The Korean Journal of Internal Medicine: 21:213-217, 2006

Tachycardiomyopathy Induced by Ventricular Premature Complexes: Complete Recovery after Radiofrequency Catheter Ablation

Kyoung-Hoon Rhee, M.D., Ju-Young Jung, M.D., Kyoung-Suk Rhee, M.D.[2], Hyun-Sook Kim, M.D.[2], Jei-Keon Chae, M.D.[2], Won-Ho Kim, M.D.[2] and Jae-Ki Ko, M.D.[2]

Department of Internal Medicine, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea; Division of Cardiology, Department of Internal Medicine, Chonbuk National University[2] Medicine School, Jeonju, Korea

Ventricular premature complexes (VPCs) are known to be one of the most benign cardiac arrhythmias when they occur in structurally normal hearts. We experienced a 32-year old man who presented with dyspnea, palpitations and very frequent VPCs (31% of the total heart beats). Echocardiography revealed a dilated left ventricle (LV 66 mm at end-diastole and 57 mm at end-systole) and a decreased ejection fraction (34%). Very frequent VPCs had been detected 10 years previously and he underwent a failed radiofrequency catheter ablation (RFCA) procedure at that time. The patient had been treated with heart failure medications including betablockers, ACE inhibitors and spironolactone for the two most recent years. Six months after we eliminated these VPCs with a second RFCA procedure, the heart returned to normal function and size. Long standing and very frequent VPCs could be the cause of left ventricular dysfunction in a subset of patients who suffer with dilated cardiomyopathy, and RFCA should be the choice of therapy for these patients.

Key Words : Ventricular premature complexes, Cardiomyopathies, Catheter ablation

INTRODUCTION

Ventricular premature complexes (VPCs) are the most common arrhythmias observed in the patients who are without structural heart disease. It has been recently reported that frequent VPCs could evoke left ventricular (LV) dilation, and this might be reversed by suppression of the VPCs[1-3]. We experienced a patient with severe LV dysfunction, the so-called tachycardiomyopathy, that was induced by very frequent and longstanding VPCs. The LV dilation and dysfunction were completely reversed with performing radiofrequency catheter ablation for eliminating the VPCs, which originated from the right ventricular outflow tract (RVOT).

CASE REPORT

A 32-year-old male patient visited the emergency room and presented with resting dyspnea and palpitations. He had suffered from intermittent palpitations and dyspnea (class II) for more than 10 years. He had undergone a radiofrequency catheter ablation procedure 10 years ago for these frequent VPCs without success at another hospital. He had received intensive heart failure medications including beta-blockers, angiotensin-converting enzyme inhibitors and spironolactone from a local hospital during the last two years.

A physical examination revealed jugular vein engorgement and slightly rapid and irregular heart sounds. His vital signs were as follows: a blood pressure of 110/70 mmHg, a pulse

• Received : December 27, 2005
• Accepted : February 8, 2006
• Correspondence to : Kyoung-Suk Rhee, M.D., Division of Cardiology, Department of Internal Medicine, Chonbuk National University, 634-18 Keumam 2 Dong, Duckjin Ku, Jeonju 561-712, Korea Tel : 82-63-250-1389, Fax : 82-63-250-1680, E-mail : ksee@chonbuk.ac.kr

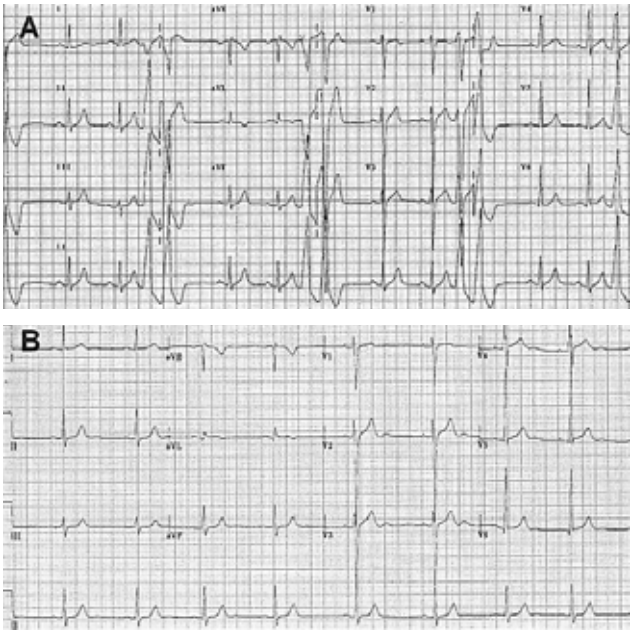

Figure 1. The electrocardiograms taken before (A) and after (B) the catheter ablation. A. The frequent pairs of ventricular premature complexes (VPCs) with a negative QRS deflection in lead V1, the QRS transition in lead V4 and the positive deflections in leads II, III and aVF suggest that the right ventricular outflow tract is the agent provocateur. B. No VPC is observed after performing radiofrequency catheter ablation.

