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Letter to the editor

Pitfalls in coronary artery stenosis assessment in takotsubo syndrome: The role of microvascular dysfunction

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A 53-year-old lady with unremarkable medical history presented with chest pain after a robbery of her shop. ECG showed mild ST elevation in the anterior leads. Troponin I was slightly elevated (max value 1.36 ng/ml, ULN 0.04 ng/ml). In coronary angiography, diffuse coronary atheromatosis was noted. Left ventricular (LV) angiography showed a moderately depressed LV function (EF = 38%). There was akinesia of the mid-segments and majority of the apex with a small area of preserved contractility of the tip of the apex, consistent with the diagnosis of Takotsubo syndrome (TTS) with minimal apical sparing (Fig. 1A and B). At 1 month follow-up, echocardiography documented complete recovery of the LV function. The ECG evolution was characteristic for TTS with initial negative T waves associated with prolonged QT interval and a complete normalization after 2 months.

Six years later, during a second robbery of her shop, she presented again with chest pain. ECG showed a new complete Left bundle branch block (LBBB). Coronary angiography documented a moderate 50% LAD lesion with normal flow. LV angiography showed moderately depressed LV function (EF of 40%) and akinetic apical and mid-segments without apical sparing (Fig. 1C and D). The wall motion abnormalities extended beyond the territory of the Left anterior descending (LAD), consistent with a diagnosis of typical TTS.

At functional assessment under maximal myocardial hyperemia induced by intravenous adenosine, fractional flow reserve (FFR) of 0.92 (normal FFR > 0.80) indicated the presence of nonsignificant LAD lesion. Moreover, the index of myocardial resistance (IMR) was 43 (normal IMR < 22). Medical therapy with perindopril and bisoprolol was initiated. Given the elevated IMR potentially clouding the accuracy of the FFR assessment of the LAD lesion, repeat catheterization was scheduled 1 month later. LV angiography demonstrated a complete recovery of the LV function. Angiographically, the LAD lesion remained stable with 50% diameter reduction. It should be noted that functional assessment under maximal hyperemia induced by intravenous adenosine showed a preserved FFR value of 0.89 and a normalized IMR value of 10 (Fig. 1E and F). The moderate LAD stenosis was thus considered functionally nonsignificant bystander coronary artery disease (CAD) in a typical TTS. On ECG, the LBBB persisted.

This case demonstrates two proven episodes of TTS with a different anatomical variant despite being provoked twice by an identical emotional trigger. The recurrence of different anatomic variants has been well described in previous cases with a recurrence rate of ~1.8% per year.[1, 2] Our case highlights the coincidence of the recently described apical nipple sign variant with the classical initially described type of the disease.[3] At the same time, our patient posed a diagnostic dilemma because of the coincidence of CAD, which requires a cautious approach in TTS.

At the second episode, an angiographically moderate lesion at the mid part of LAD was evident. Coexisting CAD is reported to occur in ~15.3% of patients with TTS, and they are accountable for a substantial percentage of mortality among patients with this syndrome.[3] Functional assessment in the acute phase revealed an FFR value of 0.92. Of note, IMR was potentially elevated, compromising the accuracy of pressure-derived FFR. The repeat evaluation 1 month later demonstrated normalization of the myocardial resistance. This finding is consistent with microvascular dysfunction, sharing a fundamental role in transient LV impairment.[4] Note, microvascular dysfunction is known to blunt pharmacologically induced myocardial hyperemia and thereby confounding the functional assessment of coronary stenosis. Indeed, while still remaining above the ischemic threshold, the FFR value decreased from 0.92 to 0.89 after normalization of myocardial resistance. In this particular patient, this proved the presence of functionally nonsignificant bystander coronary atherosclerosis of the LAD.

In conclusion, in addition to different presentation of TTS with and without apical sparing, the present case demonstrates the reversibility of microvascular dysfunction and its impact on the functional assessment of the epicardial lesion using the pressure-derived FFR. Thus, in the case of CAD with moderate epicardial stenosis, the repeat functional evaluation should be considered to unequivocally determine the lesion severity.

