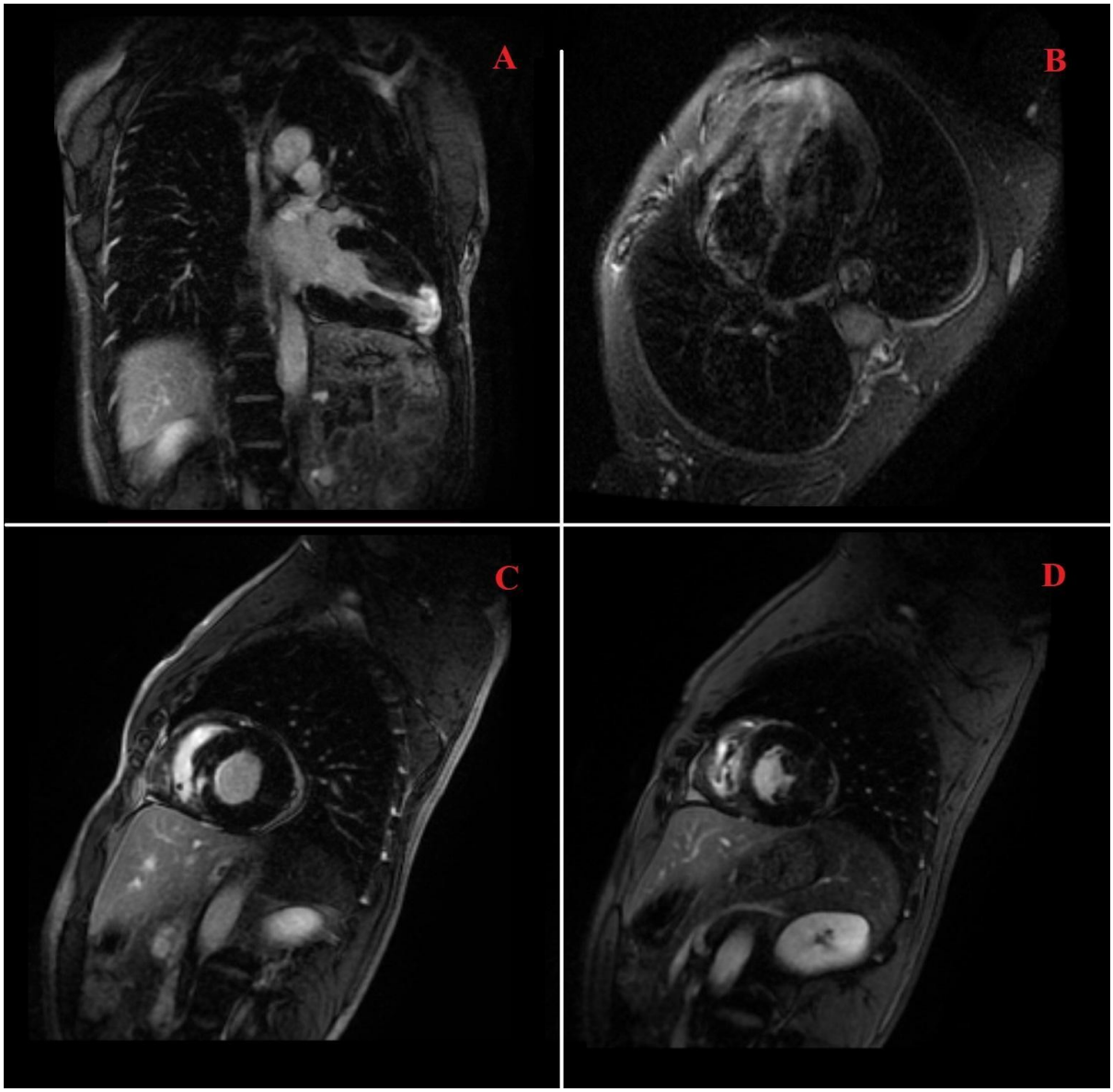

Figure 4. Cardiac magnetic resonance: **A.** Transmural late gadolinium enhancement of the left ventricle apex; **B.** Hyperintense signal at the left ventricular apical part in long axis double inversion recovery dark blood image; **C.** Intramural focal late gadolinium enhancement in the basal inferoseptal segment; **D.** Intramural late gadolinium enhancement in the mid septal segments, mild pericardial effusion along the lateral wall

Therapeutic procedure

Coronarography results were ambiguous so interventional procedures were not performed. Conservative treatment was implemented: enoxaparin, acetyl salicylic acid (ASA), atorvastatin, captopril, protein-protein interactions (PPIs), beta-blockers (bisoprolol,

nebivolol). The patient responded well to the therapy, but an increased concentration of serum troponin was observed (1.990 ng/ml). The patient was discharged after 4 days in good general condition with recommendations for further treatment: ASA, atorvastatin, nebivolol, PPIs, zofenopril, a control appointment in the cardiology clinic in 2–3 weeks, and contraindications to smoking, drugs, and alcohol.

Final diagnosis

Considering the past medical history and the results of the diagnostic process, MI type II was diagnosed. It was caused by decreased oxygen supply due to the coronary artery spasm and increased oxygen demand because of myocardial hypertrophy.

Discussion

When searching for aetiology of acute coronary syndromes (ACS) among young patients, apart from the genetic background and metabolic disorders, stimulant drugs and muscle growth stimulants need to be considered.