rate of 88 beats/min and a respiration rate of 21 breaths/min. The standard 12-lead electrocardiography (ECG) revealed frequent VPCs. The right ventricular outflow tract was suspected as being the origin of this condition due to the negative deflection of the VPCs in lead V1, the positive deflection in leads II, III and aVF, and the QRS transition in lead V4 (Figure 1A). The chest X-ray revealed cardiomegaly and increased broncho-vascular markings. The echocardiography demonstrated dilatation of the left ventricle (LV) and a decreased LV contractile function (LV end-diastolic dimension: 66 mm, LV end-diastolic volume: 211 mL and an ejection fraction: 34%) (Figure 2A). A 24 hour ambulatory ECG showed very frequent VPCs; there were 22,256 isolated VPCs and 16,081 couplets out of 123,139 total heart beats (31%) during 22 hours.

Electrophysiological Study and Radiofrequency Catheter Ablation

An electrophysiological study and radiofrequency catheter ablation were performed after obtaining an informed written consent on the following day after hospital admission. A 6 Fr. Quadripolar electrode catheter was positioned in the right ventricular (RV) apex. A 7 Fr. deflectable quadripolar ablation catheter (Boston Scientific EP Technologies, Natick, Massachusetts) with a 4-mm-tip electrode was introduced percutaneously into the RV using an 8 Fr. SR0 sheath (Daig®). The

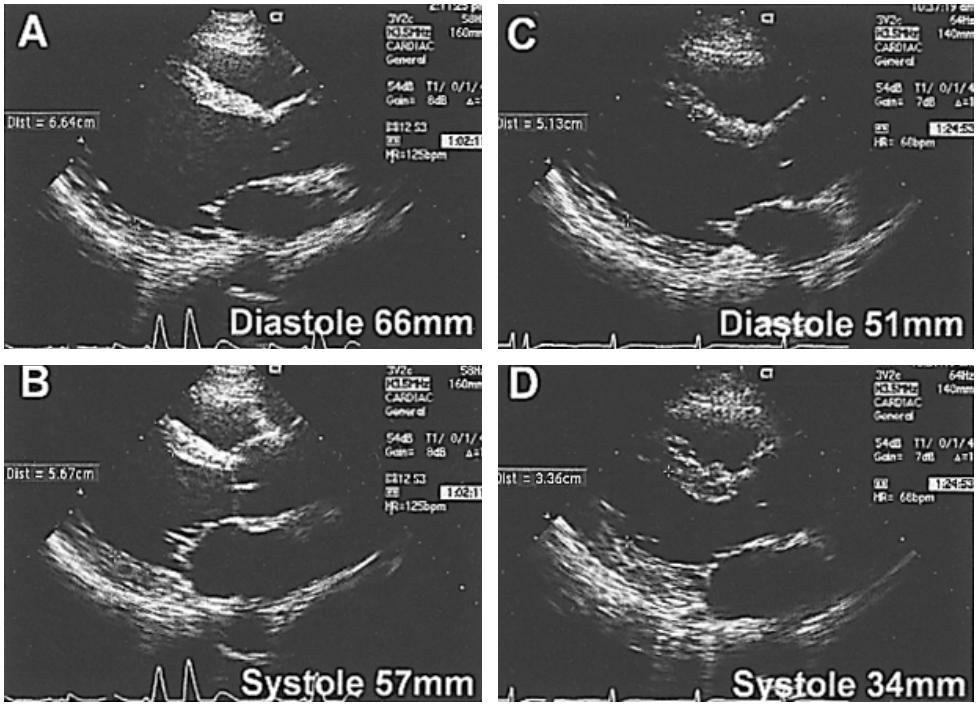

Figure 2. The two dimensional echocardiograms taken before (A, B) and 6 months after the catheter ablation (C, D). There was a markedly dilated left ventricular dimension (LVd), i.e., 66 mm at end-diastole (A), and 57 mm at end-systole (B). The completely normalized LV dimension and contractile function, i.e., an LVd of 51 mm at end-diastole (C), and 34 mm at end-systole (D).

