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Optimal upgrade of dual-chamber pacing system in patient with permanent atrial fibrillation

Optymalna rozbudowa dwujamowego układu stymulującego u pacjentki z utrwalonym migotaniem przedsionków

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Abstract

In patients with sick sinus syndrome, and previously implanted dual-chamber pacemaker, the occurrence of atrial fibrillation finally assessed as permanent is most commonly regarded as a natural course of the disease. The inappropriate heart rate control and right ventricular pacing can both negatively influence left ventricle mechanical performance leading to cardiomyopathy and heart failure.

It was presented the case of a 72-year-old woman with permanent atrial fibrillation with narrow QRS complex, after dual-chamber pacemaker implantation, admitted to our department with signs and symptoms of acute decompensated congestive heart failure. The modification of pharmacotherapy allowed to compensate heart failure, however with only small influence on structural remodelling and patient's symptoms. The successful upgrade of the pacing system to the direct His-bundle pacing enabled the restoration of proper function of the heart and full symptoms relief. The direct His-bundle pacing is an approach of choice in such a group of patients.

Key words: atrial fibrillation, congestive heart failure, direct His-bundle pacing

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Introduction

In patients with sick sinus syndrome, and previously implanted dual-chamber pacemaker, the occurrence of atrial fibrillation finally assessed as permanent is most commonly regarded as a natural course of the disease [1]. Many physicians, including cardiologists, consider the presence of a cardiac pacemaker set to right ventricular pacing just as a convenient, additional therapeutic option facilitating the heart rhythm regularization by a proper ventricular pacing rate, and an appropriate beta-adrenolytic or other agents dosing to decrease the physiologic atrioventricular conduction. This approach can be of certain use probably in the majority of patients with a normal heart, however, in some cases, in particular with the preexisting structural heart disease, the episodes of tachyarrhythmia resulting from an inadequate beta-blockade, and/or a higher percentage of right ventricular pacing can deleteriously influence the left ventricle performance, leading to cardiac remodeling, and ultimately to heart failure.

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The atrial fibrillation, and the right ventricular pacing have both a well-known negative influence on the left ventricle mechanical performance [2]. Both circumstances decrease the cardiac output contributing, by different mechanisms, to the development of cardiomyopathy whose signs and symptoms could be more pronounced in a previously existing damage or in genetically susceptible individuals [3]. Paradoxically, the possibility of ventricular pacing can negatively influence the beta-adrenolytic dosing, decreasing the deleterious right ventricular pacing on the one hand, but leading to a higher mean heart rate, and especially to a much higher temporary peak hear rate i.e. during exertion, on the other hand [4]. Probably the optimal mode of treatment should include the higher dosage of beta-adrenolytics and an upgrade of the pacing system to the biventricular one, but in patients with initial narrow QRS complexes it is not univocally recommended. In this group of patients the optimal treatment method is to upgrade the pacing system to the direct His-bundle pacing-based one [5], which is presented in this case report.

Case description

The 72-year-old woman with the dual-chamber pacemaker implanted because of a sinus node disease (now in ventricular pacing mode — VVI), with a permanent atrial fibrillation was admitted to the cardiology ward with the diagnosis of a congestive heart failure. The initial clinical assessment revealed irregular, elevated heart rate, bilateral ankle edema, pulmonary congestion, shortness of breath and a decreased exercise capacity. The blood pressure was 170/90 mm Hg, heart rate 90–130 bpm. The patient denied the discontinuation of any drug and indicated the increase of the severity of palpitation including the nocturnal period and weakness by exertion lasting a couple of weeks. The basic laboratory test revealed abnormalities in the elevated concentration of N-terminal B-type natriuretic propeptide (NT-proBNP) by 8124 pg/mL, slightly elevated creatinine concentration by 1.2 mg/dL and potassium levels of 3.4–3.8 mEq/L. The standard echocardiography showed an enlarged left heart, with the left ventricle diastolic diameter of 71 mm, a decreased ejection fraction of 34%, the left atrium diameter of 58 mm, a moderate mitral regurgitation (jet area 11.4 cm^2) and a large tricuspid regurgitation (the right ventricular systolic pressure of 41 mm Hg). The previous treatment included 50 mg of metoprolol succinate, 0.1 mg of metylodigoxin, 12,5 mg of hydrochlorotiazide, 5 mg of ramipril, warfarin and 10 mg of rosuvastatin recommended during the last visit to a general practitioner.

The patient's status was greatly improved by intravenous furosemide, digoxin loading dose and metoprolol administration. In the discharge letter, ramipril 10 mg, metylodigoxin 0.25 mg, metoprolol succinate 75 mg bis in die (bid) hydrochlorotiazide 25 mg, eplerenon 25 mg, amlodypine 2.5 mg, warfarin and rosuvastatin 10 mg were recommended. In the 4-month follow-up period the patient's status was stable, without obvious decompensation, but with complaints of palpitations and shortness of breath on exertion despite a thorough pharmacological treatment. The follow-up echocardiography revealed LV diameter of 64 mm with an increased ejection fraction up to 45%. The pacemaker, with basic rate at 70 bpm, the sleep period of 8 hours — 60 bpm, indicated 37% of ventricular pacing. The heart rate histogram noted heart rates as high as 170–180 bpm.

