The Case of Myocardial Infarction in a Fifteen-Year-Old Adolescent Caused by Toxic Substances

O. Loskutov, Yu. Markov, B. Todurov, O. Druzhyna, V. Kolesnykov & S. Maruniak

Cardiovascular Toxicology

ISSN 1530-7905
Volume 20
Number 5

Cardiovasc Toxicol (2020) 20:520-524
DOI 10.1007/s12012-020-09575-5

Springer

Cardiovascular Toxicology (2020) 20:520–524
https://doi.org/10.1007/s12012-020-09575-5

The Case of Myocardial Infarction in a Fifteen-Year-Old Adolescent Caused by Toxic Substances

O. Loskutov[1,2] · Yu. Markov[1] · B. Todurov[2] · O. Druzhyna[1,2] · V. Kolesnykov[1,2] · S. Maruniak[1,2]

Published online: 12 May 2020

Abstract

The article reveals about the clinical accident of myocardial infarction in an adolescent after visiting a night club, where he had been drinking tequila. A "sniper" test was performed at home and showed synthetic marihuana in urine. The uniqueness of this case is the adolescent's difficult premorbid condition (severe diabetes mellitus type 1 and sub-compensated hypothyreosis) and the clinical course of the disease had caused difficulty in diagnosis which led to a belated hospitalization on the third day. Considering pain syndrome, infarction-like findings in ECG (ST elevation), high levels of cardiospecific enzymes (troponin elevated 70 times, aspartate aminotransferase—two times, creatine phosphokinase—4 times, lactate dehydrogenase and alkaline phosphatase—two times) and their slow tendency to normalize—a non-Q myocardial infarction diagnosis was performed. After complex treatment, the general condition of the patient improved; he was discharged from the clinic in satisfactory general condition and was given recommendations for further rehabilitation. Given the potential for premature death and long-term disability with the development of negative individual and social consequences, the correct diagnostic assessment in such cases becomes particularly relevant.

Keywords Myocardial infarction in adolescent · Toxic substances · Infarction-like findings in ECG · Cardiospecific enzymes

Abbreviations

ALP	Alkaline phosphatase
AST	Aspartate aminotransferase
BP	Blood pressure
CPK-MB	Creatine phosphokinase-MB
DM	Diabetes mellitus
ECG	Electrocardiography
HbA1c	Glycated hemoglobin
HDL	High density lipoprotein
HR	Hear rate
LDH	Lactate dehydrogenase
LDL	Low density lipoprotein
MI	Myocardial infarction
T_3	Triiodothyronine
TG	Triglycerides

Handling editor: Y. James Kang.

✉ S. Maruniak
maruniak.stepan@gmail.com

[1] Anesthesiology and Intensive Care Department, P. L. Shupyk National Medical Academy of Postgraduate Education, Kyiv, Ukraine

[2] State Institution "Heart Institute Ministry of Health of Ukraine", Kyiv, Ukraine

Introduction

Cardiovascular diseases cause more than 17.3 million deaths per year and are the leading cause of death worldwide. In 2017, 40,786 cases of myocardial infarction (MI) were registered in Ukraine, out of this number 11853 heart attacks in people of working age [1]. Less than 2% of all acute MI occur in people under 35 years [2]. As far as MI is common in the adult population, it is rare in children. Thus, according to Maslennikova, the prevalence of myocardial ischemia in the population of children and adolescents aged 0 to 18 years is 4.48% [3]. Most cases of acute MI in childhood are secondary to congenital abnormalities or Kawasaki disease (total occlusion of the coronary artery caused by fibromuscular dysplasia) [4]. Although there are cases of MI even in newborns [5, 6].

The development of MI with the elevation of the ST-segment and elevated cardiospecific enzymes as a result of a motorcycle injury in a 16-year-old guy is described [7]. MI

occurs even in adolescents without anatomical abnormalities of the coronary vessels [8]. Briassoulis et al. describe a case of MI in a 10-year-old child with meningococcemia [9].

Below is a case of developing MI, provoked by exogenous toxic factors on the body of a teenager.

Case Reports

Patient M., 15 years old, was admitted on the third day of the disease to the Heart Institute of the Ministry of Health of Ukraine in connection with complaints of unmotivated general weakness, pain in the left half of the chest, changes on the ECG similar to a heart attack.