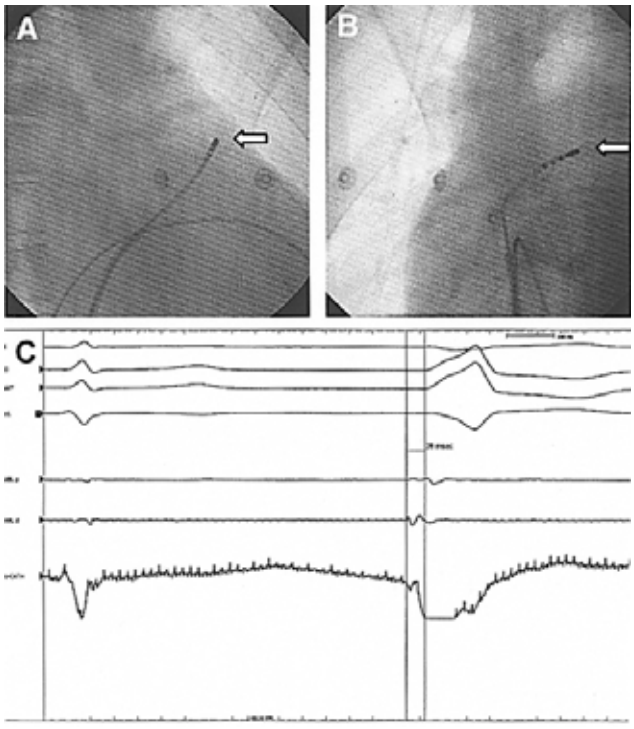

Figure 3. Fluoroscopic images and electrograms. The right anterior oblique (A) and left anterior oblique (B) views. Two electrode catheters were placed in the right ventricle. A mapping catheter (upper) is pointing at the triggering focus of the ventricular premature complexes (white arrows). C. The upper four signals are the surface electrocardiograms (ECG). The onset of the distal bipolar electrogram (the 6th line) recorded from the mapping catheter is 38 ms earlier than the ventricular premature complexes recorded on the surface ECGs; further, the unipolar electrogram (the lowest line) has an abrupt negative deflection. Both of those observations suggest the catheter is located at the optimal site for ablation.

origin of the spontaneous VPCs was determined based on the 12-lead-surface ECG and it was further located with the mapping catheter that was placed into the RV outflow tract. The triggering focus of the VPCs was located in the high anteroseptal region (Figure 3A, 3B).

The electrograms recorded from the ablation catheter were very tiny in the area that was mapped, and this was probably due to the scar formation created by the previous unsuccessful radiofrequency catheter ablation. The onset of the bipolar electrogram of the spontaneous VPCs recorded by the ablation catheter was 38 ms earlier than that of the surface ECG, and the unipolar electrogram had abrupt negative deflections without an initial positive component at the site we chose for ablation (Figure 3C). Radiofrequency energy was delivered for 60 seconds with a preset temperature of 60℃ and power limit of 40 W. The VPCs disappeared within 5 seconds after reaching a tissue-electrode temperature of 50℃. A booster application of energy was delivered to four sites around the successfully ablated focus for 30 seconds each.

Immediate progress and Follow-up

A 24 hour ambulatory ECG taken the same day as the radiofrequency catheter ablation procedure recorded only a few atrial premature complexes without any VPCs. The medications that has been administered for heart failure before the treatment were continued thereafter. No VPCs were observed throughout a series of ECGs that were recorded during the 6 months following his discharge. The LV dimensions and function returned to normal, as determined by the 6 month follow-up echocardiography: the LV end-diastolic dimension was 51 mm and the ejection fraction was 55% (Figure 2B).

