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Case Report

Lead Dependent Tricuspid Valve Dysfunction Disaster Turned into Success

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Introduction

Control of heart rhythm using Cardiac Implantable Electronic Devices (CIEDs) can be associated with certain complications such as lead perforation, lead fracture, lead dislodgement, Lead-Related Endocarditis (LRIE), venous obstruction and Lead-Dependent Tricuspid Dysfunction (LDTD) [1]. The main mechanism of LDTD is abnormal leaflet coaptation caused by: loop of the lead, leaflet pressure or too intensive lead impingement of the leaflets. Transvenous Lead Extraction (TLE) is the gold standard in the treatment of patients with CIED-related complications [1-3]. We present really rare case significant tricuspid regurgitation with stenosis caused by 30y old, looped, dysfunctional lead, and how to turn complex disaster into clinical success.

Case Report

61-year-old woman with a DDD pacemaker implanted 30y ago due to sinus node disease was admitted to the hospital. Both leads were bipolar passive (Biotronik JP53BP and TIR60BP). She was pacemaker-dependent and underwent three generator replacement procedures (20, 14 and 9 years ago). For several years the patient's symptoms of severe Right Ventricular (RV) failure had been increasing gradually. She was hospitalized several times for this reason, but only during the last hospitalization in our hospital the origin of right ventricular failure was precisely determined. It turned out to be caused by severe lead-related Tricuspid Valve (TV) dysfunction. She was admitted to the cardiac surgery department with symptoms of heart failure in III class NYHA for causal treatment. Echocardiography (transthoracic echocardiography - TTE and Transesophageal Echocardiography - TEE) was performed showing significant tricuspid regurgitation (vena contracta 9mm, Vmax regurgitation 3m/s; large right atrium 24cm^2) due to the lead loop ingrown into the septal leaflet and probably with chordae tendineae. Severe regurgitation coexisted with tricuspid stenosis (transvalvular flow velocity - 1,7 m/s and mean transvalvular pressure gradient - 5mmHg) and with high likelihood of pulmonary hypertension (Figure 1). Additionally deep penetration of the tip of ventricular lead up to pericardium was revealed. Left Ventricular Ejection Fraction (LVEF) was normal (LVEF 62%). She was qualified for a TLE procedure as first step of management of the problem.

A fluoroscopic examination confirmed unnecessary loop of the ventricular lead in the right ventricle. Venography showed the occlusion of subclavian, brachiocephalic and superior cava veins with collateral circulation (Figure 1).

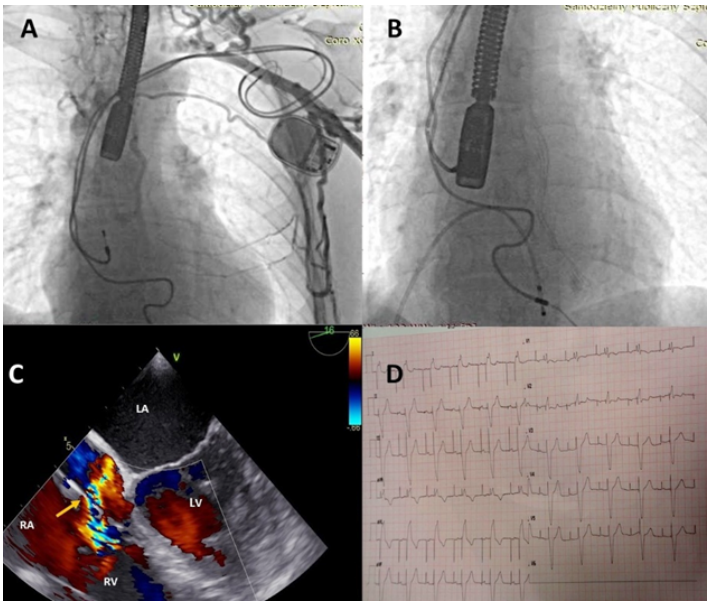

Figure 1: Before TLE. The occlusion of subclavian, brachiocephalic and superior cava veins with collateral circulation – venography (A). Loop in the right ventricle -fluoroscopy (B). 2D TEE color Doppler.Significant tricuspid regurgitation, lead- yellow arrow, left atrium (LA), right atrium (RA), left ventricle (LV), right ventricle (RV). (C). Atrial and ventricular stimulation DDD -ECG (D).

