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LONG-TERM OUTCOMES OF CATHETER ABLATION PULMONARY VEINS ON EXAMPLE OF A CLINICAL CASE PATIENT WITH PAROXYSMAL ATRIAL FIBRILLATION

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Catheter ablation of the pulmonary veins is the method of choice for the treatment of patients with symptomatic paroxysmal atrial fibrillation (AF). However, there are may be complications or recurrence of AF paroxysms and as we have described in our clinical case 2 after ablation really important to conclude that ablation does not eliminate drug therapy, but modifies it.

KEY WORDS: paroxysmal atrial fibrillation, catheter ablation, long-term outcomes, autonomic regulation

ВІДСТРОЧЕНІ РЕЗУЛЬТАТИ КАТЕТЕРНОЇ АБЛЯЦІЇ ЛЕГЕНЕВИХ ВЕН НА ПРИКЛАДІ КЛІНІЧНОГО ВИПАДКУ ПАЦІЄНТА З ПАРОКСИЗМАЛЬНОЮ ФОРМОЮ ФІБРИЛЯЦІЇ ПЕРЕДСЕРДЬ

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Катетерна абляція легеневиx вен є методом вибору для лікування пацієнтів із симптоматичною пароксизмальною фібриляцією передсердь (ФП). Проте, можуть бути ускладнення чи рецидиви пароксизмів ФП і, як ми описали у нашому клінічному випадку 2 роки після абляції, дуже важливо зробити висновок про те, що абляція не скасовує медикаментозну терапію, але модифікує її.

КЛЮЧОВІ СЛОВА: пароксизмальна фібриляція передсердь, катетерна абляція, віддалені наслідки, вегетативна регуляція

ОТСРОЧЕННЫЕ РЕЗУЛЬТАТЫ КАТЕТЕРНОЙ АБЛЯЦИИ ЛЕГОЧНЫХ ВЕН НА ПРИМЕРЕ КЛИНИЧЕСКОГО СЛУЧАЯ ПАЦИЕНТА С ПАРОКСИЗМАЛЬНОЙ ФОРМОЙ ФИБРИЛЛЯЦИИ ПРЕДСЕРДИЙ

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Катетерная абляция легочных вен является методом выбора для лечения пациентов с симптоматической пароксизмальной фибрилляцией предсердий (ФП). Тем не менее, могут быть осложнения или рецидивы пароксизмов ФП и, как мы описали в нашем клиническом случае 2 после абляции, очень важно сделать вывод о том, что абляция не отменяет медикаментозную терапию, но модифицирует ее.

КЛЮЧЕВЫЕ СЛОВА: пароксизмальная фибрилляция предсердий, катетерная абляция, отдаленные исходы, вегетативная регуляция

INTRODUCTION

Despite good progress in the management of patients with atrial fibrillation (AF), this arrhythmia remains one of the major causes of

stroke, heart failure, sudden death, and cardiovascular morbidity in the world. Since the initial description of triggers in the pulmonary veins that initiate paroxysmal AF, catheter ablation (CA) of AF has developed from a

specialized, experimental procedure into a common treatment to prevent recurrent AF. As first-line treatment for paroxysmal AF, randomized trials showed only modestly improved rhythm outcome with CA compared to antiarrhythmic drug therapy [1].

Complete pulmonary vein isolation (PVI) on an atrial level is the best documented target for catheter ablation, achievable by point-by-point radiofrequency ablation, linear lesions encircling the pulmonary veins. PVI was initially tested in patients with paroxysmal AF, but appears to be no inferior to more extensive ablation in persistent AF as well.

Antiarrhythmic drug therapy is commonly given for 8–12 weeks after ablation to reduce early recurrences of AF after catheter ablation, supported by a recent controlled trial where amiodarone halved early AF recurrences compared with placebo. Prospective studies have not been done, but a meta-analysis of the available (weak) evidence suggests slightly better prevention of recurrent AF in patients treated with antiarrhythmic drugs after catheter ablation.

Many patients are treated with antiarrhythmic drug therapy after catheter ablation (most often amiodarone or flecainide), and this seems a reasonable option in patients with recurrent AF after ablation [1–2].

The clinical case described below shows the long-term outcomes (2 years) of CA pulmonary veins of the patient with paroxysmal atrial fibrillation, which requires supportive medical therapy.

CLINICAL CASE

The patient S., a woman born in 1950, was admitted to the clinical base of internal medicine department in the Kharkiv city polyclinic #24 in October, 2016 with complaints of dyspnea during ordinary physical activity and absent at rest.

HISTORY OF DISEASE

Since 2000 autoimmune thyroiditis III degree with nodular goiter, euthyroid state; right-sided thyroidectomy and isthmus resection; according patient's report – euthyroidism all the time.

Since 2001 hypertension with max level 220/110 mm Hg, usual BP 150/90 mm Hg on the background of drugs therapy.

In 2010 was paroxysmal tachycardia and palpitations, AF was first diagnosed. Since

2012 diagnosis: Paroxysmal atrial fibrillation, EHRA III. CHA₂DS₂-VASc – 2. HAS-BLED score – 2. Essential arterial hypertension stage II, 3 grade. hypertensive heart (LVH). Heart failure with preserved ejection fraction.

