

Cardiac resynchronization therapy in an elderly patient with left and right ventricular noncompaction

Rafał Dąbrowski, Maciej Sterliński, Alicja Kraska, Joanna Petryka, Bohdan Firek, Hanna Szwed

Institute of Cardiology, Warszawa, Poland

We present a case involving a 67-year-old man with newly diagnosed heart failure and left bundle branch block. Echocardiography revealed the primary cause of heart failure, namely, ventricular noncompaction, confirmed by cardiac magnetic resonance imaging (MRI). Due to ventricular dyssynchrony, a cardiac resynchronization system was implanted. It was a rare etiology of heart failure that required cardiac resynchronization, but the indications had been overlooked until older age.

On presentation to our department, the patient was diagnosed with dilated cardiomyopathy, heart failure symptoms, and New York Heart Association (NYHA) class III. Electrocardiography revealed left bundle branch block (QRS duration, 196 ms; **FIGURE 1A**). No abnormalities were observed on coronary angiography. Echocardiography revealed left ventricular (LV) enlargement (73/63 mm) with global hypokinesis and low ejection fraction (<30%). In the distal parts of the middle inferolateral segments, septum segments, and in the apex, increased muscle trabeculation with the presence of muscle recesses was observed. In the inferolateral segments, the noncompacted-to-compacted myocardium ratio was 2.4 (17/7 mm) and, in the septum, it was 2.3 (16/7 mm). Additionally, an increased dimension of the free wall of the right ventricle with trabeculation (7 mm) was observed (**FIGURE 1B**). Cardiac MRI showed increased LV volume and mass. Hypertrabeculation and intertrabecular recesses were most prominent in the apex and in the lateral wall. The noncompacted-to-compacted myocardium ratio was 2.3. The mass of noncompacted myocardium was 29% of the total muscle mass (**FIGURE 1C**; steady state free precession, i.e., cine). LV noncompaction was diagnosed and cardiac resynchronization therapy (CRT-D) was introduced. The CRT-D system was implanted (electrodes to the left and right ventricles and right

atrium) on February 12, 2010 (**FIGURE 1D**). The last control of CRT-D was performed after 32 months, on October 11, 2012. There was 1 episode of ventricular tachycardia terminated by a cardioverter intervention. The patient's condition stabilized (NYHA class II). He was additionally administered

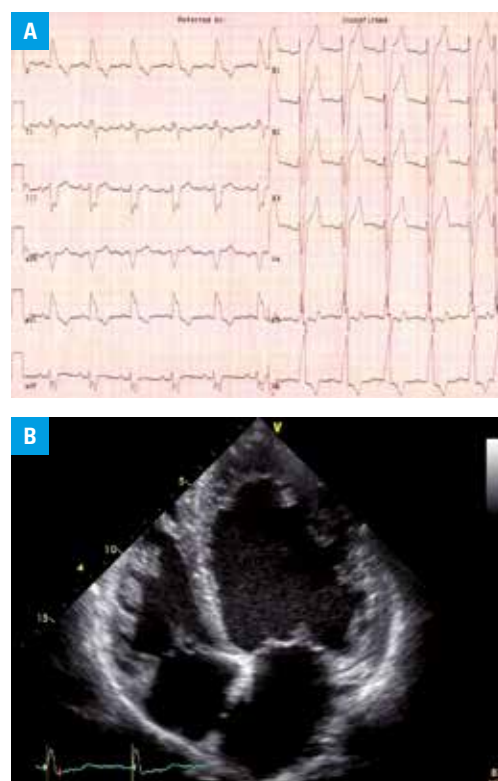
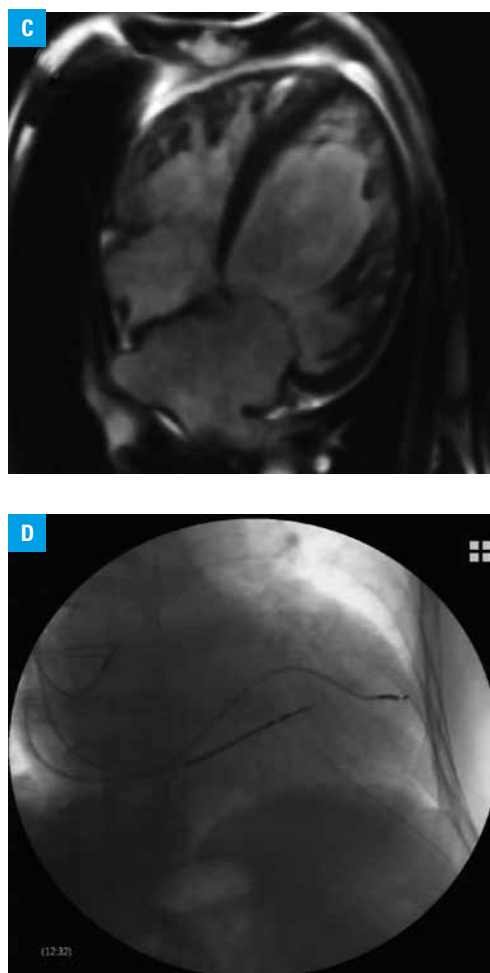