It is known from the history that at night 2 days before the beginning of the disease he visited a nightclub, where after drinking tequila he felt worse: there were complaints of weakness, which increased in the morning, then there was retrosternal pain. Before this case, the patient tried low alcohol drinks, did not consume drugs/psychotropic drugs, there was no medical history. On the morning of the next day, the condition worsened at 12.00. There was one-time vomiting, aching in the body, the body temperature rose to 37.3 °C.

After about 12 h of drinking an alcoholic beverage, according to a record of the doctor of emergency medical care (9.35 pm): "The condition of the child is moderate, sluggish, adynamic. There was one-time vomiting. Skin and visible mucous membranes of normal color, without pathological rash. Mucous oropharynx is brightly hyperemic, tonsils without features. Nasal breathing is free. The frequency of respiratory excursions is 18 per minute. Vesicular breathing in the lungs, no wheezing. The heart tones are rhythmic, loud. Heart rate (HR)—88 beats per 1 min. Blood pressure (BP)—120/80 mmHg. Abdomen with no specific findings No symptoms of peritoneal irritation. Diuresis is sufficient. No meningeal symptoms. Thermometry + 37.3 °C. The patient's mother and he refused the proposed hospitalization."

The next day, according to the patient's mother, a "Sniper" urine test was performed at home, which found synthetic marijuana in it. After that, the parents called an emergency medical team. In the latter case, an ECG was recorded, where the elevation of the ST-segment in the lower and thoracic leads was detected.

At the time of admission to the hospital, the patient's general condition is regarded as severe and stable. Respiratory rate—24/min., HR—112 beats/min., BP—100/60 mm Hg., capillary blood oxygen saturation—97%. Consciousness-clear, productive verbal contact is available, the nature of the contact corresponded to the essence. Correctly comprehensively oriented. The skin is pale. Signs of vasospasm were observed (capillary filling time exceeded 3 s). Vesicular breathing in the lungs. Percussion borders of the heart are not increased.

On the ECG: sinus rhythm, regular with HR of 75 beats per 1 min., pronounced elevation of the ST-segment in standard and thoracic leads (V_4-V_5) (Fig. 1).

According to echocardiography: hypertrophy of the posterior wall of the left ventricle. Segmental contractility of the left ventricle is preserved at rest. Heart cavities within the normal range. Mitral valve prolapse with mitral regurgitation grade I–II. Slight mitral valve regurgitation. The diastolic function of the left ventricle is not impaired. No signs of pulmonary hypertension. Magistral blood flow type in abdominal aorta. A small amount of fluid in the pericardial cavity. The left atrium and right atrium are within normal range. End-diastolic volume—90 ml, End-diastolic index—49 ml/m^2, ejection fraction—60%, interventricular

