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The answer to the riddle: Multimodality imaging for diagnosing a double hit of acute coronary syndrome and takotsubo syndrome

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Diagnosing the cause of a non-ST-segment elevation myocardial infarction (NSTEMI) may be challenging in the absence of clear angiographic signs of plaque rupture. Heitner et al. [1] demonstrated how delayed-enhancement cardiac magnetic resonance imaging (DE-CMR) identified a new culprit lesion or revealed a non-ischemic cause in nearly half of studied NSTEMI patients compared with judgment by coronary angiography alone. In the presented case of an older man with a NSTEMI, imaging modalities helped us to diagnose the underlying pathological mechanisms in the rare circumstance of a double hit by an acute coronary syndrome (ACS) and takotsubo syndrome (TTS).

A 75-year-old male patient initially presented at an external hospital with chest tightness and dyspnea for several hours. The patient was pain free at presentation and the clinical examination was unremarkable. Cardiac biomarkers were elevated [high-sensitive troponin T: 868 ng/L (normal < 14 ng/L); creatine kinase: 963 U/L (normal < 308 U/L)]. Electrocardiogram (ECG) initially showed non-significant ST-segment elevations in the inferior leads and slight T-wave alterations in leads V5–6. An ECG several hours later demonstrated dynamic changes with T-wave inversions in the majority of leads (except aVR, aVL and V1–2). The patient was referred to our hospital for coronary angiography that revealed severe three-vessel disease with normal coronary artery flow, but severe stenoses in all three major vessels (Fig. 1A–C). Left ventriculography showed a moderately reduced left ventricular ejection fraction (LVEF) with apical ballooning (Fig. 1D–F) consistent with TTS and extending beyond the vascular distribution of the left anterior descending artery (LAD). There were no clinical or angiographic signs of ongoing ischemia and due to the equivocal findings, the decision was made to perform a DE-CMR to differentiate between TTS and ACS. Meanwhile, therapeutic heparin and acetylsalicylic acid were continued, and an angiotensin-converting enzyme inhibitor was started.

Surprisingly, DE-CMR showed not only extensive myocardial edema of the midventricular and apical segments consistent with TTS (Fig. 1G), but also late gadolinium-enhancement of the mid-ventricular and apical inferolateral wall suggestive of myocardial infarction in the territory of the right coronary artery (RCA) or left circumflex artery (Fig. 1H).

Only few hours after CMR, the patient developed an acute infero-posterior ST-segment eleva-

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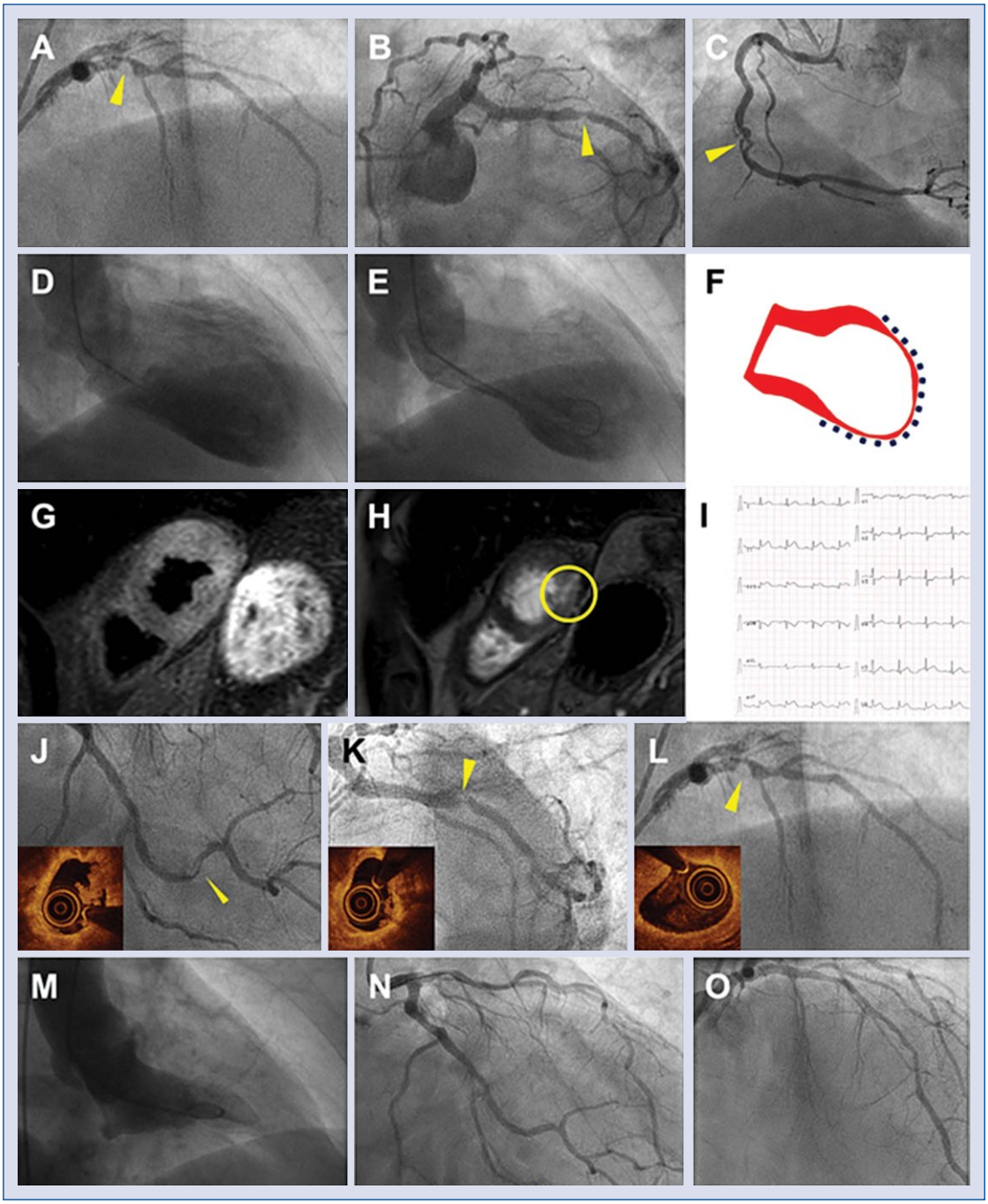