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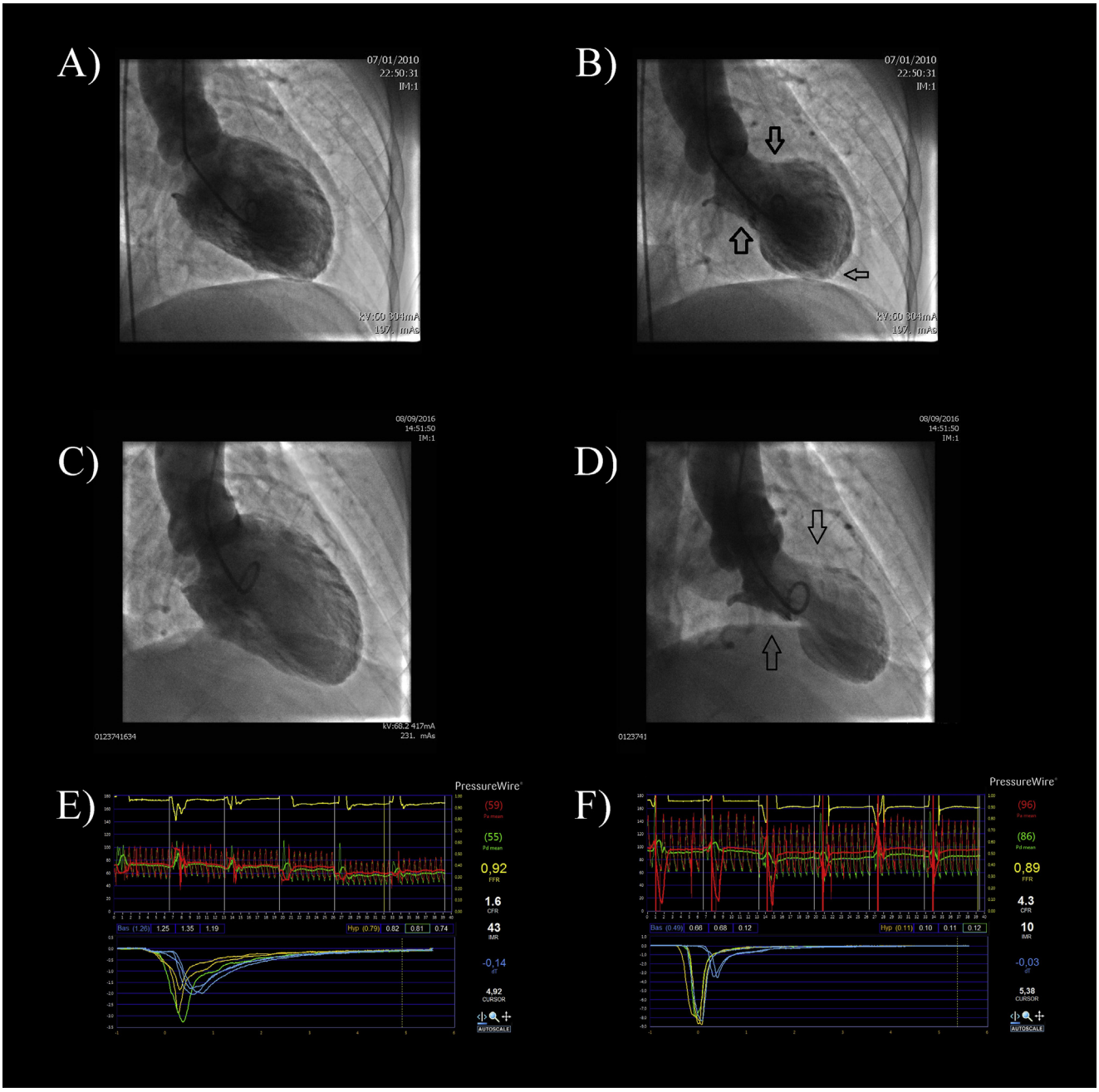

Fig. 1. Panel A,B: LV angiography in the acute phase of first episode in diastole (panel A) and systole (panel B) showing hypercontractility in basal segments (arrows) and a small zone of contraction of the distal apex (arrow) with akinesia of mid and majority of apical segments. Panel C,D: LV angiography in the acute phase of second episode in diastole (panel C) and systole (panel D) showing hypercontractility in basal segments (arrows) and akinesia of mid and apical segments. Panel E,F: FFR and IMR measurements in the acute phase (panel E) showing FFR 0.92 above ischemic threshold and elevated IMR of 43, measurements after 1 month (panel F) showing FFR 0.89 and IMR 10 within the normal range.

Conflict of interest

None declared.

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