A 66-year-old man was admitted to the emergency room because of 3-hour-long acute chest pain with dyspnea and nausea while fishing on a high-altitude (2400 m) lake in the very early morning. The patient was transferred by helicopter. Persistent chest pain without pathological clinical findings (SpO₂, 99%; shock index, 0.9; axillary temperature, 36.7°C) was present. Medical history revealed no previous cardiovascular events and no further cardiovascular risk factors except smoking (40 packs a year). ECG demonstrated ST-segment elevation (Figure 1A). ST-segment–elevation acute coronary syndrome was initially suspected. Immediate selective coronary angiography showed extensive left ventricular (LV) apical and midventricular akinesia with hyperkinesia in the basal segments and a moderate reduction in the estimated LV ejection fraction (Figure 1B and Movie I in the online-only Data Supplement). These large LV wall motion abnormalities were inconsistent with slightly elevated cardiac enzymes (troponin I, 0.48 μg/L [normal, <0.09 μg/L]; creatine kinase-MB, 18.7 UI/L [normal, <24 UI/L]). Clinical presentation and the absence of elevation of inflammation markers elevation and pathological viral tests and bacterial cultures made it possible to reasonably exclude acute myocarditis. ECG repolarization alteration (Figure 1C) and LV wall motion abnormalities recovered spontaneously and fully in 4 days (Figure 1D and Movie II in the online-only Data Supplement). Therefore, the diagnosis of apical-ballooning takotsubo cardiomyopathy (TTC) was made.

One year later, the patient, who incidentally stopped smoking after the TTC episodes, was admitted to the emergency room of another hospital because of acute chest pain. No precipitating episode was identified. ECG (Figure 2A), troponin I, blood samples, transthoracic echocardiography, selective coronary angiography, and LV volume (Figure 2B and Movie III in the online-only Data Supplement) displayed findings comparable with the previous episode. Furthermore, exclusion of late gadolinium enhancement on magnetic resonance imaging made it possible to exclude myocarditis. ECG repolarization alteration (Figure 2C) recovered fully and spontaneously. Four weeks after admission, transthoracic echocardiography demonstrated full recovery of the LV wall motion abnormalities (Figure 2D and Movie IV in the online-only Data Supplement), consistent with a diagnosis of a recurrent TTC.

Two years later, the patient was admitted again to the emergency room of a small district hospital because of acute chest pain and dyspnea while fishing on a lake on a cold morning. ECG showed T-wave inversion in the inferior leads and precordial leads V₃ through V₆ (Figure 3A). The patient was transferred to primary percutaneous coronary intervention–capable center, where transthoracic echocardiography, selective coronary angiography, and LV volume (Figure 3B and Movie V in the online-only Data Supplement) were comparable with those of the 2 preceding episodes (troponin I, 4.06 μg/L; creatine kinase-MB, 38 UI/L). ECG repolarization alteration (Figure 3C) recovered fully and spontaneously, and LV wall motion abnormalities recovered spontaneously and completely within 1 week (Figure 3D and Movie VI in the online-only Data Supplement). Therefore, the diagnosis of a third typical TTC episode was confirmed.

Normal 24-hour urine fractionated catecholamines and metanephrines and the absence of hypertension made it improbable that pheochromocytoma was the cause of these 3 TTC episodes.

TTC is an infrequent, mostly stress-related disease, affecting mainly postmenopausal women. Even rarer are recurrences (1.5%/y–2.9%/y), mostly seen in menopausal women and frequently with different LV wall motion abnormality patterns. To the best of our knowledge, this is the first reported case of 3 episodes of recurrent typical apical-ballooning TTC in an adult man. Moreover, this case underscores that both invasive ventriculography and transthoracic echocardiography should be implemented to obtain a correct diagnosis and during follow-up.

Disclosures

None.

References


Figure 1. A, ECG showing ST-segment elevation in the precordial leads. B, Ventriculography (longitudinal view, end systole) illustrates extensive left ventricular (LV) apical and midventricular akinesia (arrows) with hyperkinesia in the basal segments and moderately reduced LV ejection fraction (Movie I in the online-only Data Supplement). C, ECG shows spontaneously normalized repolarization within 4 days and left anterior fascicular block. D, Transthoracic echocardiography (4-chamber view, end systole) shows fully recovered LV wall motion abnormalities with normal ejection fraction within 4 days (Movie II in the online-only Data Supplement).
Figure 2. A, ECG showing T-wave inversion in inferior leads and precordial leads V₅ through V₆. B, Ventriculography (longitudinal view, end systole) proves extensive left ventricular (LV) apical and midventricular akinesia (arrows) with hyperkinesia in the basal segments and moderately reduced LV ejection fraction (Movie III in the online-only Data Supplement). C, ECG shows spontaneously normalized repolarization within 1 day and left anterior fascicular block. D, Transthoracic echocardiography (2-chamber view, end systole) shows fully reversible LV wall motion abnormalities with normal ejection fraction within 4 weeks (Movie IV in the online-only Data Supplement).

Figure 3. A, ECG showing T-wave inversion in inferior leads and precordial leads V₅ through V₆. B, Transthoracic echocardiography (TTE; 4-chamber view, end systole) shows extensive left ventricular (LV) apical akinesia and midventricular hypokinesia (arrows; Movie V in the online-only Data Supplement). C, ECG shows spontaneously normalized repolarization within 2 days and left anterior fascicular block. D, TTE (4-chamber view, end systole) demonstrates fully recovered LV wall motion abnormalities with normal ejection fraction within 1 week (Movie VI in the online-only Data Supplement).
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