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Nuances of Cardiac Resynchronization Therapy in Patients with Dilated Cardiomyopathy and Atrial Fibrillation (a Clinical Case)

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Dilated cardiomyopathy is a steadily developing disease characterized by progressive chronic heart failure resistant to drug therapy. Cardiac resynchronization therapy significantly improves the prognosis in these patients if they have indications for implantation of resynchronization devices. The article presents a clinical case of successful implantation of a cardiac resynchronization device with defibrillator in a patient suffering from DCM in combination with permanent atrial fibrillation. The nuances of ventricular rate control and the role of the catheter procedure for modifying the atrioventricular junction are discussed.

Keywords: dilated cardiomyopathy; cardiac resynchronization therapy; atrial fibrillation.

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Нюансы сердечной ресинхронизирующей терапии у пациентов с дилатационной кардиомиопатией и фибрилляцией предсердий (клинический случай)

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Дилатационная кардиомиопатия — неуклонно прогрессирующее заболевание, характеризующееся резистентной к медикаментозной терапии прогрессирующей хронической сердечной недостаточностью. Сердечная ресинхронизирующая терапия существенно улучшает прогноз у таких пациентов при наличии у них показания к имплантации ресинхронизирующих устройств. В статье представлен клинический случай успешной имплантации сердечного ресинхронизирующего устройства с функцией кардиоверсии-дефибрилляции пациенту, страдающему ДКМП в сочетании с постоянной формой фибрилляции предсердий. Обсуждены нюансы контроля частоты желудочковых сокращений и роль катетерной процедуры модификации атриовентрикулярного соединения.

Ключевые слова: дилатационная кардиомиопатия; сердечная ресинхронизирующая терапия; фибрилляция предсердий.

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INTRODUCTION

DCM is a primary myocardial damage, which develops as a result of exposure to various genetic and nongenetic factors and is characterized by pronounced dilatation of the heart chambers with a decrease in the systolic function of the ventricles. [1].

Criteria for the diagnosis of DCM:

1. Dilatation of the heart chambers. The diagnostic criterion is an increase in the left ventricular (LV) of size/volume index $> 112\%$ in relation to the mean value for a given age $+ 2$ SD.

2. Decreased LV ejection fraction (EF). EF should be calculated in 2D or 3D using the Simpson method. EF less than 45% is considered a diagnostic criterion [2].

The leading clinical sign of DCM is progressive chronic heart failure (CHF) resistant to drug therapy. In the natural course of the disease without therapy, the annual mortality of patients with DCM is 10–50% [3].

CRT significantly improves the prognosis in patients with DCM having indications for implantation of resynchronizing devices [4–6]. The presence of a sinus rhythm in the patient makes it possible to synchronize the work of all chambers of the heart. In patients with AF, it is impossible to synchronize the work of the atria and ventricles. CRT is recommended for such patients only in the case of providing close to 100% biventricular stimulation (using medication or catheter procedures) [7, 8].

MATERIALS AND METHODS

Case histories, discharge epicrisis, results of instrumental examinations of patient B. for the period 1918–2021.

PURPOSE OF THE STUDY

Show the features of management of patients with DCM in combination with AF after implantation of a cardiac resynchronization device.

RESULTS AND ITS DISCUSSION

Patient B., born 05.09.1947. He considers himself ill since September 2018, when for the first time, against the background of the first episode of AF in his life, clinical signs of CHF appeared. The patient was hospitalized. Examination revealed a decrease in EF to 36%, complete left bundle branch block according to ECG with a QRS complex width of 160 ms. Sinus rhythm was restored by electro-pulse therapy. Ischemic cardiomyopathy was diagnosed. The following therapy was prescribed: metoprolol with succinate, perindopril, eplerenone, torasemide, apixaban, atorvastatin. Coronary angiography (CAG) is recommended routinely to verify the diagnosis.

