To pace or not to pace? Or how to pace?

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Abstract

Chronic apical right ventricular pacing may impair left ventricular function and cause heart failure in patients with indication for antibradycardia pacing and normal left ventricular ejection fraction at baseline, through multiple electro-mechanical changes. We describe the case of a patient who needed an upgrade to cardiac resynchronization therapy and developed angina early after single chamber right ventricular pacing and discuss pacing induced cardiomyopathy.

Keywords: *cardiac resynchronization therapy, apical right ventricular pacing, upgrade, heart failure*

Introduction

Right ventricular pacing is preferred in patients with preserved left ventricular function who need antibradycardia pacing due to a lower complication rate and longer service life over biventricular systems. However, in a significant proportion of patients, it may cause pacing induced cardiomyopathy. This has previously been described as a decrease of more de 10% in left ventricular ejection fraction and heart failure symptoms, for which intraventricular and interventricular dyssynchrony is the main cause. In Pacing to Avoid Cardiac Enlargement trial [1] designed to compare biventricular pacing and right ventricular pacing with regards to left ventricular function in patients with standard indications for pacing, 8 of 9 patients in which the left ventricular ejection fraction decreased to less than 45% after 12 months of pacing were in the right ventricular pacing group.

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Case report

A 75 year-old Caucasian female patient, with history of moderate arterial hypertension, elective angioplasty with drug eluting stent on anterior descending artery and permanent single chamber VVI pacemaker implanted for atrial fibrillation with slow ventricular response and symptomatic ventricular pauses 4 months earlier, was admitted in the cardiology department for progressive exertional dyspnea and repeated episodes of typical angina pectoris. At the moment of pacemaker implantation, the documented echocardiography described a non-dilated left ventricle, with mild concentric hypertrophy and preserved ejection fraction (60%), and mild mitral regurgitation. The patient was on treatment with beta-blocker (bisoprolol 2.5 mg), angiotensin converting enzyme inhibitor (ramipril 2.5 mg), loop diuretic (furosemide 40 mg), calcium channel blocker (amlodipine 5 mg) and oral anticoagulant (apixaban 2.5 mg b.i.d).

On physical examination, the patient presented with moderate peripheral edema and bibasilar crackles, heart rate 60 beats-per-minute, blood pressure 150/90 mmHg, systolic murmur grade III/VI in mitral and Botkin-Erb areas. Laboratory tests revealed high serum

urea and creatinin levels (100 mg/dl, 1.9 mg/dl respectively), normal troponin I and CK-MB, and high N-terminal pro-brain natriuretic peptide levels, 22 012 pg/ml. On ECG, there was atrial fibrillation with paced ventricular rhythm with left bundle branch block morphology (Figure 1).

The 2D transthoracic echocardiography revealed a severely impaired systolic function of the left ventricle, with an ejection fraction of 30% (monoplane Simpson) and severe mitral regurgitation (Figure 2), with apical rocking and septal flash.

