Sudden cardiac death with recovery of cardiac activity (clinical case)

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SOUHRN

Článek prezentuje klinický případ náhlé klinické smrti s obnovením srdeční činnosti u pacienta se specifickými klinickými prediktory náhlé smrti. Všechny elektrické události předcházející smrti byly zaznamenány Holterovým monitorem, který v té době pacienti nosili.

ABSTRACT

The article presents a clinical case of sudden clinical death with recovery of cardiac activity in a patient with specific clinical predictors of sudden death. All electrical events preceding the death were recorded by a Holter monitor worn by the patients at the time.

Introduction

Every year approximately 5 million people in the world die of sudden death (SD).[1,2] According to the International Classification of Diseases X revised and defined by WHO, the following are distinguished:

I 46.1 – sudden death;
I 45.0 – sudden death with recovery of cardiac activity;
I 46.9 – cardiac arrest (irreversible).[3]

In recent years, the global cardiology community has made a more detailed distinction between cardiac and non-cardiac causes of sudden death.[3] Definition of sudden death – natural unexpected death in an apparently healthy subject, in unwitnessed cases, when a person was last seen alive and functioning normally less than 24 hours before being found dead; in witnessed cases, as an acute change in cardiovascular status with time to death being less 1 hour. Definition of sudden cardiac death (SCD) – sudden, natural unexpected death due to cardiac causes, with or without an autopsy, in witnessed cases within 1 hour of onset of symptoms. Definition of sports-related sudden cardiac death – it is non-traumatic SCD occurring during or within 1 hour after moderate or high-intensity exercises. And the last definition – sudden arrhythmic death syndrome.[2,3]

Case report

In our case report we analyzed the ECG changes during the ambulatory Holter monitoring of a patient with clinical SD with recovery of cardiac activity.[4] The patient Y., 47 years old, was seen on 29 September 2022 by a cardiologist for a preventive examination. He did not complain

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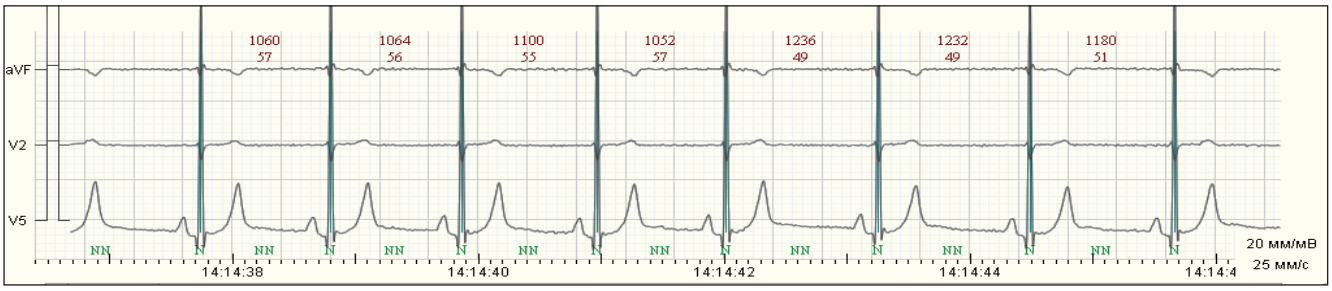

Fig. 1 – ECG phenomena of patient Y.: At the beginning of the monitoring the base rhythm was sinus regular (2:14:40 p.m.).

