Case Report

Cardiac Pacing Suppressed Macroscopic T Wave Alternans in a Patient with Heart Failure Caused by Non-ischemic Cardiomyopathy

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A 67-year-old male with dilated cardiomyopathy and chronic renal failure who received chronic hemodialysis for 9 years admitted with pulmonary edema. Three days after admission, electrocardiogram showed transient prolongation of QT interval which was followed by macroscopic T wave alternans (TWA) and ventricular fibrillation (VF). Temporary pacing from right ventricular apex suppressed TWA and VF effectively. Combined cardiac resynchronization therapy and implantable cardioverter defibrillator device was implanted for the secondary prevention of VF. Both prolongation of QT interval and TWA disappeared for 10 days after therapy and no arrhythmic event occurred since then.

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Key words: QT prolongation, T wave alternans

Introduction

T wave alternans (TWA) is an electrocardiogram (ECG) phenomenon characterized by beat-to-beat changes in amplitude, shape and frequently, the polarity of the T wave during sinus rhythm without concomitant QRS changes. It is known to be an important prognostic indicator in that it is often observed preceding episodes of torsade de pointes.1–3) TWA is observed under diverse conditions including coronary artery occlusion, hypothermia, vasospastic angina, Brugada syndrome and the long QT syndrome.4,5) Some agents are reported to suppress the TWA, but to the best of our knowledge, there are few reports in which cardiac pacing suppressed the macroscopic TWA with the long QT syndrome. We report a patient with cardiomyopathy who showed macroscopic TWA and ventricular fibrillation (VF), both of which were eliminated by the temporary right ventricular pacing.

Case report

A 67-year-old male with dilated cardiomyopathy (DCM) and chronic renal failure who received chronic hemodialysis for 9 years admitted with...
pulmonary edema and congestive liver. He had no family history of sudden cardiac death. He had been treated with oral digoxin and enalapril. His left ventricular ejection fraction (LVEF) worsened from 55 to 27 percent on echocardiography within one year.

On physical examination, the blood pressure, pulse rate and respiratory rate were 180/110 mmHg, 80 beats/min and 36/min, respectively. Jugular venous distension was visible, S3 and S4 sounds were auscultated and pitting edemas were seen in the lower extremities. His liver and renal function decreased markedly on admission and the values revealed extremely high level of glutamic oxaloacetic transaminase (GOT) (1,270 IU/l), glutamic pyruvic transaminase (GPT) (502 IU/l), lactate dehydrogenase (LDH) (944 IU/l), prothrombin time international normalized ratio (PT-INR) (2.09), blood urea nitrogen (BUN) (60 mg/dl) and serum creatinine (Cre) (9.6 mg/dl), respectively. The value of brain natriuretic peptide (BNP) also increased to 8,683 pg/ml, but creatine kinase (CPK) was within normal level. The serum noradrenaline level was elevated significantly (4,135 pg/ml). The electrolytes did not show remarkable abnormalities (Ca 11.9 mg/dl, Na 139 mEq/l, K 5.4 mEq/l, Mg 2.3 mg/dl). The chest X-ray film revealed marked cardiomegaly, bilateral pleural effusion and pulmo-

### Table Echocardiographic findings

<table>
<thead>
<tr>
<th>M-mode</th>
<th>Valvular lesion</th>
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<tbody>
<tr>
<td>LAD (mm)</td>
<td>34</td>
</tr>
<tr>
<td>LVDd (mm)</td>
<td>57</td>
</tr>
<tr>
<td>LVDs (mm)</td>
<td>50</td>
</tr>
<tr>
<td>IVS (mm)</td>
<td>12</td>
</tr>
<tr>
<td>LV PW (mm)</td>
<td>12</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>268.59</td>
</tr>
</tbody>
</table>

PE < 100 ml

LAD: left atrial diameter, LVDd: left ventricular end-diastolic dimension, LVDs: left ventricular end-systolic diameter, IVS: interventricular septal wall thickness, LV PW: left ventricular posterior wall thickness, LV mass: left ventricular mass, PE: pericardial effusion, AR: aortic valve regurgitation, AS: aortic valve stenosis, MS: mitral valve stenosis, TR: tricuspid valve regurgitation, PR: pulmonary valve regurgitation

Figure 1

The 12-leads ECG on admission. This shows left ventricular hypertrophia with a strain pattern and QT and QTC interval prolongation (0.48 s and 0.558 s, respectively).