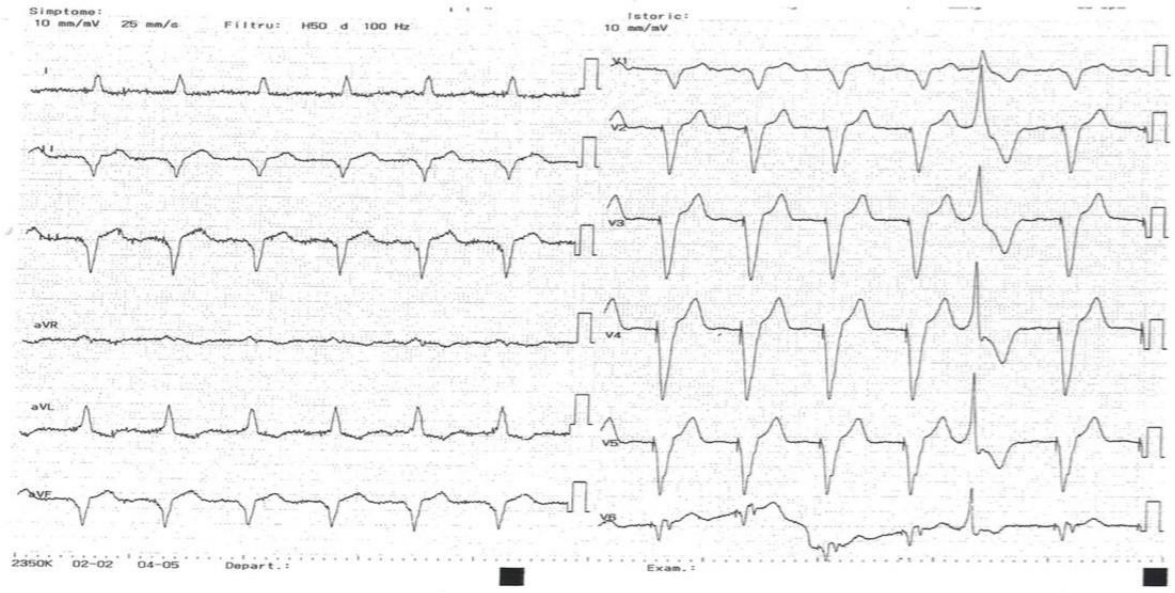

Fig. 1. 12-lead ECG with VVI 60/min pacing. QRS with left bundle branch block morphology. Isolated premature ventricular beats

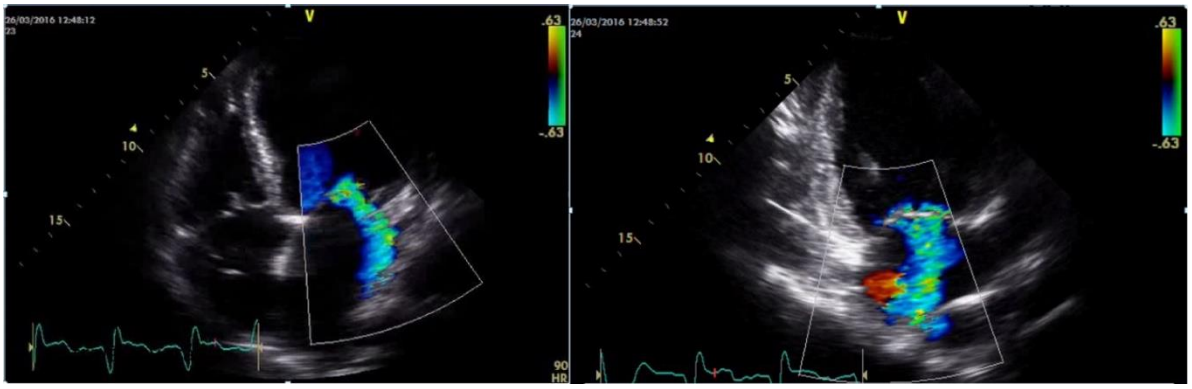

Fig. 2. 2D Transthoracic echocardiography apical 4-chamber and apical 2-chamber views during VVI pacing. Dilated left ventricle. Severe mitral regurgitation

Invasive coronary angiography was unremarkable, without coronary stenosis and patent stent. During hospitalization, the patient presented repeated episodes of asymptomatic non-sustained ventricular tachycardia (Figure 3). The pacemaker interrogation revealed a percentage of ventricular pacing of 76% and 1400 episodes of ventricular heart rate (>180 bpm). The underlying rhythm was atrial fibrillation with slow ventricular response and narrow QRS complex. Due to the discrepancies between the documented echocardiography before pacemaker implant and the current presentation, the pacemaker was set on VVI 30 bpm and the echocardiography was repeated, revealing a mitral regurgitation significantly diminished and no left ventricular asynchrony.