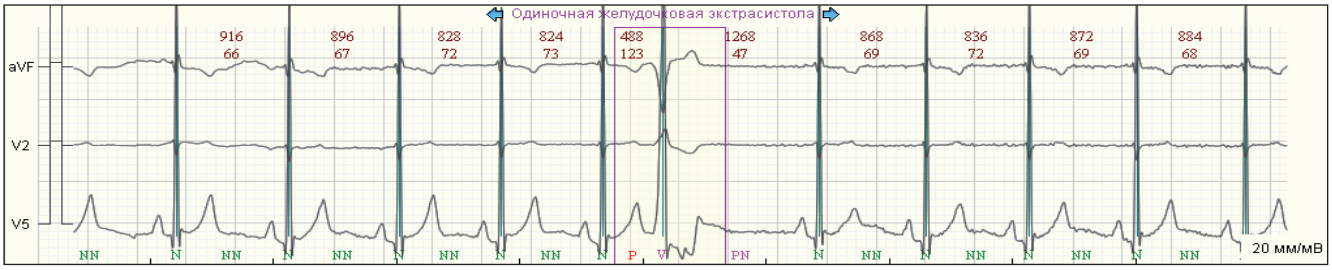

Fig. 2 – ECG phenomena of patient Y.: The occurrence of frequent ventricular ectopy (4:17:29 p.m.).

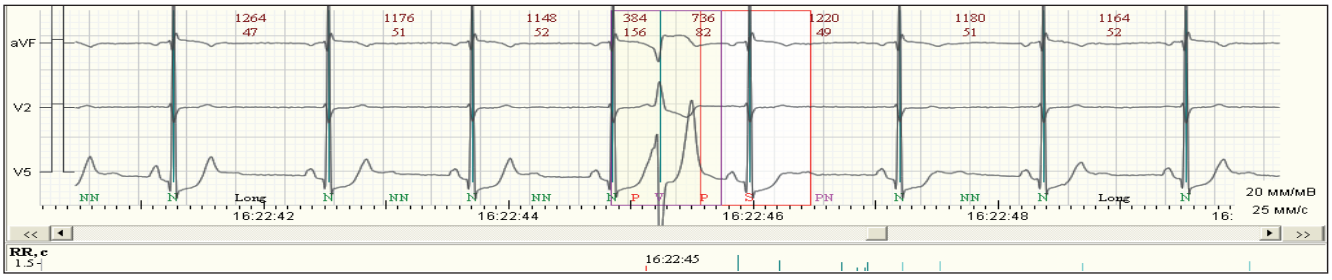

Fig. 3 – ECG phenomena of patient Y.: The "early" R on T interpolated ventricular extrasystole (4:22:48 p.m.).

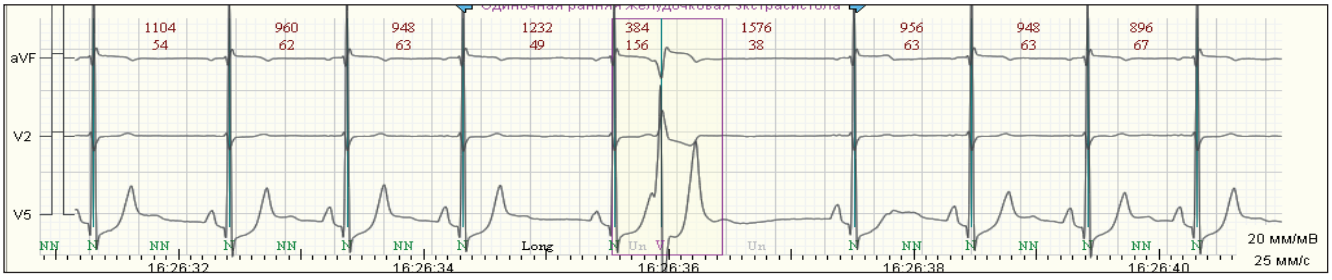

Fig. 4 – ECG phenomena of patient Y.: The "early" R on T ventricular extrasystole with post-extrasystolic suppression of the sinus node for the duration 1560 ms (4:28:36 p.m.).

