DEVICE THERAPY

COMPLEX CASE STUDY

Recurrent Pericarditis after Biventricular ICD Implantation in Patient with Isolated Left Ventricular Non-Compaction

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ABSTRACT. We describe a patient with non-ischemic cardiomyopathy caused by isolated left ventricular non-compaction who developed recurrent pericarditis following a biventricular implantable cardioverter-defibrillator implantation. We review the literature on this rare complication and discuss treatment options.

KEYWORDS. biventricular implantable cardioverter-defibrillator, pericarditis, ventricular non-compaction.

Introduction

Isolated left ventricular non-compaction (ILVNC) is a congenital cardiomyopathy characterized by numerous, prominent trabeculations and deep intertrabecular recesses in the myocardium of the left ventricle. This morphology is believed to result from an arrest in the compaction of embryonic heart muscle in the left ventricle. ILVNC commonly results in depressed systolic function of the left ventricle. Patients with ILVNC are also prone to arrhythmias, as the poor communication between muscle fibers of the excessive trabeculations may result in dysynchrony of the left and right ventricles, which may further decrease systolic function. Because of the decreased systolic function and the high risk of sudden cardiac death as a result of the arrhythmias, the implantation of a biventricular implantable cardioverter-defibrillator (BiV-ICD) has become a viable treatment option for people with ILVNC. We report a unique case of recurrent pericarditis following a BiV-ICD in a patient with non-ischemic cardiomyopathy due to ILVNC.

Case

A 58-year-old woman was diagnosed with non-ischemic cardiomyopathy due to left ventricular non-compaction. Pre-implant, a chest magnetic resonance imaging scan showed evidence of left ventricular non-compaction in the inferolateral and lateral left ventricular wall (Figure 1). She had advanced heart failure despite optimal treatment with heart failure medications. A transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 25%, and her electrocardiogram (ECG) showed sinus rhythm with a left bundle branch block (QRS duration of 170 ms). She underwent implantation of a biventricular ICD with the left ventricular (LV) pacing lead placed in a posterolateral branch of the coronary sinus (CS) (Figure 2). She presented 2 days later with dyspnea and chest pain with breathing. She was noted to have a pericardial rub on examination and a diagnosis of pericarditis was made. TTE showed trace pericardial effusion. The computed tomography (CT) scan showed no evidence of lead perforation and confirmed location of LV pacing lead in area of non-compaction (Figure 1). She had breakthrough symptoms despite treatment with naproxen and colchicine. Finally, she was treated with prednisone for 3 months, which led to relief of symptoms. She had another flare-up of pericarditis 1 year after stopping prednisone, which responded promptly to colchicine treatment for 2 weeks (0.6 mg twice a day). A repeated CT scan again showed no evidence of lead perforation. Following a cardiac resynchronization therapy device (CRT-D), the
patient’s EF improved from 22–25% pre-implant to 45–50% in her most recent echo. Her left ventricular end-systolic volume (LVESV) decreased by 63% (from 85–31 ml/m), and her left ventricular end-diastolic volume (LVEDV) decreased by 46% (from 113–61 ml/m), suggesting that she is a super responder to CRT-D.3

Discussion

Several reports show the benefits of BiV-ICD on patients with left ventricular non-compaction cardiomyopathy (LVNC) through reverse remodeling of the left ventricle and increased EF. It has been shown that patients with dilated cardiomyopathy (DCM) and ILVNC responded better to BiV-ICDs than patients with DCM without ILVNC.3 Biventricular pacing has been shown to improve non-compaction in patients with ILVNC.4 Pericarditis has not been reported as a complication of BiV-ICD implantation in patients with ILVNC.

We present a unique case of recurrent pericarditis following a BiV-ICD implantation in a patient with non-ischemic cardiomyopathy due to ILVNC. The exact mechanism for the development of pericarditis in this case is unclear. Further studies are needed to understand the mechanism of pericarditis following BiV-ICD implantation in patients with ILVNC.

Figure 1: (a) Magnetic resonance imaging before biventricular implantable cardioverter-defibrillator (BiV-ICD) shows non-compaction in the inferolateral and lateral left ventricular wall. (b) Computed tomography (CT) scan post BiV-ICD shows left ventricular lead near the area of non-compaction. There was no evidence of lead perforation by CT scan.

Figure 2: Chest X-ray post biventricular implantable cardioverter-defibrillator implantation shows left ventricular lead in posterolateral position.
case remains unclear, but it is unlikely that right atrial and ventricular leads were the cause of the pericarditis because the CT scan post procedure showed no evidence of lead perforation. The placement of the LV pacing lead in the CS matching the area of non-compaction (infero-lateral and lateral LV wall) may be a possible explanation for the pericarditis. In these areas, there is a thinner, compacted epicardial layer and a much thicker, non-compacted endocardial layer with prominent trabeculations and intertrabecular spaces. Pacing the LV epicardially through the thinner compacted layer could lead to pericarditis. The mere presence of the LV in the CS near the area of non-compaction is also a possible mechanism for pericarditis. A unique feature in our case is the presence of recurrent pericarditis after 1 year that responded very well to colchicine.

In conclusion, it is possible that patients with ILVNC are more prone to develop recurrent pericarditis after a BiV-ICD implant. Though placing the LV lead in the same area as the non-compacted muscle may cause the patient to respond better to CRT-D, there may also be a greater risk of recurrent pericarditis following the procedure.

References