In April 2019, in presence of ongoing therapy, an episode of stable hemodynamically significant ventricular tachycardia (VT), accompanied by loss of consciousness, was recorded for the first time.

In May 2019, CAG and ventriculography were performed. Coronary arteries showed no pathology. EF was visually estimated at about 40%. After CAG, the diagnosis was changed to DCM.

The ongoing optimal drug therapy did not lead to an improvement in the patient's condition. Hemodynamically significant VT, complicated by syncope, recurred. AF became permanent.

In view of repeated VT and indications for implantation of a cardiac resynchronization device, a 3-chamber pacemaker with a cardioversion-defibrillation function (CRT-D) was implanted in August 2019. After CRT-D implantation, adequate control of atrioventricular (AV) conduction was ensured by medication with a dose of metoprolol succinate titrated up to 200 mg per day; full biventricular stimulation, approaching 100%, took place. Against this background, 4 weeks after CRT-D implantation, not only the patient's condition improved, but also the EF normalized (EF 65%). In September 2019, due to several triggers of an implanted cardioverter-defibrillator for persistent VT, metoprolol was replaced with sotalol at a daily dose of 160 mg.

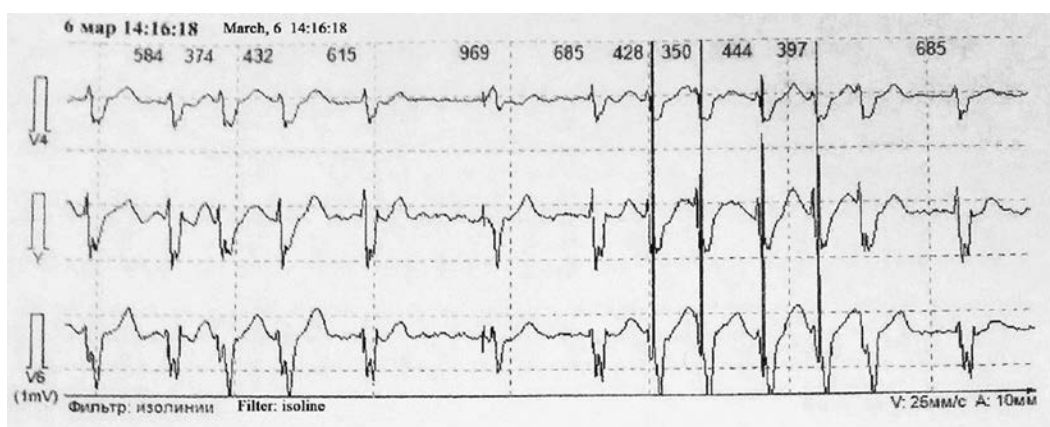


Fig. 1. Fragment of ECG monitoring after CRT-D implantation, explanation in the text.