The influence of particular substances (cocaine, tetrahydrocannabinol, AAS) on the cardiovascular system is visible in lipid metabolism derangements, endothelial damage, excessive production of free radicals. These lead to earlier atherosclerosis, affecting coronary arteries [2].

Molecular cocaine effects embrace adrenergic stimulation because of blocking noradrenaline reuptake which causes various cardiovascular complications. They can be both acute cardiac arrhythmia, a sudden increase in blood pressure, ACS and chronic LV hypertrophy, earlier atherosclerosis. ACS occurs from a few minutes to a few hours after cocaine intake [2, 3].

The influence of GH on the cardiovascular system embraces heightened angiogenesis in the heart, cardiac myocytes growth promotion, antiapoptotic activity, increased NO production. GH stimulates the proliferation of endothelial cells by activating the expression of eNOS, promoting angiogenesis. It can also increase vascular resistance and inflammation [4].

Anabolic androgenic steroids causes lipid derangement, elevated blood pressure, coagulation alterations, atherosclerosis promotion, heart muscle hypertrophy. These changes

can increase the risk of cardiovascular disorders — coronary artery disease, arrhythmias, and congestive heart failure [2, 5].

Most ACS with ST-elevation are MI type I [6]. The STEMI in the presented case had a different aetiology. The lack of occlusion in coronary angiography allowed to diagnose MI type II. In differential diagnosis, we take into consideration transient coronary vasospasm, an arterial embolism because of possible hypercoagulability after AAS use, dehydration after alcohol and chronic inflammation due to celiac disease [5].

Concentric LV hypertrophy could be caused by several factors: physiological adaptation due to physical exercises, AAS and GH intake, as well as unrecognised before hypertrophic cardiomyopathy [4, 5, 8]. Left ventricular hypertrophy heightens heart's oxygen demand and alongside coronary artery spasm, hyperkinetic circulation due to anaemia, tachycardia can lead to myocardial supply-demand imbalance [7]. Elimination of all reversible risk factors is crucial for further treatment of the patient.

Streszczenie

Wstęp. Kokaina stanowi drugą pod względem popularności substancję narkotyczną. Jej zażywanie może skutkować chorobą niedokrwienną serca w postaci ostrych zespołów wieńcowych, arytmii, kardiomiopatii czy nadciśnienia tętniczego. Przyjmowanie steroidów anabolicznych związane jest z rozwojem nadciśnienia tętniczego, kardiomiopatii oraz zaburzeń w gospodarce lipidowej.

Opis przypadku. 37-letni mężczyzna został przyjęty na oddział kardiologii z powodu przedłużającego się od dwóch dni epizodu bólu w klatce piersiowej o charakterze dławicowym bez objawów towarzyszących. Stwierdzono stan po spożyciu kokainy, tetrahydrokannabinolu, alkoholu, w przeszłości palenie tytoniu, przyjmowanie steroidów anabolicznych, hormonu wzrostu. W wywiadzie obecność celiakii, stan po operacji w obrębie kręgosłupa szyjnego. W badaniu fizykalnym nie stwierdzono odchyleń od normy (ciśnienie krwi 130/90 mm Hg, rytm serca 72/min). W badaniu elektrokardiograficznym (EKG) zaobserwowano uniesienie odcinka ST do 3 mm w V2–V5, ujemne załamki T w V3, aVF (niedokrwienie przedniej ściany mięśnia sercowego). W badaniach laboratoryjnych stwierdzono znaczący wzrost stężenia hsTnT, obniżone stężenie kreatyniny, anemię mikrocytarną. Podczas badania echokardiograficznego uwidoczniono koncentryczny przerost lewej komory (LV), hipokinezę okolicy koniuszka LV, frakcja wyrzutowa LV 58%. Wykonano koronarografię w trybie pilnym, nie stwierdzono istotnych zmian. W MRI zaobserwowano zmiany sugerujące zawał koniuszka oraz dystalnej części IVS.

Monitorowanie EKG metodą Holtera nie ujawniło arytmii. Wdrożono standardową terapię OZW: enoksaparyna, ASA, atorwastatyna, kaptopryl, IPP, bisoprolol, nebiwolol, NaCl, PWE. Po 4 dniach hospitalizacji pacjent w dobrym stanie ogólnym został wypisany z oddziału z zaleceniem kontynuacji leczenia za pomocą ASA, atorwastatyny, nebiwololu, IPP, zofenoprylu, oraz kontrolą w poradni w ciągu 2–3 tygodni.

Wnioski. Prezentowany przypadek stanowi przykład STEMI u młodego pacjenta z przerostem lewej komory o wieloczynnikowym podłożu (HCM, serce sportowca, wtórny przerost w wyniku stosowania steroidów anabolicznych i hormonu wzrostu). Wykonana w trybie pilnym koronarografia nie wykazała zmian w naczyniach wieńcowych. Chory w momencie STEMI był pod wpływem kokainy oraz innych używek, co może stanowić czynnik etiologiczny obok zakrzepicy tętniczej, dławicy wazospastycznej, kardiomiopatii Takotsubo. Eliminacja wszystkich modyfikowalnych czynników ryzyka jest kluczowa w leczeniu tego chorego.

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