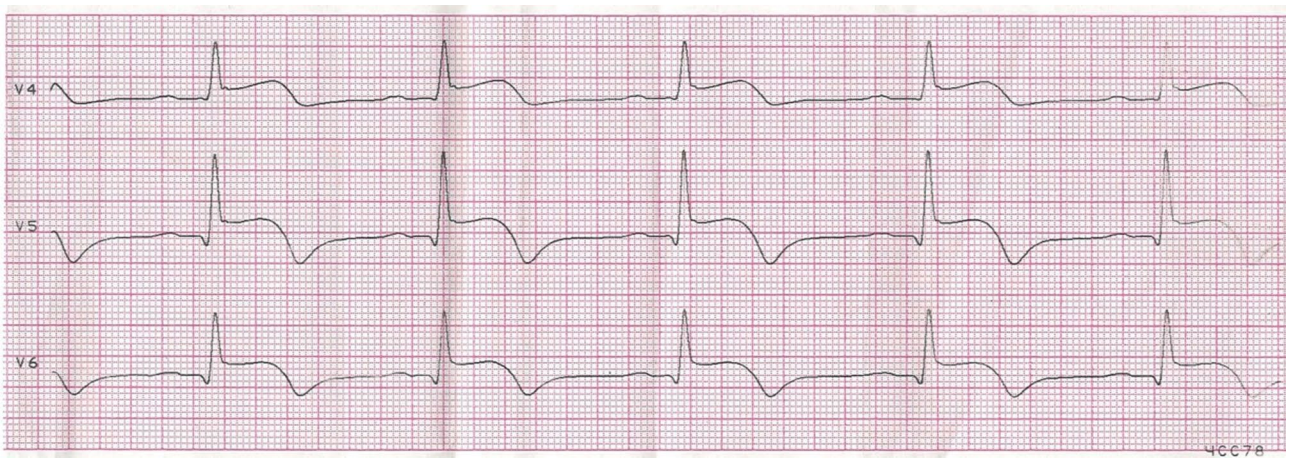

Fig. 1 Electrocardiogram of the patient M. at admission to the clinic. Sinus rhythm, correct. HR = 72–73/min. PQ = 0.13 s, QRS axis = 60°. Diffuse violations of repolarization processes in the anterior wall, septum, and apex of the left ventricle with a transition to the side wall. Acute stage of anterior prevalent lateral IM

septum—0.9 cm, posterior wall of the left ventricle—1.2 cm. Systolic pressure in the right ventricle—28 mmHg separation of pericardial leaves along the contour of the free wall of the right ventricle—4 cm of free fluid.

In order to clarify the anatomy of the coronary arteries, coronaroventriculography was performed: no hemodynamically significant stenoses of the coronary arteries, anomalies of development, aneurysms of the coronary vessels or angioectasias were detected (Fig. 2).

The examination of the endocrinologist: diabetes mellitus type 1, severe form, the degree of decompensation. Subcompensated hypothyroidism.

In the biochemical blood test for the 1-st day of hospitalization: elevation of troponin I level up to 17.38 ng/ml ($N = 0–0.3$ ng/ml), aminotransferase aspartate (AST) up to 102 E/l, creatine phosphokinase-MB (CPK-MB) up to 65.6 E/l, lactate dehydrogenase (LDH) up to 471 E/l and alkaline phosphatase (ALP) up to 157 E/l, mean daily blood glucose level—11.8 mmol/l, mean daily glycated hemoglobin (HbA1c)—up to 9.75% (Table 1).

The parameters of the lipid profile (total cholesterol, high density (HDL) and low density lipoproteins (LDL), triglycerides (TG), atherogenicity index) and aggregation profile did not exceed the reference values.

Taking into account the presence of pain syndrome, changes on the ECG similar to a heart attack (ST-segment elevation), high titers of cardiospecific enzymes (70 times increased troponin I, twice—AST, four times—CPK-MB, LDH and twice—ALP), the main diagnosis was made: acute non-Q-myocardial infarction. Mitral valve prolapse with regurgitation grade I-II. Exudative pericarditis. Concomitant diagnosis: type 1 diabetes mellitus (DM type 1), severe form. Subcompensated hypothyroidism.

Table 1 The dynamics of the main indicators in patient M

Day Indicators	1-st	2-nd	6-th	9-th
T (°C)	37,3	37,4	36,8	36,7
HR (b/min)	112	94	78	81
BP (mmHg)	100/60	110/65	125/70	120/70
Troponin I (ng/ml)	17.38	15.94	1.28	0,11
CPK-MB (E/l)	65.6	43.1	5.7	5.1
AST (E/l)	102	68	30	37
LDH (E/l)	471	348	201	167
ALP (E/l)	157	143	110	115
HbA1c (%)	9,75	9.69	9.41	9.36
LDL (mmol/l)	1,95	1,90	1,71	1,74
HDL (mmol/l)	> 0,9	> 0,9	> 0,9	> 0,9
TG (mmol/l)	0.84	0.79	0.76	0.81

T body temperature, *HR* hear rate, *BP* blood pressure, *CPK-MB* creatine phosphokinase-MB, *AST* aspartate aminotransferase, *LDH* lactate dehydrogenase, *ALP* alkaline phosphatase, *HbA1c* glycated hemoglobin, *LDL* low density lipoprotein, *HDL* high density lipoprotein, *TG* triglycerides

After conservative treatment, which included infusion therapy, quercetin (1 g × 3 times a day) as antioxidant and antitoxic drug, acetylsalicylic acid 100 mg/day to prevent of platelet aggregation, Asparkam (magnesium Asparaginate 0.175 g + potassium Asparaginate 0.175 g) 1 tablet × 3 times/day to correction of electrolyte disturbances, esomeprazole to prevent ulcer formation (40 mg/day) and insulin 50 units/day (pump) due to DM, the patient's condition improved.

