CASE REPORT

Bifid T waves induced by isoprenaline in a patient with Brugada syndrome

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Abstract

A 41 year old man with incomplete right bundle branch block and persistent coved-type ST elevation in the right precordial leads during sinus rhythm had an episode of syncope while driving. He had never had syncope before and there was no family history of sudden cardiac death. Ventricular fibrillation was induced during electrophysiological study (EPS) by double extrastimuli applied to the right ventricle. Disopyramide was effective in preventing ventricular fibrillation during EPS. β Adrenoceptor stimulation manifested bifid T waves and reduced ST segment elevation in right precordial leads. Simultaneously recorded monophasic action potential (MAP) duration at 90% repolarisation did not change in the right ventricular outflow tract, while it shortened in the left ventricular septum. These findings suggest that right precordial bifid T waves might result from relatively early repolarisation of the left ventricles. Moreover the gradient of action potential duration might explain the mechanism of ST segment abnormalities in a patient with Brugada syndrome.

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Keywords: ventricular fibrillation; Brugada syndrome; monophasic action potential; bifid T waves

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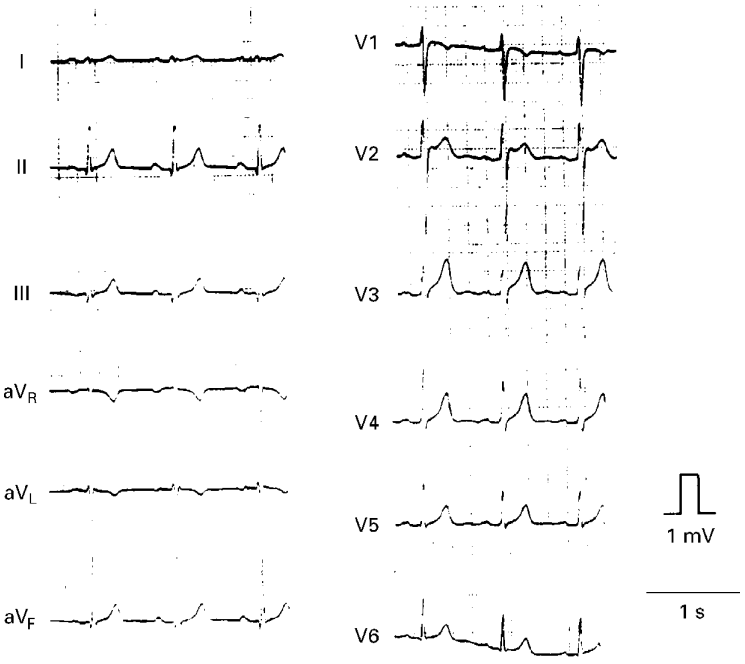

Figure 1 12 lead surface ECG on admission.

Bifid T waves are frequently observed in healthy subjects as a normal variant or in certain pathological conditions, such as heart failure, ischaemia, and left ventricular hypertrophy. The mechanism producing bifid T wave remains unclear and might not be the same in each case.

Patients with right bundle branch block, persistent ST segment elevation in precordial leads, and sudden cardiac death are a clinical entity of idiopathic ventricular fibrillation (VF) as reported by Brugada *et al.*[1] We report a patient with Brugada syndrome who showed morphological changes of bifid T waves in precordial leads corresponding with the change in monophasic action potential (MAP) duration when given isoproterenol. Asynchronous repolarisation seemed to be the mechanism of bifid T wave in this case. These findings might partially explain the mechanism of ST segment elevation in Brugada syndrome.

Case report

A 41 year old man had a syncopal episode while driving, causing a traffic accident. He had never experienced syncope before and there was no family history of sudden cardiac death. He was hospitalised, at another hospital, and there were no abnormalities on laboratory findings. Coronary angiography showed normal coronary arteries, and vasospasm was not induced by intracoronary ergonovine infusion. Left ventriculogram showed normal ventricular performance. He was referred to our hospital for further evaluation.

Surface ECG showed sinus rhythm and incomplete right bundle branch block with an ST segment elevation in precordial leads (fig 1). The QT and corrected QT (QTc) intervals were 0.40 seconds and 0.46, respectively. The ST segment elevations showed day to day variation and were minimised during exercise