Preoperative risk of lead extraction was estimated. The sum of age of two leads was 60y (30y each lead). We know from practice that the sum of the leads age > 50y means high risk of major TLE complication (cardiac tamponade or TV damage). Additionally if the sum of the leads age exceeds the age of the patient the risk of Major Complications (MC) is very high. Available official calculators confirmed high risk of planned TLE procedure: EROS score 3 (max 3) [4], SAFeTY TLE - risk of MC 13.3% [5]. Patient's risk factors are the following: female gender, young age on the day of first implantation, two very old leads (the risk is cumulative), passive ending of leads difficult for extraction (due to non-isodiametric shape, with tendency of tissue tearing during removal and defragmentation). And the worst of all, the ratio of the age of the leads to the age of the patient was the most alarming [6]. Revealed extensive venous occlusion increased the degree of procedure difficulty but without increasing the risk of MC [7]. Old deep ventricular lead penetration was associated with possible bleeding from the RV wall after TLE. The lead loop pressing the leaflet of TV and ingrown into its structure indicated high risk of damage tricuspid apparatus during lead extraction. Stenosis of the superior vena cava might cause problems with its cannulation in case of the need for extracorporeal circulation.

On the other hand 30y old leads should have been replaced even for prophylactic reason, especially in patient completely pacemaker-dependent - in every moment such old leads may stop working and get patient into trouble [8]. Tricuspid regurgitation is an independent factor of shortened survival [3]. Also drug-resistant RV failure due to TR is an independent factor of shortened survival [3]. Theoretically, in that case there was a little chance to have an improvement in the TV function after lead removal. It works, but for much younger leads, not after 30y of constant interference with TV. After considering the risk of TLE and expected long-term benefits, a decision was made to perform transvenous lead extraction with the intention of implanting new leads into the right atrium and into the coronary sinus for left ventricular pacing (avoiding lead passage through the TV). As the patient was pacemaker-dependent, necessary temporary pacing was provided conventionally via the femoral venous approach. The procedure was performed in a hybrid room by two experienced in lead extraction operators with support of two cardiac surgeons experienced in the treatment of TLE complications, under general anaesthesia and with a perfusion team on standby. Monitoring of TLE procedure using TEE was a standard procedure in the centre.

During TLE procedure we used different polypropylene Byrd dilator unpowered sheaths and mechanical powered rotating sheaths. We encountered difficulties with lead preparation in the subclavian vein, the anonymous vein and the superior vena cava, as well as with a very strong mutual connection of both leads with the calcified connecting tissue scar. Both leads were released alternately step by step, finally with incomplete removing the atrial lead (a few centimetres of its distal fragment remained). The ventricular lead was released as far as the right ventricle. However, 3 cm from its tip, the resistance of the connective tissue stopped the procedure. The patient became unstable – TEE showed 1cm fluid in pericardium and massive tricuspid regurgitation due to break of one of chordae tendineae and tear of leaflets (Figure 2).

