A 77-year-old woman was referred for a dobutamine stress test. She had a prior history of hypertension. Basal ECG was normal (Figure 1A). At 40 μg \cdot kg^{-1} \cdot \text{min}^{-1} she developed typical chest pain with ST-segment elevation in DI, DII, and the anterior leads with ventricular bigeminy (Figure 1B and 1C). The echocardiogram showed apical and mid-wall myocardial segment akinesis with basal hyperkinesis and left outflow tract gradient obstruction of 60 mm Hg. The ejection fraction was estimated to be 34%, and severe mitral regurgitation was found with no organic valvular disease (Figure 2 and Movie I in the online-only Data Supplement). Troponin I, creatinine kinase, and brain natriuretic peptide levels were 2.4 ng/dL (normal value, <0.04 ng/dL), 80 mg/dL (within normal range), and 928 pg/dL, respectively.

Emergent cardiac catheterization showed mild stenosis in the left anterior descending coronary artery with no signs of plaque rupture while the patient was still having chest pain and ST elevation. Left ventriculography displayed hyperdynamic basal myocardial segments with cavity obliteration and apical akinesis (Figure 3 and Movie II in the online-only Data Supplement).

Cardiac magnetic resonance depicted findings similar to the echocardiogram with no late gadolinium enhancement (Figure 4 and Movie III in the online-only Data Supplement).

The rest single-photon emission computed tomography sestamibi revealed a great perfusion defect that extended beyond the left anterior descending coronary artery territory (Figure 5A).

The patient made favorable progress and was discharged with β-blockers, aspirin, and simvastatin. Repeated echocardiography after 7 days demonstrated normal left ventricular wall motion, the ejection fraction was 55% (Figure 6 and Movie IV in the online-only Data Supplement), and single-photon emission computed tomography 3 months later showed normal perfusion (Figure 5B).

Dobutamine-precipitated takotsubo cardiomyopathy is an unusual presentation of this disease,1–4 which suggests catecholamine participation in the pathophysiology.

Disclosures
None.

References
Figure 1. A, Basal ECG. B, Peak stress ECG. C, Recovery (5 minutes) ECG.
Figure 2. Echocardiogram of the acute phase showing apical akinesis.

Figure 3. Left ventriculography in diastole (A) and systole (B) showing apical ballooning typical of takotsubo cardiomyopathy.

Figure 4. MRI showing no late gadolinium enhancement.
Figure 5. SPECT myocardial perfusion in the acute phase showing extensive apical compromise (A) and normalization (B) at 3 months follow-up. SPECT indicates single-photon emission computed tomography.

Figure 6. Echocardiogram showing normal left ventricular wall motion at 7 days follow-up.
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