Rapid positive dynamics of the general condition, ECG parameters (ST interval, QRS voltage), normalization of the activity of cardiospecific enzymes were noted on the background of treatment. On day 9, the patient was discharged from the hospital for further rehabilitation.

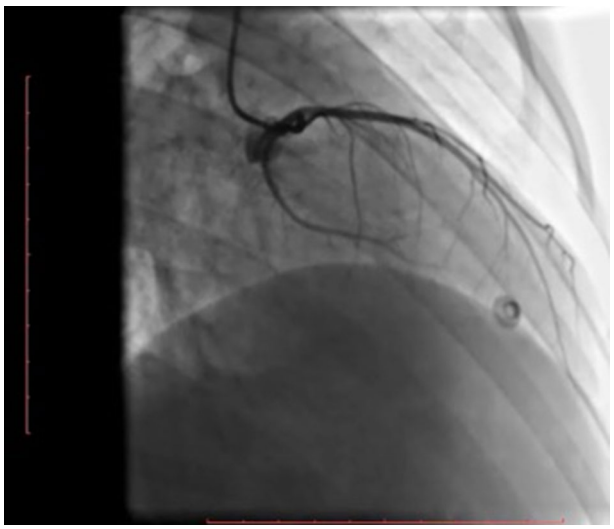

Fig. 2 Coronarography of the patient M. Left coronary artery without pathology: no stenotic changes, developmental abnormalities, aneurysms, angioectasia were detected

Discussion

Among patients with MI, comorbidities such as DM (25.4%), tobacco smoking (42.2%), and left ventricular hypertrophy (5.2%) are found [10]. In young patients, such a risk factor as the use of illegal psychoactive substances among other factors is important [11].

From the standpoint of clinical toxicology, this case should be considered as the course and change of the toxicogenic stage of acute poisoning to somatogenic one. The latter arose as a result of the combined action of two toxic factors (tequila and marijuana), masked the clinical manifestations of acute coronary syndrome, which transformed into acute MI. In such cases, there is a risk of

misdiagnosis, since as a rule, there are no common risk factors associated with cardiovascular diseases [12].

Marijuana (cannabioids) cause tachycardia, an increase in systolic and diastolic BP due to the activity of the sympathetic nervous system. This leads to increased oxygen demand by the myocardium. There are also worse complications, including MI, cerebral ischemia, etc., which can lead to death [13].

The most common symptoms of acute coronary syndrome in these patients are: chest pain, almost half with irradiation, hyperhidrosis, nausea, and vomiting.

It has been shown that smoking marijuana increases the risk of MI by 4.8 times within 60 min of its consumption and increases the annual risk of MI with daily cannabis consumption from 1.5 to 3% per year. Modeling this effect on humans and animals suggests that this effect may be due to spasm of the coronary arteries [13].

In our case, marijuana could also affect the level of triiodothyronine (T_3) against the background of existing hypothyroidism, which could cause negative inotropic cardiac effects and aggravate myocardial ischemia.

The effects of cannabinoids on thyroid function were first noted in 1965, when it was shown that marijuana extract reduces the accumulation of iodine in the thyroid gland of rats [14]. Reports by Porcella et al. showed that delta tetrahydrocannabinol, the main psychoactive cannabinoid component of marijuana, is able to suppress the hormonal activity of the thyroid gland [15]. An animal study by Rosenkrantz et al. showed that marijuana smoke reduced the level of thyroid hormone T_3 by 17–29% [16].

The clinical picture of myocardial infarction in the described patient was blurred by dyspeptic phenomena, which can be explained by the peculiarities of the pathophysiological effect of marijuana in patients with DM type 1. For example, in studies of Akturk et al. with a change in intestinal motility, which can distract the doctor's attention in the diagnosis of MI [17]. Moreover, as the authors note, cannabis users had an average of 0.41% higher levels of HbA1c than those who did not use drugs, which increases the risk of heart attack, stroke, renal failure, and death.

Conclusion

Given the potential for premature death and long-term disability with the development of negative individual and social consequences, proper diagnostic assessment in such cases is particularly relevant.

Compliance with Ethical Standards

Conflict of interest The authors declare no conflicts of interest and do not have any financial disclosures.

