Life-Threatening Ventricular Arrhythmia Following Exercise-Induced Vasospastic Myocardial Ischemia at the Site of a Myocardial Bridge With Progressive J-Wave Manifestation

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Figure. (A) Progressive J-wave manifestation (black triangle, leads I, II, III, aVF; blue triangle, lead V4) over time during treadmill exercise test. (B) At 3 min 26 s after starting exercise test, progressively manifested J-wave (black triangle, leads I, II, III, aVF; blue triangle, lead V4) accompanied ST-segment elevation at heart rate (HR) <126 beats/min, preceding induction of ventricular arrhythmia (VA). (C) Non-progressive J-wave manifestation (black triangle, leads II, V3, V4) during atrial pacing at 120 beats/min, which was almost concordant with the HR in (B) compared with the J-wave at rest. (D) Non-progressive J-wave manifestation accompanying ST-segment elevation in the anterior lead (black triangle, leads V3–V5) at 70 beats/min during implantable cardioverter defibrillator (ICD) implantation. (E) Coronary angiography obtained during the ICD implantation (right anterior oblique [RAO] 25 views) showing a sub-total occlusion in the mid-left anterior descending artery (mid-LAD; white triangles). Black arrows, septal perforator branch. (F) Normalized ST-segment and J-wave (black triangle, leads V3–V5) in the anterior lead after i.c. nitrate treatment. (G) Coronary angiography obtained after i.c. nitrate treatment (RAO, 35 views) resolved the sub-total occlusion (white triangles). Black arrows, same septal perforator branch as in (E). (H) Coronary computed tomography showing a myocardial bridge in the mid-LAD (white triangles) and diagonal branch (white arrow). Black arrows, same septal perforator branch as in (E,G).
A 73-year-old male current smoker complained of a pre-syncopal attack during farm work. Baseline electrocardiogram indicated a notch-type J-wave inscribed on the S-wave in the inferolateral and anterior leads. On treadmill exercise test, a progressive J-wave manifestation was inscribed on the latter part of the R-wave (Figure A). J-wave accompanied ST-segment elevation at heart rate (HR) <126 beats/min, and a polymorphic ventricular arrhythmia (VA) was induced (Figure B). The VA terminated at 24 s after the exercise test was stopped, and recovered to a sinus rhythm (SR) of 83 beats/min. The patient complained of the same pre-syncopal attack during VA. On echocardiography and cardiac magnetic resonance imaging there was no evidence of a structural heart disease or arrhythmogenic substrate. A non-progressive J-wave manifestation (Figure C) was seen during atrial pacing at 120 beats/min, which was almost concordant with the HR in Figure B. A non-progressive J-wave manifestation was also seen during implantable cardioverter defibrillator (ICD) implantation at 70 beats/min, accompanied with ST-segment elevation in the anterior lead without induction of the VA (Figure D). Coronary angiography during the ICD implantation showed a subtotal occlusion in the mid-left anterior descending artery (LAD; Figure E; Movie S1). The occlusion was resolved with i.c. nitrate (Figure G; Movie S2) with normalization of the ST-segment and the J-wave (Figure F). The diagnosis was confirmed as vasospastic angina (VSA). Coronary computed tomography showed a myocardial bridge, concordant with the culprit site of the VSA (Figure H). The VSA at the site of the myocardial bridge in the mid-LAD caused the non-progressive J-wave manifestation and the ST-segment elevation at the same time.1

The VSA was considered to be the cause of the ST-segment elevation during the treadmill exercise test, as well as during ICD implantation. The likely mechanism of the VSA at the site of the myocardial bridge during both exercise test and ICD implantation was hypersensitivity to catecholamine because of endothelial dysfunction by chronic mechanical stress due to systolic squeezing.2

Exercise-induced VSA and J-wave manifestation could occur in patients with prior stent implantation in the LAD.3 Increases in HR could manifest as J-wave in patients with VSA and slight myocardial damage.4 In the present case, the J-wave was progressively manifested by both the increases in HR and the VSA during exercise test, despite the lack of arrhythmogenic substrate. The progressive J-wave manifestation and the VSA-induced global myocardial ischemia during exercise test could provoke the life-threatening VA. Calcium channel antagonist (benidipine 8 mg/day) was given to prevent onset of VSA, and the patient was free from pre-syncopal attack or VA episode on benidipine during the following year, despite not reducing his workload. In daily practice, careful attention should be paid to intense exercise in patients with J-wave.

Disclosures
The authors declare no conflict of interest.

References

Supplementary Files
Supplementary File 1
Movie S1. Coronary angiography as in Figure E.
Supplementary File 2
Movie S2. Coronary angiography as in Figure G.
Please find supplementary file(s):