of any chest pain or dyspnea on presentation and did not have any history of loss of consciousness. It is known from anamnesis that he had transient mild arterial hypertension (after some stress). He was not a smoker. The physical examination did not reveal any significant clinical findings. Ambulatory Holter monitoring was started at 9:17 AM. During the first hours of ambulatory Holter monitoring we diagnosed a decreased level of total power (heart rate variability) from 4768 ms^2 to 789 ms^2, low level of turbulence heart rate (TO = **0**.09%, TS = 1.93% ms/RR), and low level deceleration coefficient of heart rate – 1.51 ms.[5–11] We established that the patient had a very high risk of life-threatening arrhythmias and SD based on electrophysiological indicators[5,10,11] and we convinced him to be hospitalized in the clinic. From 3:30 PM that day, he stayed in the intensive care department of the Regional Clinical Cardiological Center (Ivano-Frankivsk, Ukraine). Some selected ECG strips that preceded the incident are shown below.[4] At the beginning of the monitoring, around 09:12 AM on 29/09/2022, it was determined that his base rhythm was sinus regular (Fig. 1). At around 4:17:29 PM, a sharp change in the electrical properties of the ventricle myocardium was noted: the occurrence of ventricular ectopy (VE) was noted. First, they were single, monotopically monomorphic VE in nature, then appeared 'early' R on T interpolated VE with non- and post-extrasystolic suppression of the sinus node (Fig. 2–4). At 4:20:44 PM a new phenomenon – hori-

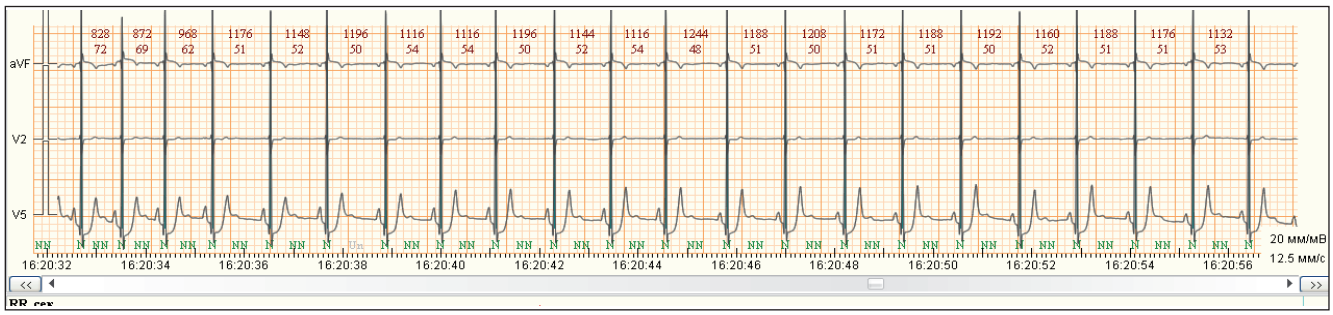

Fig. 5 – ECG phenomena of patient Y.: The occurrence of myocardial ischemia (horizontal depression of the ST segment within 150–200 μV) (4:20:44 p.m.).

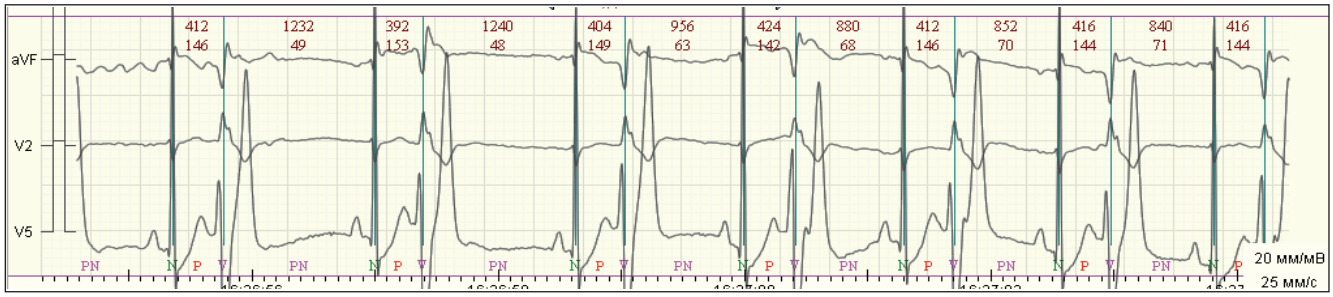

Fig. 6 – ECG phenomena of patient Y.: Ventricular bigeminy (4:30:56 p.m.).