Figure 2

a) The 12-leads ECG before the occurrence of VF (day 2). Macroscopic TWA is present and QT intervals are prolonged. Heart rate is 70 beats/min. b) The rhythm strip of the 12-leads ECG which shows TWA and PVC. Shown is II lead and the paper speed is 12.5 mm/sec. The longer QT interval is 0.76 seconds and the shorter is 0.6 seconds. PVC occurs on the preceding longer T wave (the coupling interval was 0.66 seconds).
The ECG showed left ventricular hypertrophy with a strain pattern and prolongation of QT and QTc interval (0.48 sec and 0.558 sec, respectively) (Figure 1). The echocardiography revealed generalized mild hypertrophy of left ventricle and diffuse hypokinesis with 22 percent of LVEF by modified Simpson’s method. The other echocardiographic findings are shown in Table. After hemodialysis was initiated, his general conditions improved slightly, however QT interval became more prolonged together with the appearance of macroscopic TWA (Figure 2a). Premature ventricular contraction (PVC) occurred on the preceding T wave frequently (Figure 2b) and self-terminating VF appeared repeatedly (Figure 3). Coronary angiography showed no organic stenosis or coronary vasospasm. The temporary pacing from right ventricular apex was initiated (Figure 4) and it successfully eliminated TWA and suppressed the occurrence of VF for 4 days since then. As pacing rate was increased from 80/min to 100/min, the R wave voltage in V4 through V6 decreased gradually, and TWA was suppressed in all of the pacing rates similarly. However, after the pacing failure accompanied with dislodgment of the lead induced by his unconscious pull, VF spontaneously occurred again. Thereafter the QT interval remained prolonged without recovery to normal range for 3 more days.

Consequently, combined cardiac resynchronization therapy and implantable cardioverter defibrillator device (CRTD) was implanted and pacing was performed by the AAI mode with the rate of 80/min. Although left ventricular function didn’t improve on echocardiography, the morphologic changes of QT-T wave returned gradually to the baseline level before the occurrence of TWA and no VF occurred during the hospitalization.

**Discussion**

The etiologies of acquired long QT syndrome are known to be due to the drugs, heart block, abnormal electrolytes (hypokalemia and hypomagnesemia), acute myocardial infarction, intracranial injury, myocarditis and heart failure. In the present case, the patient had only congestive heart failure among the above etiologies. The echocardiography after the CRTD implantation didn’t exhibit improvement in the contractility, suggesting that the pathogenesis of impaired myocardium in this case was unlikely to be caused by myocarditis and Takotsubo cardiomyop-
atrialty. It is reported that action potential duration is prolonged due to delayed repolarization process in heart failure.\(^7\) Moreover, ventricular hypertrophy may be the cause of the longer action potential duration in acquired long QT syndrome.\(^5\) In the experimental models mimicking the long QT syndrome and also in patients with the congenital long QT syndrome, sympathetic stimulation and catecholamines are reported to have a relationship with QT prolongation.\(^8\)–\(^12\) And some authors report that in the clinical settings, TWA is detectable in patients with chronic heart failure and cardiomyopathy.\(^13\),\(^14\)\(^,\)\(^15\) In this case, DCM and hemodialysis seems to be the basis of cardiac disease and which are thought to have caused the myocardial disarray and fibrosis more than expected from the ventricular wall thickness on echocardiography. These structural abnormalities, heart failure and high level of catecholamine might have impaired the ionic property and cellular calcium handling, leading to occurrence of TWA and VF.

It is unclear why TWA disappeared after the pacing from right ventricular apex. Moreover, the QRS morphology of the paced beat in this case is different from that of the normal paced beat. There are R waves in V1 and V2 leads and no S waves in V5 and V6 leads. Auricchio et al. reported that patients with heart failure and left bundle-branch block have functional block line in the left ventricle during the sinus and paced rhythm.\(^16\) Although the ECG of this case didn’t show left bundle-branch block pattern, the activation of the left ventricle might have been blocked functionally, which resulted in the atypical QRS morphology in the paced rhythm. Shimizu et al. reported that both blocking the sarcoplasmic reticulum (SR) Ca channel by ryanodine and depletion of SR calcium with low extracellular Ca\(^{2+}\) concentration could eliminate the TWA.\(^1\) And it is also reported that some agents have influences on the action potential duration restitution curve of myocardial cell and can prevent VF. We thought it unlikely that right ventricular pacing itself could act on these factors and eliminate TWA, but altered left ventricular activation pattern might have some benefits to the ionic basis derived from heart failure and VF was suppressed successfully under the paced rhythm in this case.

References

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