DISCUSSION

Chronic supraventricular and ventricular tachycardias can lead to congestive cardiomyopathy in animal models[4] and in humans too[5]. Sustained rapid atrial or ventricular pacing in animals results in systolic heart failure that is neurohormonally and hemodynamically similar to the left ventricular systolic failure seen in humans[6-8]. The characteristic cardiac morphologic alterations in dogs include dilation of all 4 chambers and a normal or reduced ventricular wall thickness, with little or no change in the myocardial mass[8, 9]. The cellular changes include the loss of myocytes, cellular elongation, myofibril misalignment and the loss of the sarcomere register[7], which may be due to the derangement of the extracellular matrix. One of the proposed mechanisms for tachycardia-induced cardiomyopathy involves myocardial energy depletion and impaired energy utilization; this is manifested as reduced myocardial energy stores (including creatine, phosphocreatine and adenosine triphosphate), enhanced activity of the Krebs cycle oxidative enzymes, mitochondrial structural injury and functional abnormalities[7, 10]. The contractile reserve in response to inotropic agents, volume loading and postextrasystolic potentiation is either diminished or absent[11]. Reductions in the beta-adrenergic receptor density and also postreceptor abnormalities of adenylate cyclase and calcium processing that decrease the cardiac sympathetic responsiveness have been observed as well[12, 13]. It is not clear, however, if these factors lead to cardiac dysfunction or if they are merely a consequence of rapid pacing. Myocardial ischemia may impair the systolic function in the presence of a supraphysiologic heart rate, reduced systemic arterial pressure or increased ventricular diastolic pressure[14, 15]. Abnormal calcium processing may be responsible for tachycardia-induced cardiomyopathy. Extensive abnormalities of calcium channel activity and calcium transport

in the sarcoplasmic reticulum appear as early as 24 hours after the initiation of rapid pacing, and these abnormalities may persist for up to 4 weeks after the discontinuation of pacing[10, 16]. The severity of the calcium cycling abnormalities correlates with the degree of ventricular dysfunction[10]. In this manner, the calcium available to the myocytes may be decreased, with a subsequent reduction in the contractility. In the clinical field, achieving pharmacological control[17] and elimination of these tachycardias via surgical[18] or transcatheter ablation[19, 20] have demonstrated that this tachycardia-induced phenomenon is surely reversible.

Although the presence of VPCs has been found to be an independent risk factor for sudden death occurring for the patient who is in a post-myocardial infarction status, isolated VPCs are known to be the most common and benign arrhythmia that doctors encounter in routine examinations[21, 22]. Further, there is only minimal risk of VPCs, and especially right ventricular extrasystoles, in the patients without structural heart disease[23]. A series of patients with RVOT-VPC have recently been reported on. This was a common arrhythmia in that report, and it was able to cause LV dilation[1], which is a well-recognized precursor of LV dysfunction and heart failure. We experienced and report here on a patient with VPC-induced cardiomyopathy, whose left ventricle was not only dilated, but it was also accompanied with severe functional impairment. Although the frequency of the VPCs in our patient was similar to that in the previous report, our patient had suffered from them for a much longer duration than in the previous report (> 10 years vs. < 2 years, respectively). The LV dimension and function in our case completely recovered within six months after eliminating the RVOT-VPCs with performing radiofrequency catheter ablation. VPCs are one of the most common and benign arrhythmias, and they can cause marked LV dilation and severe functional impairment even in structurally normal hearts when they are very frequent and longstanding phenomena. RFCA should be the therapy of choice to treat this malady in this type of patient.