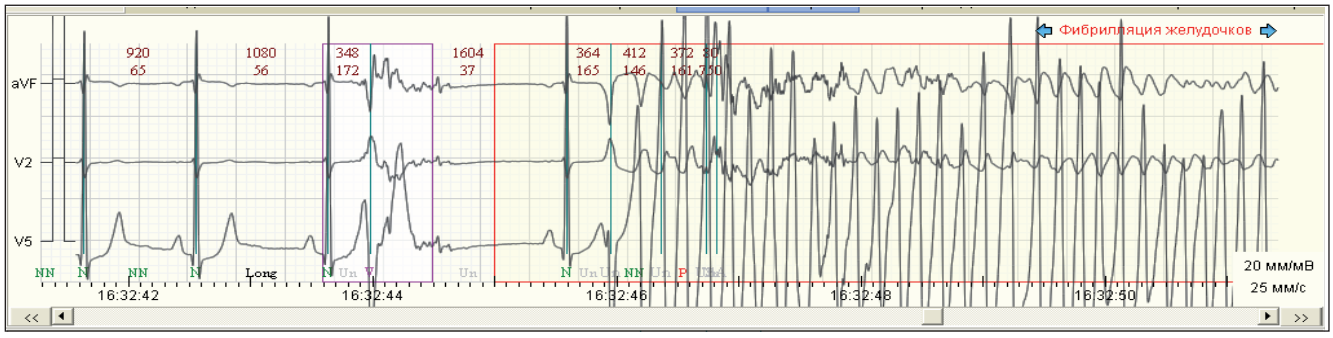

Fig. 7 – ECG phenomena of patient Y.: The "early" R-T ventricular extrasystole bigeminy with its initiation of the fluctuations in the width and amplitude of the ventricular tachycardia (4:32:44 p.m.).

zontal depression of the ST segment within 150-200 μV appeared, which may indicate disorders in coronary blood flow (Fig. 5). At 4:22:48 PM, the saddle-like pattern – 'early' R on T ventricular extrasystole, bigeminy were marked (Fig. 3,4,6), and at 4:32:44 PM paroxysm of tachycardia with wide QRS complexes developed, with ventricular rate around 200 beats/min (Fig. 7). Tachycardia can be categorized by the high probability of ventricular involvement. The dominant negative deviation of the QRS complexes in V5 should be emphasized, their predominantly positive deviation in the allocation of ventricular fibrillation. These signs suggest that the source of tachycardia is located in the left ventricle (LV), namely – in its anterior-septal zone.[12] The emergence of tachycardia was preceded by VE. QRS complexes of extrasystoles and tachycardia were identical. Interestingly, the adhesion intervals of the VE and tachycardia were different, which suggests a different mechanism of their formation despite their monomorphic nature. VE can be classified as early; they occur immediately after the ending of the T wave (Fig. 7). Tachycardia, on the contrary, arose after a delay in the ventricular tissue, which characterizes its possible mechanism as a "re-entry".[13] Nevertheless, their monomorphic character allows us to assume that the structures of the LV, where the VE were formed, as well as the path that depolarized the myocardium, were identical. However, the frequency of the second tachycardia was significantly higher (around 300 beats/min), as the variability of individual complexes. The VT was characterized by fluctuations within the broad limits of its electrophysiological properties and very quickly, within one minute, began to degrade into ventricular fibrillation (VF). Further, the disorganization of tachycardia morphology continued to increase, and it quickly became a large-wave VF (Figs 8, 9). From 4:32:54 till 4:34:24 resuscitation measures were carried. Seven shocks were applied: one time – 100 J, 2 times – 200 J , 2 times – 300 J and 2 times – 360 J) (Figs 9, 10). The post-defibrillation elevation of the segment ST decreased from 2000 μV to the level isoline (Figs 10, 11). All resuscitation measures were carried out according to the ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death and they lasted 1 min 44 s.[3] When electrical defibrillations were ineffective, we ad-