Informed Consent Written, informed consent for publication was obtained from the next of kin.

References

1. Kovalenko, M. V., Kornatsky, M. V. (2018). Actual health problems and their minimization in the context of armed conflict in Ukraine. Kyiv. 214 p. (in Ukrainian).
2. Fournier, J. A., Cabezón, S., Cayuela, A., Ballesteros, S. M., Cortacero, J. A., & Díaz De La Llera, L. S. (2004). Long-term prognosis of patients having acute myocardial infarction when %3c/= 40 years of age. *American Journal of Cardiology, 94*, 989–992.
3. Maslennykova, G.P. (2006). Prevalence of myocardium ischemia in children and adolescents. Vestnyk Orenburhskoho Hosudarstvennoho unyversyteta. Spets. Vypusk Nauka Tekhnolohyy Proyzvodstvo Rynok. 13(63):256 (in Russian).
4. Lin, M. C., Lee, W. L., & Fu, Y. C. (2015). Successful percutaneous transluminal coronary angioplasty for acute myocardial infarction in a 12-year-old boy with fibromuscular dysplasia: A case report. *Cardiology in the Young, 25*(1), 177–180.
5. Papneja, K., Chan, A. K., Mondal, T. K., & Paes, B. (2017). Myocardial infarction in neonates: A review of an entity with significant morbidity and mortality. *Pediatric Cardiology, 38*(3), 427–441.
6. Perrier, S., Parker, A., Brizard, C. P., Sheridan, B., Konstantinov, I. E., d'Udekem, Y., et al. (2017). Surgical management of extensive perinatal myocardial infarction. *Annals of Thoracic Surgery, 104*(6), 435–437.
7. Park, W. S., Jeong, M. H., Hong, Y. J., Park, O. Y., Kim, J. H., Kim, W., et al. (2003). A case of acute myocardial infarction after blunt chest trauma in a young man. *Journal of Korean Medical Science., 18*(6), 889–893.
8. Lane, J. R., & Ben-Shachar, G. (2007). Myocardial infarction in healthy adolescents. *Pediatrics, 120*(4), 938–943.
9. Briassoulis, G., Kalabalikis, P., Thanopoulos, V., & Hatzis, T. (2000). Non-Q wave acute myocardial infarction in acute meningococcemia in a 10-year-old girl. *Pediatric Emergency Care, 16*(1), 33–38.
10. Foraker, R. E., Guha, A., Chang, H., O'Brien, E. C., Bower, J. K., Crouser, E. D., et al. (2018). Survival after MI in a community cohort study: Contribution of comorbidities in NSTEMI. *Global Heart., 13*(1), 13–18.
11. Fazila-Tun-Nesa, M., Kalimuddin, M., Ahmed, N., Badiuzzzaman, M., Ahmed, M. N., Dutta, A., et al. (2016). AMI in very young (aged ≤35years) Bangladeshi patients: Risk factors & coronary angiographic profile. *Clinical Trials and Regulatory Science in Cardiology., 13*, 1–5.
12. Hill, D., Waldman, A., & Vivek, D. (2016). A 16-year-old with ST elevation myocardial infarction: Case report and review of the literature. *Cardiology in the Young, 26*(2), 230–236.
13. Franz, C. A., & Frishman, W. H. (2016). Marijuana use and cardiovascular disease. *Cardiology Review, 24*(4), 158–162.
14. Brown, T. T., & Dobs, A. S. (2002). Endocrine effects of marijuana. *Journal of Clinical Pharmacology, 42*(S1), 90S–96S.
15. Porcella, A., Marchese, G., Casu, M. A., Rocchitta, A., Lai, M. L., Gessa, G. L., et al. (2002). Evidence for functional CB1 cannabinoid receptor expressed in the rat thyroid. *European Journal of Endocrinology, 147*(2), 255–261.

16. Rosenkrantz, H., & Esber, H. J. (1980). Cannabinoid-induced hormone changes in monkeys and rats. *Journal of Toxicology and Environment Health, 6*(2), 297–313.
17. Akturk, H. K., Taylor, D. D., Camsari, U. M., Rewers, A., Kinney, G. L., & Shah, V. N. (2019). Association between cannabis use and risk for diabetic ketoacidosis in adults with type 1 diabetes. *JAMA Internal Medicine, 179*(1), 115–118.