CASE REPORT

Diffuse Inverted T Waves in a Young Man with Structurally Normal Heart: A Case Report

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We discuss a young man with episodes of chest pain and dyspnea accompanied with transient T-wave inversion in precordial and inferior leads that repeated several times. Cardiovascular evaluation did not disclose any structural abnormality. Memory T wave following episodes of idiopathic left septal ventricular tachycardia was the reason of these changes and did not repeat after arrhythmia ablation.

Ann Noninvasive Electrocardiol 2013;18(4):409–412

memory T wave; idiopathic ventricular tachycardia; inverted T wave

CASE PRESENTATION

A 25-year-old man was brought to emergency department by his friends because of atypical chest pain and dyspnea. Physical examination did not disclose any abnormality.

There was no significant past medical illness and drug abuse history. Laboratory data including serum cardiac enzyme levels and sedimentation rate were within normal limits.

The 12-lead electrocardiography (ECG) showed normal sinus rhythm with deep inverted T waves in leads of I, II, III, aVF, V₃–V₆ (Fig. 1).

He was referred to our center for evaluation of cardiovascular system next day. More detailed history taking did not add any more information except history of palpitation occurring frequently but persisting less than 5 minutes. The new ECG was normal (Fig. 2).

Transthoracic echocardiography showed normal heart chambers with normal contractility.

Two weeks later, the patient experienced another episode of dyspnea and palpitation. The ECG again showed normal sinus rhythm with

inverted T waves in inferior and precordial leads that changed to normal later. More detailed echocardiography including tissue Doppler imaging did not show any abnormal finding. Twenty-four-hour Holter monitoring did not show any significant arrhythmia. Multislice computed tomography (Siemens SOMATOM Sensation 64, Siemens, Erlangen, Germany) showed patent epicardial coronary arteries. Signal-averaged ECG was normal.

The scenario repeated four times among 3 months and cardiovascular studies did not show any abnormality except transient diffuse T-wave inversion. Because of the history of frequent palpitation, we decided to perform electrophysiological study. The ECG was normal at the beginning of study. Ventricular extra-stimulation from right ventricle apex induced a wide complex tachycardia with right bundle branch block (RBBB) morphology pattern and superior axis and cycle length of 398 ms (Fig. 3) repetitively that could be terminated with overdrive ventricular pacing. Intracardiac electrocardiograms showed ventriculo-atrial dissociation and negative H-V interval (Fig. 4). The

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No disclosure.

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DOI:10.1111/anec.12013

Figure 1. Twelve-lead electrocardiogram taken at emergency department showing sinus rhythm with diffuse T-wave inversion.

Figure 2. Twelve-lead electrocardiogram taken next day showing sinus rhythm with normal upright T waves.

ECG after arrhythmia termination showed the same T-wave changes that had been observed before. According to our findings, we decided to ablate the arrhythmia. Radiofrequency catheter ablation of arrhythmia was performed in poster-

obasal area of left ventricle septum and then no arrhythmia was inducible even after isoproterenol administration.

During 3 months follow-up, the patient was asymptomatic and the ECG was normal.

Figure 3. Twelve-lead electrocardiogram taken at electrophysiology laboratory showing the inducible arrhythmia with RBBB pattern morphology.

Figure 4. Intra-cardiac electrocardiograms (EGM) showing ventriculoatrial dissociation and negative H-V interval. (A: Atrial EGM, H: His EGM, V: Ventricular EGM)

DISCUSSION

T-wave changes may be secondary to abnormalities of depolarization like preexcitation or bundle branch block or may be unrelated to any QRS abnormality, in which case they are called primary repolarization abnormalities.¹

The term of diffuse T-wave inversion is used for the widespread T-wave inversion in ECG leads which always shows a reciprocal upright T wave in lead aVR and may also spare leads V₁, III, or aVL.²

Some important causes of diffuse T-wave inversion are myocardial ischemia, myocarditis, pericarditis, hypertrophic cardiomyopathy,

stress-induced cardiomyopathy, cardiac metastases, metabolic disorders, central nervous system disorders especially cerebrovascular accidents, pheochromocytoma, cocaine use, high-grade atrioventricular block, pulmonary embolism, and nonischemic pulmonary edema.^{1,2}

In our case, cardiovascular evaluation during 3 months did not disclose any structural abnormality and only after electrophysiologic study, we realized the presence of ventricular tachycardia. The T-wave inversion occurred after arrhythmia termination, disappeared the next day, and was not repeated during 3 months follow-up. Hence, cardiac memory T wave is the most probable pathophysiology of diffuse T-wave inversion in our patient.

Cardiac memory T wave is described as the repolarization changes subsequent to depolarization changes caused by ventricular arrhythmia, ventricular pacing, intermittent pre-excitation, or intermittent left bundle branch block.³

This process occurs when the heart resumes a sinus rhythm after a period of abnormal depolarization. It can occur even after episodes of abnormal ventricular activation with duration of as short as 1 minute.⁴

T-wave changes lasting minutes to hours, often observed after brief tachyarrhythmia episodes, are described as short-term memory. In contrast, long-term memory exhibit T-wave changes lasting hours to days can be seen in patients with temporary

cessation of permanent pacing or after ablation of accessory pathways.⁴

It is a consequence of the antecedent aberrant conduction pattern and can be an adaptive process to allow resynchronization of myocardial repolarization in a new activation sequence setting. The underlying molecular mechanism is complex. Ion channel function changes, factors of transcription, changes in action potential, calcium handling, and gap junction redistribution triggered by altered local myocardial strain are some of these mechanisms.⁴⁻⁶

It is interesting that palpitation was not the primary symptom in our patient. The relatively slow rate of arrhythmia and spontaneous termination of arrhythmia may be the reason.

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