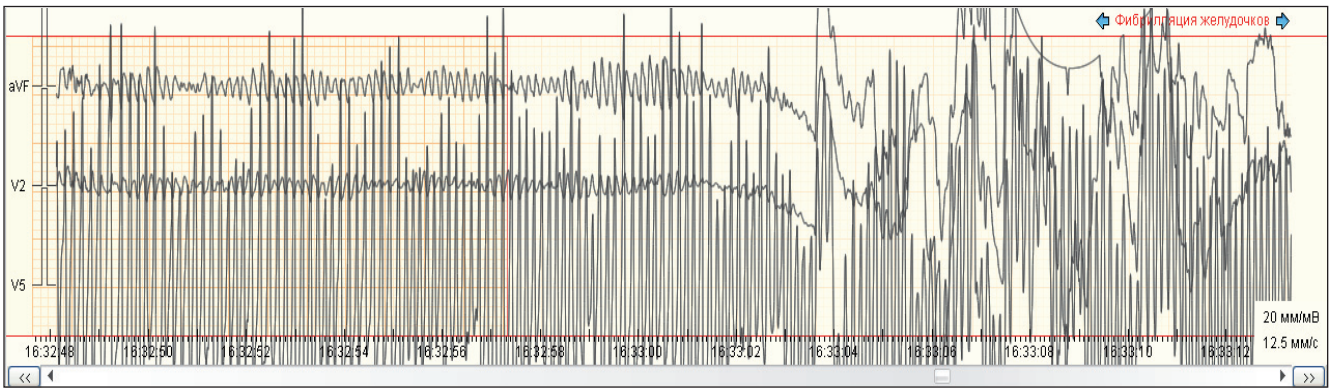

Fig. 8 – ECG phenomena of patient Y.: Progressing disorganization of ventricular tachycardia morphology and it transformation into large--wave ventricular fibrillation (4:32:54–4:33:04 p.m.).

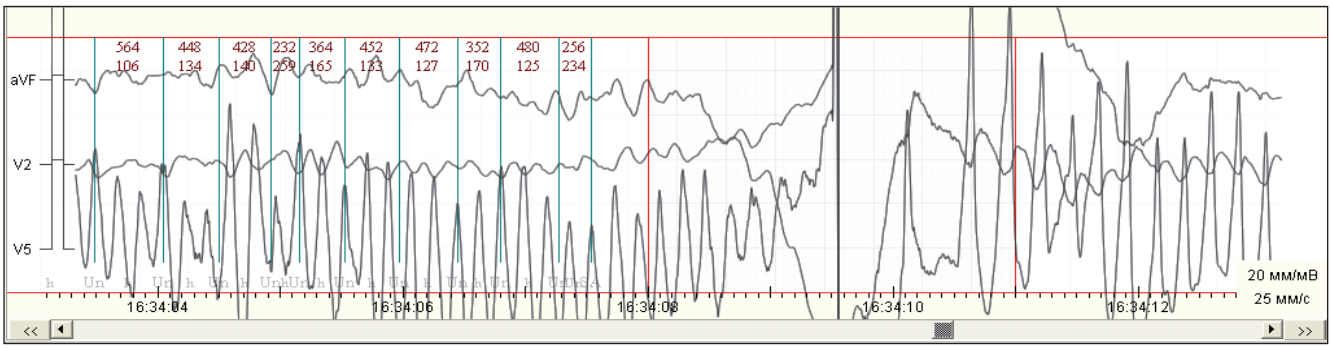

Fig. 9 – ECG phenomena of patient Y.: Ventricular fibrillation and moment of defibrillation, biphasic shock 200 J (4:34:09 p.m.).