REFERENCES

1) Takemoto M, Yoshimura H, Ohba Y, Matsumoto Y, Yamamoto U, Mohri M, Yamamoto H, Origuchi H. *Radiofrequency catheter ablation of premature ventricular complexes from right ventricular outflow tract improves left ventricular dilation and clinical status in patients without structural heart disease. J Am Coll Cardiol 45:1259-1265, 2005*
2) Shraishi H, Ishibashi K, Urao N, Tsukamoto M, Hyogo M, Keira N, Hirasaki S, Shirayama T, Nakagawa M. *A case of cardiomyopathy induced by premature ventricular complexes. Circ J 66:1065-1067, 2002*
3) Chugh SS, Shen WK, Luria DM, Smith HC. *First evidence of premature ventricular complex-induced cardiomyopathy: a potentially reversible cause of heart failure. J Cardiovasc Electrophysiol 11:328-329, 2000*
4) Chow E, Woodard JC, Farrar DJ. *Rapid ventricular pacing in pigs: an experimental model of congestive heart failure. Am J Physiol 258:H1603-H1605, 1990*
5) Coleman HN 3rd, Taylor RR, Pool PE, Whipple GH, Covell JW, Ross J Jr, Braunwald E. *Congestive heart failure following chronic tachycardia. Am Heart J 81:790-798, 1971*
6) Armstrong PW, Stopps TP, Ford SE, de Bold AJ. *Rapid ventricular pacing in the dog: pathophysiologic studies of heart failure. Circulation 74:1075-1084, 1986*
7) Spinale FG, Hendrick DA, Crawford FA, Smith AC, Hamada Y, Carabello BA. *Chronic supraventricular tachycardia causes ventricular dysfunction and subendocardial injury in swine. Am J Physiol 259:H218-H229, 1990*
8) Scott BD, Sharma MK, Levett JM, Marinelli CC, Kieso RA, Schmid PG, Kerber RE. *Cardiac geometry and mass changes associated with pacing-induced cardiomyopathy in the dog. Am Heart J 125:1047-1053, 1993*
9) Shannon RP, Komamura K, Stambler BS, Bigaud M, Manders WT, Vatner SF. *Alterations in myocardial contractility in conscious dogs with dilated cardiomyopathy. Am J Physiol 260:H1903-H1911, 1991*
10) O'Brien PJ, Ianuzzo CD, Moe GW, Stopps TP, Armstrong PW. *Rapid ventricular pacing of dogs to heart failure: biochemical and physiological studies. Can J Physiol Pharmacol 68:34-39, 1990*
11) Komamura K, Shannon RP, Ihara T, Shen YT, Mirsky I, Bishop SP, Vatner SF. *Exhaustion of Frank-Starling mechanism in conscious dogs with heart failure. Am J Physiol 265:H1119-H1131, 1993*
12) Calderone A, Bouvier M, Li K, Juneau C, de Champlain J, Rouleau JL. *Dysfunction of the beta- and alpha-adrenergic systems in a model of congestive heart failure: the pacing-overdrive dog. Circ Res 69:332-343, 1991*
13) Marzo KP, Frey MJ, Wilson JR, Liang BT, Manning DR, Lanoce V, Molinoff PB. *Beta-adrenergic receptor-G protein-adenylate cyclase complex in experimental canine congestive heart failure produced by rapid ventricular pacing. Circ Res 69:1546-1556, 1991*
14) Spinale FG, Grine RC, Tempel GE, Crawford FA, Zile MR. *Alterations in the myocardial capillary vasculature accompany tachycardia-induced cardiomyopathy. Basic Res Cardiol 87:65-79, 1992*
15) Shannon RP, Komamura K, Shen YT, Bishop SP, Vatner SF. *Impaired regional subendocardial coronary flow reserve in conscious dogs with pacing-induced heart failure. Am J Physiol 265:H801-H809, 1993*
16) Perreault CL, Shannon RP, Komamura K, Vatner SF, Morgan JP. *Abnormalities in intracellular calcium regulation and contractile function in myocardium from dogs with pacing-induced heart failure. J Clin Invest 89:932-938, 1992*
17) Kugler JD, Baisch SD, Cheatham JP, Latson LA, Pinsky WW, Norberg W, Hofschire PJ. *Improvement of left ventricular dysfunction after control of persistent tachycardia. J Pediatr 105:543-548, 1984*
18) Giorgi LV, Hartzler GO, Hamaker WR. *Incessant focal atrial tachycardia: a surgically remediable cause of cardiomyopathy. J Thorac Cardiovasc Surg 87:466-469, 1984*
19) Packer DL, Bardy GH, Worley SJ, Smith MS, Cobb FR, Coleman

RE, Gallagher JJ, German LD. *Tachycardia-induced cardiomyopathy: a reversible form of left ventricular dysfunction. Am J Cardiol 57:563-570, 1986*

20) Park HW, Jong YJ, Kim JH, Kim W, Ahn YK, Jeong MH, Cho JG, Park CJ, Kang JC. *Radiofrequency catheter ablation in a patient with tachycardiomyopathy due to incessant fascicular tachycardia. Korean Circ J 34:1107-1112, 2004*

21) Wang K, Hodges M. *The premature ventricular complex as a diagnostic aid. Ann Intern Med 117:766-770, 1992*

22) Hallstrom AP, Bigger JT Jr, Roden D, Friedman L, Akiyama T, Richardson DW, Rogers WJ, Waldo AL, Pratt CM, Capone RJ. *Prognostic significance of ventricular premature depolarizations measured 1 year after myocardial infarction in patients with early postinfarction asymptomatic ventricular arrhythmia. J Am Coll Cardiol 20:259-264, 1992*

23) Zweytick B, Pignoni-Mory P, Zweytick G, Steinbach K. *Prognostic significance of right ventricular extrasystoles. Europace 6:123-129, 2004*