INTRODUCTION

AF is the most common supraventricular tachyarrhythmia, in which the presence of foci of uncoordinated electrical activity in the atria, as well as disruption of the sequence and pulse propagation through the myocardium lead to a decrease in the contractility of the heart [1].

AF is the most common form of heart rhythm disturbance. This type of arrhythmia occurs in 1–2 % of the entire adult population and predominates in older men. In young people under the age of 25 almost does not occur, with age, its frequency increases from 0.5 % at the age of 40–50 years and to 5–15 % in 80-year-olds [1–3].

Over the past two decades, there has been an increase in the prevalence of AF among the urban population 6-fold, and 3-fold – in the rural population. The appearance of AF increases the overall mortality among men by 50 %, and among women by 90 %, after excluding the influence of age and other factors [1, 4].

For the onset of AF, a trigger mechanism is necessary, and often this mechanism is the foci of automatism located near the pulmonary veins. Cardiovascular diseases (arterial
hypertension, ischemic heart disease, chronic heart failure, hypertrophic cardiomyopathy, etc. including MB) may precede AF and cause it to develop. These diseases lead to myocardial heterogeneity, violation of the electric pulse, dispersion of refractory periods, which causes the formation of the mechanism of re-entry and contribute to the preservation of AF [1–2]. There is also an idiopathic AF, the cause of which is not established [1].

OBJECTIVE

To show the features of the management of a young patient with AF in combination with the MB. MB is a congenital anomaly of the development of the coronary arteries, in which part of the artery passes in the thickness of the myocardium and can be squashed during its operation [5].

Types of bridges: superficial (short (3–5 mm), long (30–40 mm)) and deep (penetration thickness up to 1 cm) [6]. The incidence of MB can vary from 5 % to 87 %. MB, as a rule, benign pathology of vascular development, which in 0,5–4,9 % of cases is of clinical importance and can be the cause of angina, myocardial infarction, ventricular tachycardia, AF, and sudden death [7].

MATERIALS AND METHODS

Clinical case. A man is 26 years old, he complains of periodic interruptions in the work of the heart, palpitation without a clear connection with the provoking factor more often after playing sports; dizziness; general weakness. There are complaints from other bodies and systems.

Anamnesis of the disease. The first appearance of the AF was during the study in February 2013. The patient was hospitalized in the city clinical hospital №27, the rhythm was restored by amiodarone. After that, he occasionally took metoprolol, while observing short-term episodes of uneven rhythm up to 10 seconds with physical activity. There was an episode of AF during the sport. Man was hospitalized with repeated paroxysm in the hospital, where the sinus rhythm was restored by amiodarone in 2015.

The next relapse of AF was after drinking alcohol in May 2016. The attack was stopped by amiodarone, the consultation of the cardiosurgeon-arrhythmologist was recommended. He was hospitalized in KhNION in May 2016, where on May 27, 2016 the patient underwent radiofrequency ablation (RFA) with the isolation of pulmonary veins, linear ablation on the roof of the LP.

The patient asked for a consultation at the Department of Internal Medicine of V. N. Karazin Kharkov National University with complaints about periodic interruptions in the work of the heart, palpitation without a clear connection with the provoking factor; dizziness; general weakness in 10 April 2017.

Anamnesis of life. Living conditions are satisfactory. He is working as a system administrator. In childhood, he notes varicella, colds. Chronic diseases are denied. Tuberculosis, viral hepatitis, diabetes, mental and venereal diseases are denied. Injuries and other operations are denied. The presence of AF in the mother. The allergic anamnesis is not burdened. Bad habits are not abusing alcohol and are not taking drugs.

Objective status. The general condition is relatively satisfactory, the consciousness is clear, the position is active. Asthenic physique, height – 192 cm, weight – 80 kg, BMI – 21.7 kg/m². Skin covers and visible mucous membranes are clean, pale pink, there is no cyanosis. Lymph nodes are not enlarged. Thyroid gland is not visually determined, clearly not palpable, is painless. Musculoskeletal system without features. Peripheral edema are absent. Respiratory system: above the lungs percussionally pulmonary sound, vesicular breathing. The HDR is 23 beats per minute.

Cardiovascular system: the boundaries of relative cardiac dullness are not biased. Cardiac activity is rhythmic, tones are muffled, heart rate is 95 beats per minute, BP on both hands is 110/70 mm Hg.

The abdomen is of regular shape, not enlarged. Superficial palpation is painless, the symptom of irritation of the peritoneum is negative. The liver at the edge of the costal arch is painless during the palpation, the edge is smooth. The spleen is not palpable. The symptom of «effleurage» is negative on both sides.