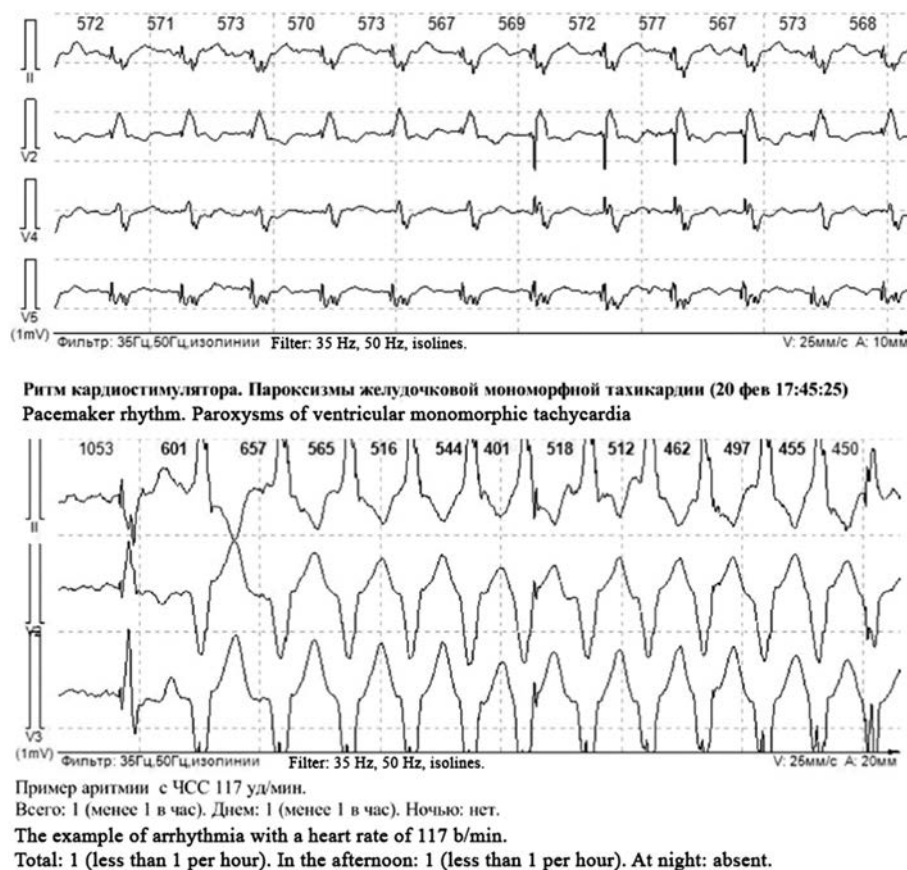


Fig. 2. Two fragments of 24-hour ECG monitoring after destruction of the AV junction. Explanation in the text.

Unfortunately, sotalolol in a daily dose of 160 mg was unable to adequately control AV conduction. The percentage of biventricular stimulation decreased to 70%. With underlying critically low values of biventricular stimulation in February 2020, dyspnea reappeared and began to increase.

Hospitalized with decompensated CHF. When programming the pacemaker, a low percentage of biventricular stimulation due to tachysystolic AF and inadequate drug control of AV conduction was observed.

According to 24-hour ECG monitoring (from March 06, 2020): in presence of tachysystolic AF (heart rate up to 150 beats/min.), along with episodes of full-fledged biventricular pacing (Fig. 1), multiple loss of biventricular ventricular pacing with its replacement with triggered LV stimulation in the performed supraventricular and ectopic ventricular complexes was observed. From echocardiography (March 06, 2020) a decrease in EF to 41.5% was revealed.

The following therapy was prescribed: metoprolol 150 mg per day, dabigatran etexilate 150 mg 2 times a day, spironolactone 25 mg per day. Due to hypotension, further titration of the metoprolol dose was not possible. For the same reason, inhibitors of the renin-angiotensin system were not prescribed. There were no signs of fluid retention and therefore no diuretics were prescribed. The therapy did not improve the clinical condition. Dyspnea persisted during household physical activity.

Indications for the destruction of the AV connection were established due to the impossibility of drug control of AV conduction. The procedure was planned and performed on March 20, 2020. After the destruction of the AV junction, which provided 100% biventricular stimulation, the clinical situation improved, the dyspnea disappeared.

According to echocardiography from February 20, 2021 there was a tendency to EF improvement (EF 42.6%).

According to the daily ECG monitoring from February 20, 2021: permanent AF; 3rd degree AV block; pacemaker rhythm in biventricular stimulation mode; VT episode without clinical manifestations, to which CRT-D did not respond (probably due to a lower (than the programmed) frequency rhythm disturbance was not perceived as VT) (Fig. 2).

CONCLUSIONS

The case presented by us illustrates the efficacy of CRT in patients with DCM in combination with AF under the condition of full, approaching 100% biventricular stimulation. Patients need rigid medication (or catheter procedures if medication is ineffective) control of AV conduction, since only suppression of the conduction of supraventricular complexes to the ventricles can provide full biventricular stimulation.

ADDITIONAL INFORMATION

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