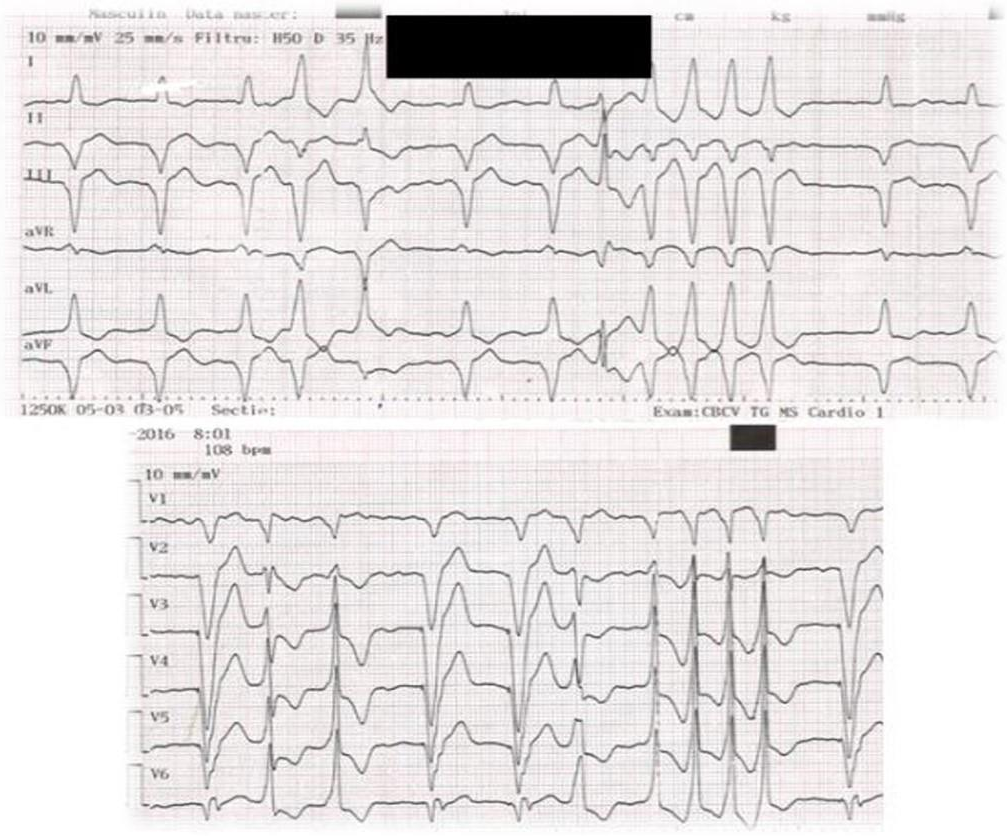

Fig. 3. 12-lead ECG. Frequent premature ventricular beats and episodes of non-sustained ventricular tachycardia originating in the basal septal area (QRS complex negative in lead II, III, aVF, transition in V2)

The medical treatment for heart failure was up-titrated at maximum tolerated doses (carvedilol 12.5 mg b.i.d, spironolactone 25 mg o.d., ramipril 5 mg o.d.) and amiodarone 200 mg b.i.d. was added for ventricular arrhythmia. The pacemaker was upgraded to cardiac resynchronization therapy system with biventricular pacing (Figure 4). A left ventricular lead was implanted in a posterior-lateral vein via coronary sinus, in a distally suboptimal position due to vein caliber.

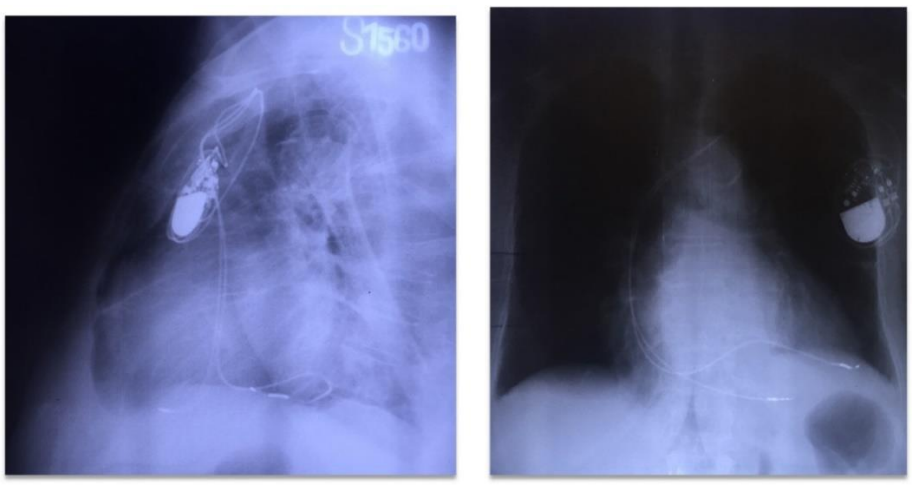

Fig. 4. Thoracic X-ray, left lateral and posterior-anterior views. Dual chamber pacemaker with a lead placed in the right ventricular apex and a lead in a posterior vein via coronary sinus

The echocardiography post-CRT revealed an improved mitral regurgitation and left ventricular function, with an ejection fraction of 34% and no intra or interventricular asynchrony (Figures 5 and 6).