After another 2 months of follow-up, without any further clinical or echocardiographic improvement, the decision was made to upgrade the pacing system to the direct His-bundle pacing. The approach consisted of the atrial lead removal by a helix screw-out and a simple mechanical traction, and the His-bundle electrode implantation (Medtronic 3380 Select Secure lead) via the same venous access, using Medtronic SelectSite Model C304 Deflectable Catheter System connected to atrial port, with a concomitant pacemaker replacement (Figure 1).

The selective direct His-bundle pacing was achieved by an acceptable pacing threshold of 1.8 V/0.8 ms. The pacemaker was set to 70 bpm, with the night rate of 60 bpm. The dose of metoprolol succinate was initially increased to 100 mg bid and subsequently to 150 mg bid. The selected electrographic tracings were depicted in Figure 2.

The 4-month follow-up showed a substantial clinical improvement with a complete symptoms resolution. Furthermore, the echocardiography revealed a decrease of the left ventricle diameter to 54 mm, an increase in the ejection fraction to 55–60%, only a mild mitral regurgitation and the right ventricle systolic pressure of 26 mmHg. The pacemaker, which indicated 78% of atrial pacing, was set to 66 bpm, with the night rate of 58 bpm. The His-bundle pacing threshold remained stable.

Discussion

Among the hemodynamic consequences of atrial fibrillation, the decrease of cardiac output affects a minority of patients, being usually caused by a too low ventricular response rate dependent on the decreased atrioventricular conduction. In a majority of patients, the increased heart rate contributes to the maintenance of cardiac output even if the lack of the mechanical atrial function decreases the stroke volume by 15–25% [6]. However, the high heart rate results in some forms of tachycardiomyopathy which was obviously present in our patient. Even if the structural changes are not so prominent the excessive increase in a heart rate on exertion contributes to the left ventricle

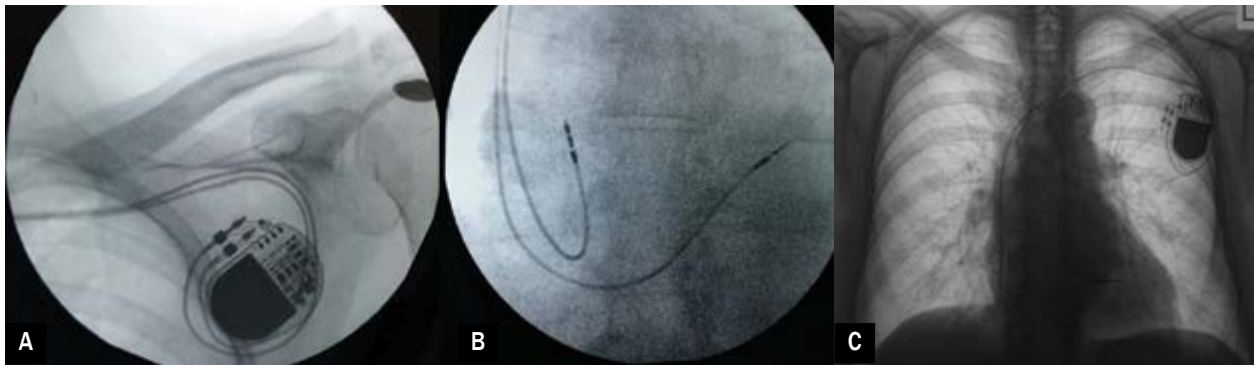

Figure 1A. The fluoroscopy image of left subclavicular region with pacemaker and two pacing leads; **B.** The fluoroscopy image of active--fixation pacing leads located in the right atrial appendage and right ventricular outflow tract; **C.** Chest radiograph during follow-up. Normal lungs, heart and great vessels image with dual leads pacing system. Note the His-bundle pacing lead location (the thinner one) connected to the atrial port

Figure 2A. Atrial fibrillation with two native QRS complexes — duration 104 ms (the first one a pseudo-fusion) — and two paced QRS complexes — duration 184 ms. Selected tracings as described in the figure, paper speed of 50 mm/s; **B.** Atrial fibrillation with two spontaneous QRS complexes and three selective His-bundle paced complexes. Selected tracings as described in the figure, paper speed of 50 mm/s, scale two times lower than in panel A. Note the pacing spike at the peak of each pacing-derived QRS (the effect of safety atrioventricular delay ventricular pacing mode — not possible to switch off in Biotronik pacemakers)

heart failure symptoms in many atrial fibrillation patients, and is known as a heart failure with preserved ejection fraction [2]. We aim at indicating a paradox in patients with an implanted device, being only able to pace the right ventricle: the increase of the beta-adrenolytic dose can of course prevent the heart rate increase but at the same time this increases the percentage of the deleterious right ventricular pacing. This problem was probably the cause of the lack of echocardiographic improvement in our patient before the His-bundle pacing implementation, with persistent left ventricle dilatation up to 60 mm.