RESULTS AND DISCUSSION

Clinical blood test. (11.04.17) Hg – 142 g/l, RBC – 4.77* 10¹²/l, WBC. – 4.1* 10⁹/L, ESR – 6 mm/hour, LYMPH. – 20 %, MONO. – 3 %.

Clinical analysis of urine. (11.04.17) Specific weight-1020, protein is absent, glucose
is absent, WBC – 0 in sp., L – 2 in p., transitional epithelium is absent.

Biochemical analysis of blood (11.04.17)
- Hormones of the thyroid gland (17.02.13)
  - TTG – 2,1mkMed/ml, T4 St. – 16,8pmol/l.
- Lipidogram (11.04.17): total blood cholesterol is 5.8 mmol/l, HDL is 1.61 mmol/L, LDL is 3.76 mmol/l, TG is 1 mmol/l, KA – 2.06
- Coagulogram (11.04.17) fibrinogen is 2.66, fibrin is 12, prothromb. ind. – 82 %.


Echocardiography (14.04.17): Myocardial noncompactness at the top of the left ventricle, the chambers of the heart are not enlarged, violations of LV wall kinetics have not been revealed.

Multidector CT coronary artery angiography (04/04/2015). Left main coronary artery (LM): atherosclerotic plaques and visible narrowing of the lumen are not revealed. Left atrial descending artery (LAD): At the level of the middle and distal part, the artery is closely attached, and also passes through the myocardium of the left ventricle, at a shallow depth, for 65 mm (variant of the myocardial bridge). Left stroke artery (LSx): Relatively significant narrowing of the lumen of the artery was not detected. Intermediate branch of left coronary artery (RI): Contrasted enough, significant narrowing of the lumen is not determined.

Right Coronary Artery (RCA): Dominance of the right coronary artery. The artery and its branches are contrasted sufficiently without apparent constriction. There were no destructive changes in the bones at the investigated level. Thinning of a compact layer of the myocardium with a thickening of the noncompact layer in the region of the apex and posterior wall, in close proximity to the apex

Daily monitoring of ECG (11/04/2017): monitored ECG was carried out for 20 hours 57 minutes. The average heart rate in the daytime is 83 beats/min, the average during the night sleep is 70 beats / min. The variability of the rhythm is moderately reduced. A total of 1215 (max 212 from 21:00 to 22:00), 9 paired, 2 runs of unstable supraventricular tachycardia, parasystole-2, monomorphic were identified. Deviations of the ST segment are not fixed.

HRV: The total power of the HRV spectrum is low (TP: 340 ms2).

The level and ratio of autonomic influences in cardiac rhythm modulation (VLF: 178ms2, LF: 95 ms2, HF: 60 ms2) indicate a predominance of humoral metabolic regulation.

Differential diagnosis of AF. The reasons for the development of AF in this patient may be several factors:
1) The presence of MB contributes to ischemic damage to the myocardium, which leads to its structural change and disruption of the normal structure of the tissue.
2) Noncompactness of the myocardium, which leads to inhomogeneity of the myocardium and impaired conduction of the pulse along it.
3) Sympathicotonia and regular physical activity can cause the formation of foci of abnormal electrical activity due to shortening of the action potential and refractory period.
4) Drinking alcohol leads to an increase in the tone of the sympathetic nervous system.
5) Burdened heredity – the presence of AF in the patient's mother could contribute to its occurrence in our patient.

In this way, it is not possible to single out one single cause of AF, and hence the patient's treatment should be comprehensive and directed to all causes of the onset of the disease.

CONCLUSIONS
Clinical diagnosis. Main: Myocardial bridge of the LAD. Persistent form of atrial fibrillation, tachysystolic form. Radiofrequency ablation of the arrhythmogenic focus (isolation of pulmonary veins, linear ablation on the roof of the LP in 2016). Frequent supraventricular extrasystolic arrhythmia. Running unstable supraventricular tachycardia. CH 0 tbsp.

Treatment plan:
1) Clinical follow-up at a cardiologist, neuropathologist, endocrinologist.
2) Control daily monitoring after 2 months.
3) Motor mode with moderate dynamic physical loads (increasing the walking distance to 30 km per week).
4) Medication:
  - Bisoprolol 5 mg heart rate control with dose selection,
  - Magnesium, pyridoxine for 1 tablet 3 times a day during 1 month.
PROSPECTS FOR FUTURE STUDIES

In the daily practice of a cardiologist, it is rarely possible to meet patients younger than 30 with a persistent AF. In this clinical case, the features of the course and methods of diagnosis of AF with a concomitant myocardial bridge in a young patient after radiofrequency ablation are displayed.

REFERENCES