2014 – Catheter ablation of pulmonary veins.

After 3 days of ablation the patient had a paroxysm of AF – an electrical cardioversion was performed, continued to intake prescribed antiarrhythmic treatment for 3 months (betaxolol 10 mg/day, propafenon 300 mg/day). Despite of drug intaking, ones in 3 weeks she had episodes of AF which were being stopped by intaking additional 300 mg of propafenon.

After 3 months paroxysms of AF became more infrequent (once in 3 months) with shorter duration (1–2 hours), stopped after intaking propafenon 300 mg with mild/moderate symptoms of paroxysms of AF.

2015 – Gross hematuria on warfarin (the drug intaking was stopped); since 2015 – takes aspirin for prevention thromboembolic complications.

Following months (over 3) she notes poor control of BP (despite taking hypotension drugs).

After 8 months to the present day of CA she started suffer from paroxysmal tachycardia and heart palpitations with HR 120–130 bpm with mild symptoms, which are not related to physical exercise (mostly at night) 1 time per 2 months, sometimes related to incensement of blood pressure (BP) with duration from 1–2 min to 6 hours and converted to sinus rhythm by taking additional propafenon 300 mg and sometimes procainamide 500 mg.

ANAMNESIS VITAE

1981 – Appendectomy.

1993 – Acute pyelonephritis.

2004 – Radical hysterectomy, iatrogenic menopause.

2007 – Cyst in the right breast was removed.

PHYSICAL EXAMINATION

General condition is satisfactory, consciousness is clear, emotionally stable, optimistic mood. Height = 174 cm, weight = 105 kg, BMI = 34.68 kg/m², waist-to-hip ratio 1,07.

Skin is normal colored, without any scars. Peripheral lymph nodes, the thyroid gland are not palpable in the right side, slightly in the left.

Signs of eyelid retraction, periorbital edema, proptosis are absent.

Respiratory system: pulmonary percussion – resonant sound, auscultation – weakened vesicular breathing, no adventitious sounds.

Cardiovascular system: heart borders extended to the left on 1,5 cm of midclavicular line, HR =78 bpm, regular. Ps= 78 bpm. No pulse deficiency. Auscultation of the heart – heart sounds are muted, accent of the II tone above the aorta. Systolic murmur above the aorta. BP dextral = 150/90 mm Hg, BP sin = 175/100 mm Hg, (on the background of antihypertensive therapy).

Gastrointestinal system: abdomen is soft, painless, symmetrical, no discrepancies of the abdominal muscles. No visible peristalsis. Liver edge is smooth, painless, palpated 1.5 cm below the costal arch. Spleen and pancreas are not palpable. Symmetrical mild shin pitting edema.

REFERRAL DIAGNOSIS

Essential arterial hypertension. Paroxysmal atrial fibrillation. Heart failure. Autoimmune thyroiditis. Systemic atherosclerosis. Obesity.

RESULTS OF LABORATORY AND INSTRUMENTAL DIAGNOSIS

Complete blood count (16/10/2016): normal.

Urinalysis (16/10/2016): normal.

Biochemical analysis (16/10/2016): decreased kidney function (GFR by MDRD 54 ml/min/1.73 m²).

Thyroid-stimulating hormone (TSH) (16/10/16): normal.

Fasting glucose test (16/10/2016): normal.

Blood lipid spectrum (16/10/2016): II a type of dyslipidemia.

Electrocardiography (ECG) 2 years after CA: sinus rhythm, regular, heart rate 78 bpm, signs of left ventricular hypertrophy.

24 h -ambulatory ECG monitoring 2 years after CA: during the monitoring 22 h 38 min was registered sinus rhythm with a mean heart rate 74 bpm (maximum HR 120 pm, at 20:05:15, minimum HR 66 bpm – 16:50:55). Was recorded: single supraventricular premature contractions (total 266); single monomorphic ventricular premature contractions (total 49); short episodes of supraventricular tachyarrhythmia (total 4) with an average heart rate of 160 bpm with max duration for up to 5 seconds. Ischemic changes have not been identified. Circadian index 1.07 (N 1.24–1.44).

Heart rate variability (HRV) 2 years after CA: the character of the rhythmogram and HRV indicates the structure to stabilize the heart rhythm with the transition of its regulation from the reflex autonomic level to a lower humoral-metabolic, are not able to quickly provide homeostasis. Functional heart capabilities are reduced. Condition of a poor adaptation with a sharp decline in the functional capacity of the body.

Echocardiography 2 years after CA: atherosclerosis of aorta and aortic valves mild degree. Moderate dilatation of left atrium. Concentric left ventricle hypertrophy (LV Mass Index 100 g/m²; RWT 0.49). Dyssynergic areas were not identified. Diastolic function – relaxation violation (E/A – 0.8).

RECOMMENDATIONS FOR FURTHER EXAMINATION

Repeat 24h – ECG monitoring in a month.

T4, T3, Anti-TPO.