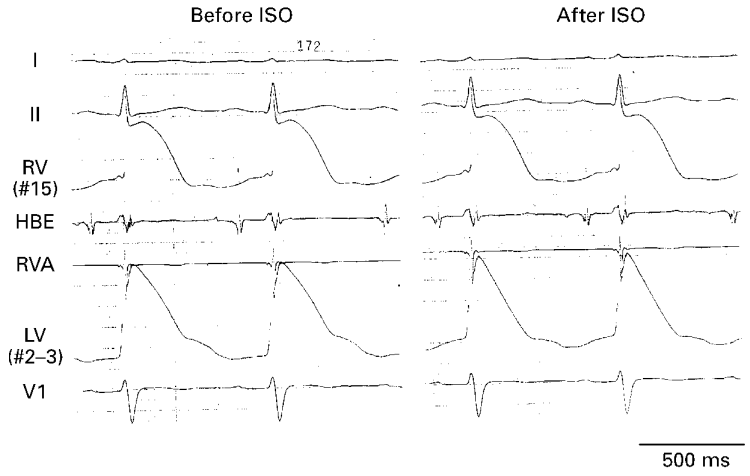

Figure 2 Monophasic action potential recordings before and after isoprenaline (ISO) administration. RV, right ventricle; HBE, His bundle recording site; RVA, right ventricular apex; LV, left ventricle.

stress test. Metaiodobenzylguanidine (MIBG) uptake was slightly reduced in the inferoposterior area of the left ventricle. After obtaining written informed consent, electrophysiological study (EPS) was performed by the standard procedure.[2] Under fluoroscopy, three 6 F quadripolar catheters with a 5 mm interelectrode distance (USCI Inc, Division of CR Bard, Boston, Massachusetts, USA) were positioned against the high right atrium, at the His bundle electrogram recording site, the apex, and/or the outflow tract of the right ventricle. No abnormalities were detected in sinus node function or atrioventricular conduction. VF was induced by double ventricular extrastimuli from the right ventricular outflow tract and DC shocks were required for termination.

Class IA drugs have had beneficial effects in patients with idiopathic VF,[3] so disopyramide was chosen as the first choice drug for pharmacological treatment. After two weeks of disopyramide (300 mg/day), non-sustained ventricular tachycardia (VT) was induced in a second EPS. Addition of intravenous disopyramide (50 mg) prevented ventricular tachyarrhythmias by programmed stimulation even after the use of isoprenaline. At the second EPS, MAP was recorded during sinus rhythm from the right and left ventricular septum using a 7 F MAP catheter (EP Technologies Inc, Sunnyvale, California, USA). MAP signals were amplified and filtered at a frequency of 0.03–500 Hz. MAP duration was measured at 90% repolarisation ($MAPD_{90}$). Before and after isoprenaline infusion (1 μg/min), MAP at both ventricular sites were recorded with a surface ECG. In the control state, $MAPD_{90}$ was 270 ms at the right ventricle and 310 ms at the left ventricle, and a notch was seen in the T wave of the precordial leads. After isoprenaline infusion, although $MAPD_{90}$ at the right ventricle was shortened by only 5 ms, at the left ventricle it was shortened by 40 ms (fig 2). The difference in $MAPD_{90}$ between the right and left ventricles decreased from 40 ms to 5 ms. On the surface ECG, clear bifid T wave was induced. ST elevation was decreased in precordial leads after isoprenaline infusion (fig 3), but QTc interval in V5 remained unchanged (450 ms). The patient has remained asymptomatic for two years taking disopyramide 450 mg/day.

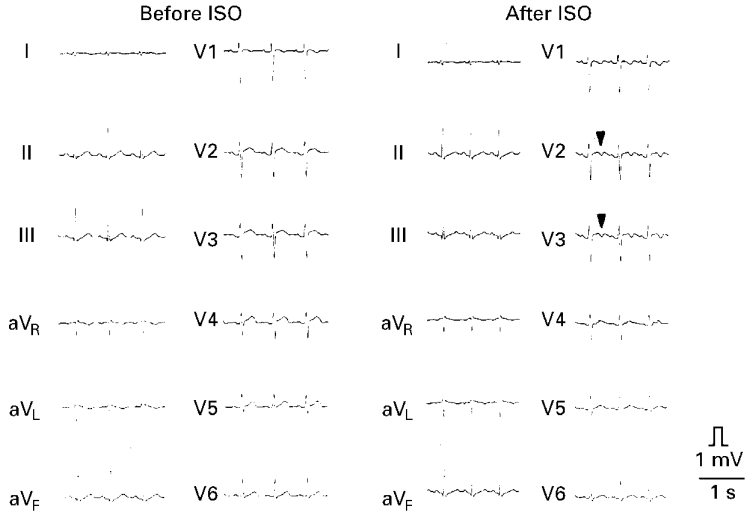

Figure 3 12 lead surface ECGs before and after isoprenaline (ISO) administration. Arrowheads indicate prominent bifid T waves.