FIGURE 1 A – electrocardiographic examination (sinus rhythm, 72 bpm; PQ, 200 ms; QRS, 196 ms); left bundle branch block; B – echocardiographic 4-chamber view showing increased muscle trabeculation with the presence of muscle recesses in the middle segments of the septum, in the apex, in the distal segments of the lateral wall, and in the free wall of the right ventricle

Correspondence to:
Rafał Dąbrowski, MD, PhD, Instytut
Kardiologii, ul. Spartańska 1,
02-637 Warszawa, Poland;
phone: +48-22-343-40-50,
fax: +48-22-844-95-10,
e-mail: rdabrowski45@gmail.com
Received: April 16, 2013.
Revision accepted: May 8, 2013.
Conflict of interest: none declared.
Pol Arch Med Wewn. 2013;
123 (6): 321-322
Copyright by Medycyna Praktyczna,
Kraków 2013

FIGURE 1 C – cardiac magnetic resonance imaging showing hypertrabeculation and intertrabecular recesses in the apex and lateral wall; **D** – fluoroscopy: right anterior oblique view 35%; final lead locations of the implanted cardiac resynchronization system



ramipril (10 mg), bisoprolol (5 mg), furosemide (40 mg), and spironolactone (12.5 mg).

Despite a generally poor prognosis of patients with LV noncompaction, in our patient, it was diagnosed in older age.¹ Cardiac MRI offers a more detailed examination of myocardial structure compared with echocardiography. A trabeculated LV mass, exceeding 20% of the global LV mass, is highly sensitive and specific for the diagnosis of LV noncompaction.² CRT-D is a valuable therapeutic option in heart failure of various etiologies.³ In a recent prospective study, patients with LV noncompaction had greater LV reverse remodeling after CRT-D than those with dilated cardiomyopathy at 6 months. The chance of achieving optimal CRT-D response and greater LV reverse remodeling was shown to correlate with the size of the noncompaction area.⁴

We presented a rare case of a patient with the late diagnosis (at 67 years) of an unusual cause of heart failure. A follow-up of 32 months has shown good response to CRT-D treatment.

REFERENCES

- 1 Jenni R, Oechslin EN, van der Loo B. Isolated ventricular noncompaction of the myocardium in adults. *Heart*. 2007; 93: 11-15.
- 2 Jacquier A, Thuny F, Jop B, et al. Measurement of trabeculated left ventricular mass using cardiac magnetic resonance imaging in the diagnosis of left ventricular noncompaction. *Eur Heart J*. 2010; 31: 1098-1104.
- 3 Malecka B, Ząbek A, Maziarz A, et al. Influence of heart failure etiology on the effect of upgrading from right ventricular apical to biventricular or bifocal pacing in patients with permanent atrial fibrillation and advanced heart failure. *Pol Arch Med Wewn*. 2012; 122: 89-97.
- 4 Bertini M, Ziacchi M, Biffi M, et al. Effects of cardiac resynchronization therapy on dilated cardiomyopathy with isolated ventricular noncompaction. *Heart*. 2011; 97: 295-300.