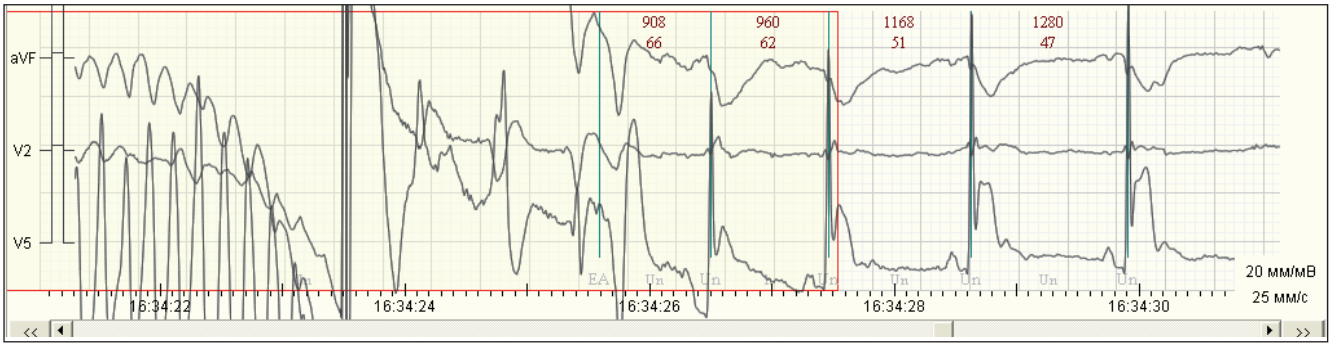

Fig. 10 – ECG phenomena of patient Y.: The large-wave ventricular fibrillation, than a moment of defibrillation by biphasic shock of 360 J, and after it the restoration of sinus rhythm. The effect of electrodamaging action after defibrillation is horizontal elevation of the ST segment up to 2000 μV (4:34:24 p.m.).

ministered a bolus of 1 gram of phosphocreatine (PC) solution intravenously to the patient.[14] The combination of a commonly accepted antiarrhythmic agent (amiodarone) with mitotrope agent (PC) allowed to enhance the antiarrhythmic effect and restore sinus rhythm, to improve microcirculation of the myocardium due to anti-aggregation properties and non-inducing electrodamaging action of defibrillation (Figs 11, 12).[15,16]

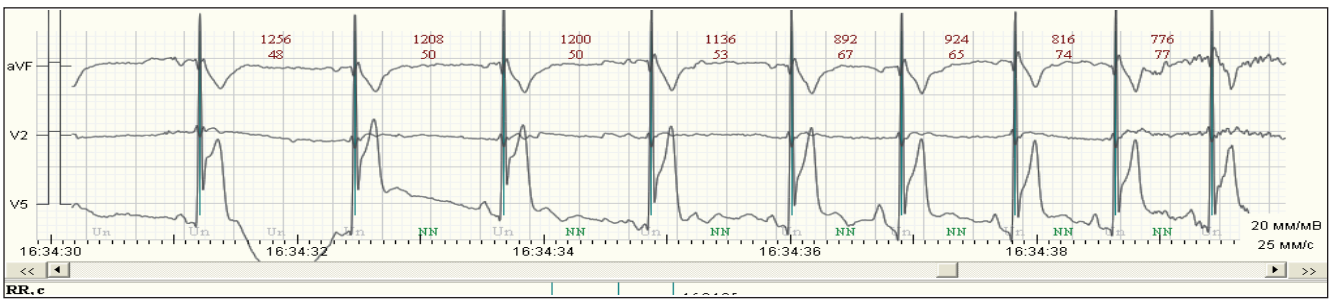

Fig. 11 – ECG phenomena of patient Y.: The deviation of the ST segment of ECG after defibrillation shocks (4:34:34 p.m.).