The irregularity of the heart rhythm itself during atrial fibrillation has a deleterious impact on the cardiac function. This concept was proved more than twenty years ago by Clark et al. in an excellent experimental study in patients with atrial fibrillation undergoing the atrioventricular node ablation and the pacemaker implantation [7]. The authors concluded that an irregular sequence of RR intervals produces adverse hemodynamic consequences that are independent of the heart rate. In fact, the results showed a specific trend towards worse hemodynamic influence with the heart rate increase, but the main result was indeed related to the rhythm irregularity.

The achievement of the regular heart rate in patients with atrial fibrillation is possible only by cardiac pacing. If the restoration of the sinus rhythm is no longer possible there are still three options of a right ventricular, bi-ventricular and direct His-bundle pacing. The right ventricular pacing can be life-saving but in the long term it can also contribute to the so-called pacing cardiomyopathy in a substantial part of paced patients [8]. The bi-ventricular pacing, better known as cardiac resynchronization therapy, was introduced to correct the left bundle branch block-related dyssynchrony and could of course be considered in the presented patient according to the Guidelines [2]. There are, however, two important drawbacks of this approach. Firstly, in our patient the QRS complex duration is normal, hence even the optimal result of resynchronization could not give the same QRS duration. Secondly, after an initial pharmacotherapy our patient was in New York Heart Association class I/II and the percentage of the right ventricular pacing was not high, which did not fulfill the resynchronization indications.

The successful implementation of the direct His-bundle pacing in our patient met all the needs expected from the optimal pacing. It enabled the appreciated regularization of the heart rate, the preservation of the native QRS complex, and the appropriate increase of beta- adrenolytic dose. All these factors contributed to the normalization of the left ventricular function that can be assessed as a total reversed remodelling. The possibility of a beneficial influence of the direct His-bundle pacing in the permanent atrial fibrillation was shown years ago by Deshmukh et al. [9]. Until recently, the procedure was performed only in selected centers because of the opinion of its complexity. The growing experience and a growing number of operated patients contributed to the increased interest in performing the His-bundle pacing as well as indicated its potential to positively influence the reversed remodelling particularly in sicker patients [10]. In this case we also indicate the need to up-titrate the beta- adrenolytic dose far above the recommended ones. If there is no fear of increased percentage of the right ventricular pacing and, on the other hand, if there is no clear target dose in patients with permanent atrial fibrillation, the clinical assessment and the personal experience can be the final indication.

Last but not least, the performed procedure contributed to the restoration of the normal conditions in the venous system. The redundant atrial electrode was removed, and the atrial channel was connected to the His-bundle electrode.

Conclusions

In the presented case, the upgrade of the dual-chamber pacemaker to the direct His-bundle pacing from the atrial channel successfully contributed to the clinical and structural improvement (reversed remodelling). This approach facilitated an appropriate pharmacological treatment.

Conflict of interest(s)

Authors declared no conflict of interest(s).

Streszczenie

U pacjentów z chorobą węzła zatokowego po implantacji stymulatora serca na stałe wystąpienie migotania przedsionków (AF), uznanego ostatecznie za utrwalone, najczęściej uznaje się za naturalny przebieg choroby. Jednak niewłaściwa kontrola częstości serca w AF, jak również wysoki odsetek komorowej stymulacji mogą negatywnie wpływać na funkcję skurczową lewej komory i obie te przyczyny mogą prowadzić do kardiomiopatii i niewydolności serca.

Zaprezentowano przypadek 72-letniej kobiety z utrwalonym AF z wąskimi zespołami QRS, po przebytej w przeszłości implantacji dwujamowego stymulatora serca z powodu choroby węzła zatokowego, którą przyjęto na oddział z powodu ostrej zdekompensowanej niewydolności serca. Modyfikacja farmakoterapii pozwoliła uzyskać kompensację układu sercowo-naczyniowego, z niewielkim jednak wpływem na remodeling strukturalny serca i objawy podawane przez chorą, w tym głównie uczucie kołatania serca. Rozbudowa układu stymulującego do stymulacji pęczka Hisa z kanału przedsionkowego pozwoliła na odwrócenie niekorzystnej przebudowy serca oraz pełne ustąpienie subiektywnych objawów. Bezpośrednia stymulacja pęczka Hisa powinna być postępowaniem z wyboru w tej grupie pacjentów.

Słowa kluczowe: migotanie przedsionków, zastoinowa niewydolność serca, bezpośrednia stymulacja pęczka Hisa

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