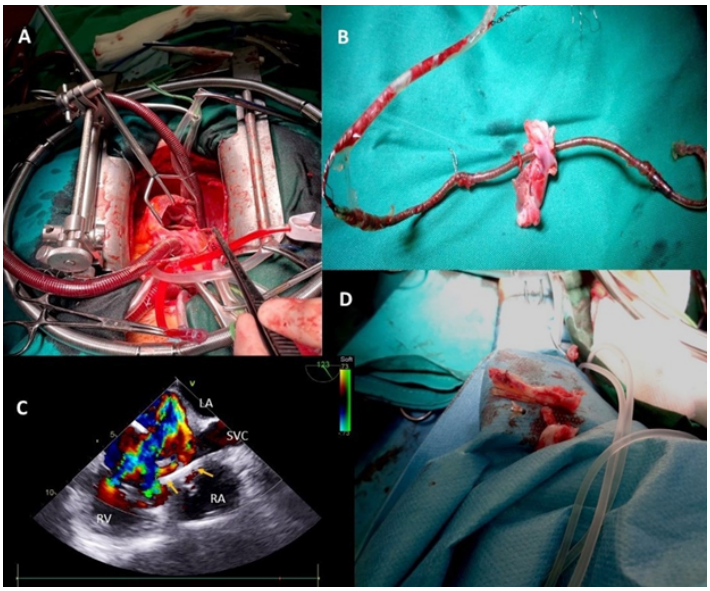

Figure 2: Tricuspid valve replacement, remove ventricular lead. Opened right atrium – remove ventricular lead which passes through tricuspid valve (A). Ventricular lead with a piece of septal and anterior leaflet of tricuspid valve (B). 2D TEE color Doppler. Massive tricuspid regurgitation due to damage to the valve leaflets, removed lead- yellow arrows, left atrium (LA), right atrium (RA), right ventricle (RV), superior vena cava (SVC) (C). The distal fragment of atrial lead and calcified tissues which ware around the leads (D).

Immediate sternotomy was performed - cardiac tamponade was confirmed and managed – approx. 400ml of blood was removed. In that moment the right localisation of bleeding in the ventricular wall was not found, perhaps because of hypotonia. The patient was connected to Cardio-Pulmonary Bypass Pump (CPB) - there was an expected problem with superior vena cava canulation. After stop the heart and opening the right atrium we removed ventricular lead with attached fragment of anterior and septal leaflet (Figure 2B). All leaflets were fibrotic, chordae were shortened – there was no chance for durable valve repair. The artificial biological valve Hancock II 29mm (Medtronic) was implanted in tricuspid orifice. After closing of the right atrium two epicardial bipolar leads were sutured – one on the left atrium and one on the left ventricle. Epicardial leads were connected to a new DDD pacemaker, which was located under the skin below the left costal arch. When the heart started beating properly and almost normal blood pressure was achieved the bleeding from the apex of right ventricle occurred. It was probably the place, where the tip of the right ventricular lead was located and from which was removed during TLE procedure causing tamponade. The perforation was closed by prolen 4-0 with dacron patches. Than the weaning from CPB was uneventful. The patient was recovering properly without any symptoms of right heart failure or other complications.

Discussion

The dramatic and very complicated clinical situation may not appear suddenly, but may be the result of certain diagnostic omissions and postponement of difficult decisions for an indefinite future. Abnormal lead route in the heart should not be ignored. During every routine unit replacement also leads route should be checked. In this patient there were three unit replacements, it means three chances to recognize severe lead loop and to solve this problem before dysfunction of tricuspid valve will occur. Such patients should be referred to lead replacement much earlier to avoid consequences of inappropriate lead route. Echocardiographic examination should be consider performed during each generator replacement – diagnosis of LDTD should result in referral for lead replacement. In every patient with intracardiac leads and symptoms of severe RV failure the relationship between leads and TV should be assessed.

One should remember about the phenomenon of asymptomatic penetration of the electrode tip towards the pericardium, which resulted in a gradual increase in the stimulation threshold in our patient. When removing electrodes perforating the heart wall, one should take into account the possibility of bleeding from the place where the electrode tip was originally. This happens rarely, but also because of this, close cooperation between cardiologists and the cardiac surgery team is necessary. Finally, as shown by the results of echocardiographic examinations and several months of observation, a double catastrophe (preoperative situation, incomplete removal of the leads and increase of dysfunction of tricuspid apparatus) was turned into a full clinical success. It was possible thanks to well-thought-out procedure, full readiness for any failure or complication and organizational preparation of the procedure. Out of which, cooperation between interventional electrocardiology and cardiac surgery seems to be the most important.

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