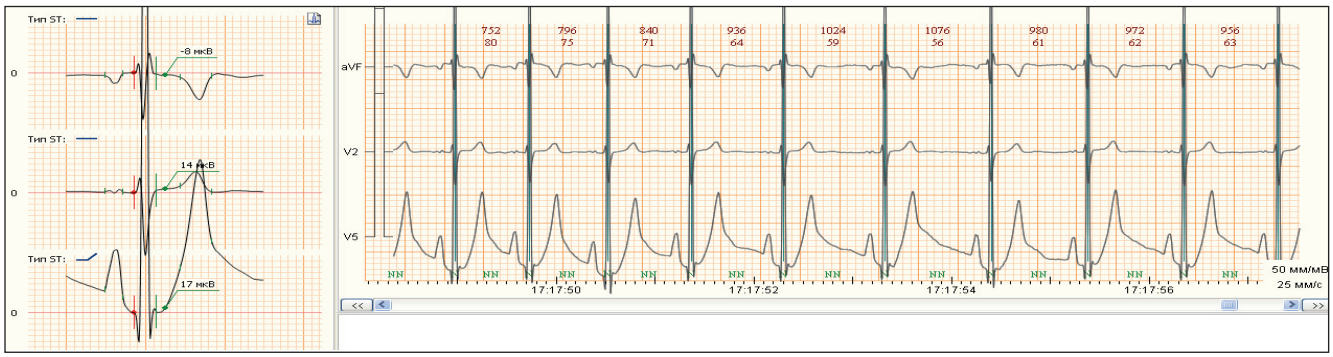

Fig. 12 – ECG phenomena of patient Y.: Sinus arrhythmia (5:17:52 p.m.).

Thanks to the timely provision of qualified medical assistance, the patient was saved. After analyzing the dynamics of all electrical phenomena and the clinical and anamnestic data of the patient, several key elements can be distinguished. First, there are many predictors of the SCD emergence.[7,10] Thus, it has long been known that there are several crucial factors in the pathogenesis of VF at once: passing myocardial ischemia and the phenomenon of reperfusion, changes in vegetative effects.[6,4,13]

Discussion

The main difficulty in the fight against SCD is that it is very rare to save patients who have had SCD: in the United States, this rate is about 5%, and the world average is less than 1%.[17,18] There are many reasons for such failures, the main one being that the provision of assistance (resuscitation measures) must be done within a few minutes, since the effect of the treatment decreases in the future,[19] while it should be taken into account that approximately 40% of patients suffer from SCD at home, in 80% of cases SCD occurs when they sleep or in the absence of witnesses, and if there are witnesses, then they, as a rule, most ordinary people do not have the necessary means or skills of resuscitation.[3,20] The vast majority of patients who die from SCD have organic pathology, but an autopsy study of 270 SCD victims showed that among 5% of SCD patients, it occurred with a "normal heart".[20] An underestimation of the prevalence of SCD is that the vast majority of SCD cases occur in the general population, in which the incidence is lowest, and conversely, a small number of SCD cases occurs in patients with known comorbidities with acute coronary syndrome, ventricular tachycardia, or reduced of the left ventricle ejection fraction (LVEF) — who are neither hypersensitive nor specific for predicting SCD. This is reflected in the fact that more than 70% of SCDs in the general population occur in patients with mildly reduced or normal LVEF.[2,3] Therefore, within the framework of the primary prevention of SCD, predictors of this pathology should be identified not only among people who have already experienced cardiovascular events but among, as we say, "practically healthy people".[3]

Conclusions

The problem of forecasting SCD becomes of primary importance and the presented case and others demonstrated it.[6,19,21] Complex cardiac arrhythmias combined with changes in the autonomic regulation of the cardiovascular system are early predictors of sudden cardiac death.[5,8,22] These facts and the presented clinical case point to difficulties in diagnosing cardiovascular pathology.[19] However, early use of a number of clinically available non-invasive electrophysiological examinations will allow early diagnosis of risk factors and prevention of SCD.[5,6,11] Here it is appropriate to repeat the well-known phrase: "There are no healthy people, there are poorly examined ones." These data indicate both the prospects for the use of mitotrope (phosphocreatine) in the treatment of patients with cardiac arrhythmias on the background of myocardial ischemia and the need for further research to determine its place among other cardiac pharmacological drugs.[14,22–26] Considering the above, it becomes clear that the main way to reduce losses from SCD is primary prevention.[3]

Conflict of interest
None declared.

Funding body
None.

Ethical statement
Authors state that the research was conducted according to ethical standards.

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