Discussion

The mechanism of the appearance of bifid T wave remains uncertain. Watanabe *et al* reported that right precordial bifid T waves in younger patients with otherwise normal ECGs resulted from delayed right ventricular repolarisation, and left precordial bifid T waves in older patients with pathological conditions resulted from repolarisation delay in the left ventricle.[4] In addition, the bifid T wave in the younger cases were accentuated by exercise while in the older cases it was diminished. In the present patient, prominent bifid T waves were induced in the precordial leads after isoprenaline infusion. Furthermore, in comparison with MAP in the left ventricle, MAP in the right ventricle showed relatively delayed repolarisation after isoprenaline. Therefore, it is likely that facilitated repolarisation of the left ventricle produced the bifid T wave. Indeed, marked shortening of left ventricular MAPD was observed after isoprenaline infusion. These results and the finding of MIBG scanning might also indicate the autonomic imbalance between right and left ventricle, and hypersensitivity of the left ventricle to sympathetic tone or isoprenaline.

Brugada syndrome is a distinct clinical syndrome that causes sudden cardiac death. There are several explanations for ST segment elevation including intraventricular conduction disturbance, early repolarisation, local ventricular repolarisation, and sympathetic imbalance.[5] In this patient, both exercise and isoprenaline infusion reduced ST segment elevation and the latter decreased the dispersion of $MAPD_{90}$ between the right and left ventricle. Therefore, the dispersion of action potential durations between the two ventricles might partially cause ST segment elevation in Brugada syndrome, and isoprenaline might

preferentially affect the site with slower repolarisation as previously reported in early repolarisation syndromes.[6] However, further investigation of the relation between augmentation of ST segment elevation and the dispersion of MAP is necessary, as we recorded MAP from only a few sites.

In conclusion, bifid T waves in a patient with Brugada syndrome may have been caused by relatively early repolarisation of the left ventricle. And the mechanism of ST segment elevation might be associated with the dispersion of action potential duration in the right and left ventricles.

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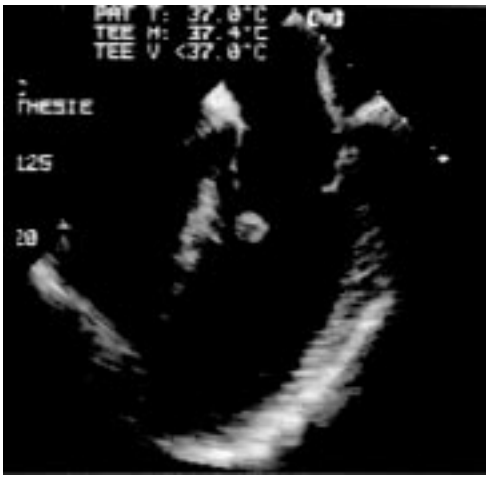
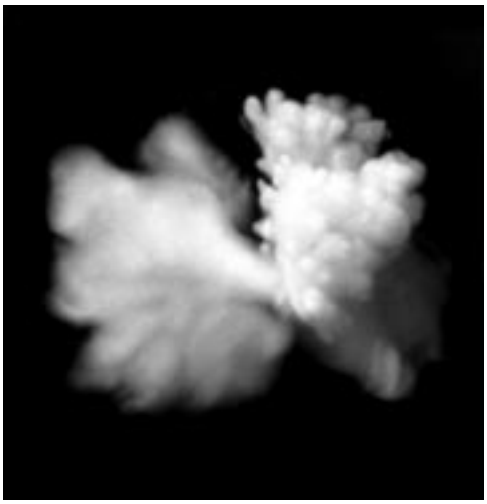
IMAGES IN CARDIOLOGY

Cardiac papillary fibroelastoma with cerebral and coronary embolic events

A 44 year old woman with no prior cardiac or neurological symptoms was admitted to the intensive care unit because of acute chest pain. On physical examination there were no initial abnormal cardiac findings. However, the patient then had a cardiac arrest with documented complete heart block and unconsciousness from which she was rescued mechanically. Electrocardiography showed a discrete transitory T wave inversion in lead V2 and there were minor creatine kinase abnormalities. Transthoracic and transoesophageal echocardiography showed a spherical tumour attached to the free margin of the anterior leaflet of the mitral valve without mitral regurgitation and normal contractions in all segments of the left ventricle. Coronary angiography showed an isolated peripheral embolic occlusion of the second diagonal branch of the left anterior descending coronary artery. Because of new mild neurological symptoms (Babinski sign, nystagmus, increased tendon reflexes on both sides) computed tomography of the CNS was performed, which showed one recent and multiple small older brain infarcts. The patient recovered well from these acute events and a few days later the tumour at the mitral valve was excised successfully. It was a white villous-like mass that was attached by a stalk to the free margin of the anterior leaflet of the mitral valve resembling a sea anemone. Histopathological examination confirmed the diagnosis of cardiac papillary fibroelastoma. The patient had an uncomplicated postoperative recovery.





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