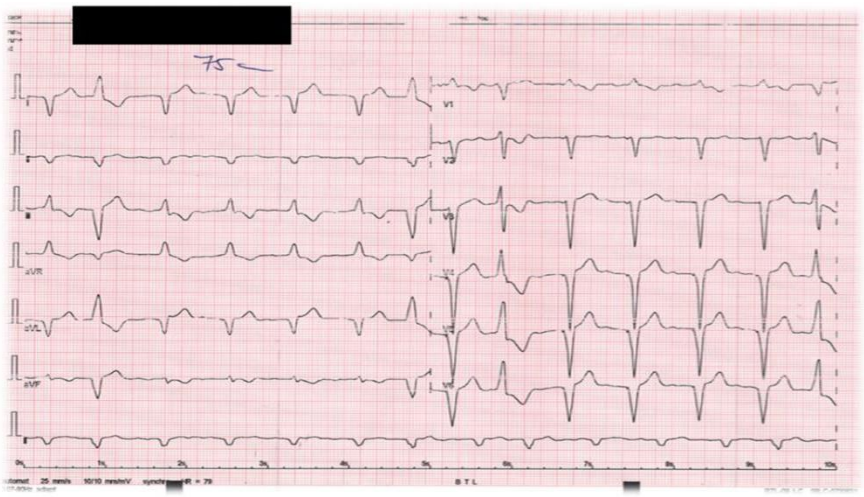

Fig. 5. 12 lead ECG after CRT

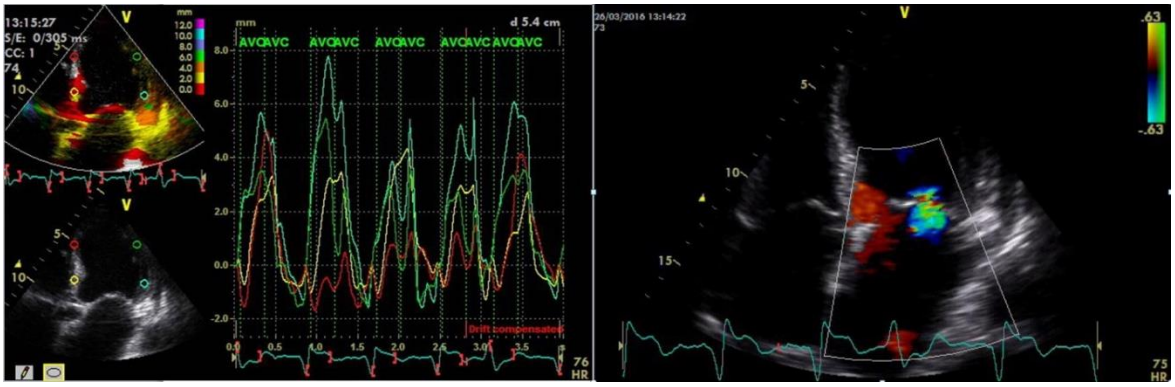

Fig. 6. Transthoracic echocardiography. Tissue Doppler in apical four-chamber view with tissue tracking (left). Apical four-chamber view, mild mitral regurgitation (right)

At discharge, the patient's symptoms were significantly improved, but the patient was lost to follow up as she moved to another country.

Discussions

We presented the case of a patient who developed heart failure secondary to right ventricular pacing, with a severely decreased left ventricular ejection fraction (from 60 to 30% in four months) and severe mitral regurgitation.

Apical right ventricular pacing induces a left bundle branch block-like electrocardiographic morphology, with a slightly different activation pattern from intrinsic left bundle branch block, with early mid-septal and late lateral wall activation [2], with the latest activation of the postero-basal left ventricular region. This leads to interventricular and left intraventricular asynchrony, and reduced cardiac output [3], causing not only worsened heart failure symptoms in patients with impaired left ventricular ejection fraction prior to pacemaker implant [4], but also new onset heart failure in approximately 20% of patients [5], particularly in patients with high percentage of ventricular pacing [6].

As a consequence of left ventricular asynchrony and dysfunction, functional mitral regurgitation is frequent in patients with right ventricular pacing, and is an independent

prognostic factor. Its pathophysiology is complex, including mitral annulus dilatation and geometrical alteration, discrepancies in tethering and closing forces on mitral apparatus leading to impaired leaflet coaptation [7]. As expected, when our patient was not paced, the mitral regurgitation was significantely reduced, most probably due to synchronous left ventricular contraction as the instrinsic QRS complex was narrow.

Left ventricular dyssynchrony also impairs septal myocardial perfusion due to high intramyocardial pressure during early diastole [8], which might explain angina in absence of angiographic coronary arteries stenosis in our patient, with premature ventricular contractions arising in the basal septal area.

In this context, the European Society of Cardiology 2013 Guideline on cardiac pacing and resynchronization therapy recommends as a class I indication, in patients with deterioration of left ventricular systolic function and heart failure symptoms after antibradycardia pacing with a conventional pacemaker, upgrade to a cardiac resynchronization therapy system (CRT) [9]. Upgrade to CRT not only improves the New York Heart Association functional class, but also causes reverse remodeling of the left ventricle with improved ejection fraction, similar to patients who undergo primary CRT [10].

Conclusions

In the case we presented, a patient with atrial fibrillation which had a single chamber VVI pacemaker implanted according to current indications developed heart failure. This supports the necessity for echocardiographic follow-up in this group of patients, with early post-implant evaluation of mitral regurgitation severity as a prognostic factor for progression towards left ventricular dysfunction.

Conflict of interest

The authors declare that they have no competing interests.

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