Biochemical blood test (liver (ALT, AST, AP) and renal function tests (BUN), coagulogram.

Blood electrolytes (K, Na).

Chest X-Ray.

Ultrasound of thyroid gland and abdomen.

Consultation with an endocrinologist.

CLINICAL SYNDROMES

- Atherosclerosis (sclerotic changes of aorta and aortic valve).
- Arterial hypertension.
- Arrhythmias (paroxysmal AF).
- Reduction of circadian index and heart spectrum, as a manifestation of reducing humoral and autonomic regulation with non-dipper HR.
- Heart failure.
- Dyslipidemia.
- Hypertensive heart (LVH, atrial enlargement, diastolic dysfunction).
- Obesity.

CLINICAL DIAGNOSIS

Main:

Condition after CA of pulmonary veins due to paroxysmal AF (25/04/14), with decrease in frequency of paroxysms from ones in 3 weeks to ones per 2 months.

EHRA II b.

CHA2DS2-VASc – 5, HAS-BLED score – 4.

Essential arterial hypertension stage II, 3 grade.

Hypertensive and arrhythmic heart (LVH, dilatation of LA).

Heart failure with preserved ejection fraction II FC, stage B.

Systemic atherosclerosis (atherosclerosis of the aorta and aortic valves, dyslipidemia II a type after Fredrickson).

Very high added total CV risk.

Autoimmune thyroiditis (focal? riedel's? hashimoto's?), condition after right – sided thyroidectomy and isthmus resection (2000), euthyroid.

Deep decline the power of all branches autonomic regulation: non-dipper HR with low degree of TP.

Comorbidity:

CKD 3a: hypertensive nephropathy (eGFR 54 ml/min/1.73 m²).

Obesity I class [3–4].

Non-alcoholic fatty liver disease?

PATIENT'S MEDICAL TREATMENT FOR LAST 6 MONTH

Bisoprolol 5 mg per day.

Propafenon 150 mg 2 times per day (without this drug –recurrence of AF paroxysms); during the paroxysms additionally 300 with/without procainamide 500 mg.

Valsartan 80 mg per day.

Atorvastatin 10 mg (do not intake regularly).

Aspirin 75 mg per day.

OUR RECOMMENDED TREATMENT ACCORDING LAST GUIDELINES

Lifestyle modification

1. DASH diet and regular physical activity lead to intensive weight reduction in addition to the management of other cardiovascular risk factors (in the range of 10–15 kg weight loss achieved), and to fewer AF recurrences and symptoms compared with an approach based on general advice in obese patients with AF [1].

2. Control of compliance to medical recommendations.

Drug treatment

1. B- blocker - CARVEDILOL 12,5 mg 2 times p/day (target HR – 60–65 b/m) under control of ECG.

2. AAD – PROPAFENONE 150 mg 3 times per day under control of ECG; additional 300 mg of propafenon in case of paroxysm of AF [5].

3. ARBs – VALSARTAN 160 mg in the morning.

4. Anticoagulant – RIVAROXABAN 15 mg p/day.

5. Statin-ROSUVASTATIN 20 mg in the evening.

6. *Consulting with other subspecials to change treatment strategy (repeat catheter ablation?)* [1].

PROGNOSIS

Prognosis for life – non-compliance to doctor's appointments – non-satisfactory.

The prognosis for recovery - an unfavorable.

PREVENTION

Secondary prevention of paroxysms of AF include lifestyle modification with weight reduction; good blood pressure control, because uncontrolled high blood pressure enhances the risk of stroke and bleeding events and may lead to recurrent AF; control of fluid balance and check up for decompensation of heart failure; control of compliance to our medical recommendations.

CONCLUSIONS

According to recent studies it has been demonstrated that pulmonary vein CA has favourable outcomes at 6–12 months post-ablation, but there are only few studies with a long-term follow-up and, as we see on our clinical case, after 2 years patient present with current deterioration of AF.

The vast majority of very longstanding paroxysmal/persistent AF patients maintained sinus rhythm at a mean follow-up time of 5 years following CA, associated with a significant improvement in symptom scores and, as we see on our clinical case, after 2 years patient maintained sinus rhythm, but with recurrence paroxysms of AF for last year with mild/moderate of symptom scores [6].

Often this procedure is not a radical solution of the problem, and most patients (as it also was shown on the example of our clinical case) are require adjunctive therapies including antiarrhythmics, DC cardioversions and re-ablation and upstream therapy (antihypertensive drugs and so on) [7].

Also our patient needs correction of the treatment of arterial hypertension and more properly diagnosis (and treatment) of thyroid disorder, and improvement the regulation at all levels - from the daily rhythm of the HR up to

relations in the activity of the vagal activity branches, first of all, interventions in the lifestyle and searching for the optimum time drug administration [8].

Of course, consider the presence of multiple syndromes on presented clinical case, we must not forget about the problem of polypharmacy

and try to avoid it (many studies in ambulatory care define polypharmacy as a medication count of five or more medications, but it is practically impossible to investigate the biochemical compatibility in vivo of more than 4 drugs) [9–10].

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