Figure 1. Coronary angiography showing three-vessel coronary artery disease with severe stenoses of the proximal left anterior descending artery (LAD) (**A**, arrowhead), the left marginal branch (**B**, arrowhead), and the mid-right coronary artery (**C**, arrowhead). Left ventriculography demonstrating akinesis of the midventricular and apical segments with normal contractions of the basal segments (**D**, **E**). Corresponding schematic of apical ballooning takotsubo syndrome (**F**, white: systole; red: diastole; blue dashed line: wall motion abnormalities). Cardiac magnetic resonance imaging, short axis, STIR sequence (T2-weighted) showing extensive edema of the midventricular segments (**G**). Cardiac magnetic resonance imaging, short axis, showing inferolateral late gadolinium enhancement (**H**, yellow circle). Electrocardiogram demonstrating ST-segment elevations in the inferior leads and V6 as well as ST-segment depression in V1–V3 consistent with infero-posterior ST-segment elevation myocardial infarction (**I**). Angiographic suspicion of an embolus in the distal left marginal branch (**J**, arrowhead) and evidence of thrombus on optical coherence tomography (OCT) (**J**, inset). Subtotal occlusion of the proximal left marginal branch (**K**, arrowhead) and evidence of thrombus on OCT (**K**, inset). Severe stenosis of the proximal LAD (**L**, arrowhead) without signs of plaque rupture, erosion, or thrombus on OCT (**L**, inset). Left ventriculography before staged percutaneous coronary intervention (PCI) of the LAD 1 month after the initial hospitalization demonstrating normalization of left ventricular function (**M**). Final result after PCI with stent implantation in the mid and distal part of the marginal branch (**N**) and in the proximal and distal part of the LAD (**O**).

tion myocardial infarction (STEMI) (Fig. 1I). By the time of immediate repeat coronary angiography, symptoms had resolved, and ST-segment changes had normalized. There was normal coronary artery flow in all three vessels. With the evidence of thrombus on optical coherence tomography (OCT) in the mid and distal part of the marginal branch (Fig. 1J and K, inset) it was considered that this vessel — rather than the RCA — is the culprit lesion of the actual STEMI and also of the initial NSTEMI with documented inferolateral scar on DE-CMR. Both lesions of the marginal branch were treated with implantation of a zotarolimus eluting stent (Resolute Onyx 2.5 mm × 12 mm proximal and 2.25 mm × 12 mm distal, Medtronic Inc., Minneapolis, MN, USA) (Fig. 1N). Furthermore, OCT was performed in the proximal LAD to rule out plaque rupture or plaque erosion as the cause of the apical ballooning (Fig. 1L, inset). Dual antiplatelet therapy was started with acetylsalicylic acid and ticagrelor.

A percutaneous approach was chosen for further revascularization of the remaining two-vessel disease due to patient preference and the distally diseased LAD being a suboptimal target for a coronary bypass graft. Based on the CMR and OCT findings, the proximal LAD stenosis was considered to be a stable lesion and it was assumed to be safer to delay revascularization for 1 month. Indeed, left ventriculography showed normalized LVEF by then (Fig. 1M) supporting the diagnosis of TTS. The stenoses in the proximal and distal LAD were treated with implantation of three stents (Resolute Onyx 3.5 mm × 15 mm, 2.5 mm × 22 mm and 2.25 mm × 15 mm) (Fig. 1O). After the patient developed shivering of unknown cause, planned revascularization of the RCA was not performed. A control DE-CMR did not show inferior ischemia and it was therefore decided to employ a conservative treatment.

The view herein, was that the most likely pathological mechanism in this case is a plaque rupture and infarction of the left marginal branch that triggered a TTS with reversible apical ballooning. ECG findings may support this sequence of events: while the ECG changes at presentation are more consistent with a marginal branch infarction, the widespread negative T-waves several hours later are more likely to have been caused by TTS, albeit T-wave inversions and their distribution in the present case are not specific for TTS or ACS [2].

Although exclusion of obstructive coronary artery disease was initially considered mandatory for the diagnosis of TTS, cases of TTS triggered by ACS have been reported, and may be underdiagnosed [3, 4]. In men, the prevalence of TTS is about 10 times lower than in women, while triggers are — as in our case — more often physical than emotional [5, 6].

This case represents a challenging clinical scenario in differentiating between ischemic and non-ischemic causes of NSTEMI on one hand, and identifying the culprit lesion in the context of three-vessel coronary artery disease on the other hand. Imaging with DE-CMR and OCT lead us to understand the case, and it is believed that the interplay between angiography and imaging modalities is critical to increase the diagnostic accuracy in NSTEMI patients.

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