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Authors: Kenan Yalta, Muhammet Gurdogan, Cihan Ozturk, Tulin Yalta

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MINOCA? Takotsubo syndrome? Or both? Pitfalls, clues and indications for advanced modalities in the differential diagnosis

Short title: Takotsubo syndrome and MINOCA

Kenan Yalta[1], Muhammet Gurdogan[1], Cihan Ozturk[1], Tulin Yalta[2]

[1]Department of Cardiology, Trakya University, Edirne, Turkey
[2]Department of Pathology, Trakya University, Edirne, Turkey

Correspondence to:
Kenan Yalta, MD,
Department of Cardiology, Trakya University,
Balkan Yerleşkesi, 22030, Edirne, Turkey,
phone: +90 50 565 79 856,
e-mail: kyalta@gmail.com, akenanyalta@trakya.edu.tr

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a challenging phenomenon attributable to conditions including microvascular dysfunction, spontaneous coronary artery dissection (SCAD), thrombo-embolism and vasospasm [1, 2]. Importantly, its differentiation from other non-coronary conditions including takotsubo syndrome (TTS) is essential for proper risk-stratification and management [1, 2]. The recent clinical vignette by Loboz Rudnicka et al. [1] has described a case of MINOCA who was initially diagnosed as having a classical TTS episode. Accordingly, we would like to highlight certain pitfalls and clues in the differentiation among MINOCA, TTS and combination of these pathologies along with indications for advanced diagnostic strategies in this context:

First, MINOCA might mimic TTS in certain cases [1]. Accordingly, acute coronary ischemia was previously suggested to elicit a 'pseudo-TTS' pattern as a consequence of ischemic or post-ischemic myocardial stunning [3, 4]. Therefore, a 'pseudo-TTS' pattern might also be possible in the present case [1]. Unlike a true TTS episode, this pattern does not extend beyond the territory of a single coronary artery [3, 4]. However, the affected myocardial portions in the patient [1] seem to be within the territories of both left anterior descending (LAD) and right coronary arteries (RCA). At first glance, this might denote a co-existing or isolated 'true TTS'in the patient largely based on normal distribution of her coronary arteries

(each coronary artery perfusing its own terrritory) on coronary angiogram (CAG). Importantly, recovery of a ‘pseudo-TTS’ pattern exhibits a potential correlation with the mitigation of myocardial ischemia [3, 4]. Therefore, was the recovery of wall motion abnormalities predominantly spontaneous or associated with the resolution of ischemic signs and symptoms?

Second, a true TTS episode might also mimic MINOCA. This particularly holds true in the setting of atypical TTS patterns that might be overlooked [4]. For instance; a missed focal TTS with a pattern of coronary slow flow CSF (due to new-onset and reversible microvascular dysfunction [3]) might be misdiagnosed as MINOCA (triggered by chronic microvascular dysfunction) with normal left ventricular systolic functions. Therefore, meticulous search for a segmentary dysfunction (and evaluation of its recovery pattern, if any) is necessary to differentiate between these two conditions.

Third, co-existence of MINOCA and TTS might also be quite possible [1, 3, 4]. These two conditions might arise concomitantly due to a common trigger including extreme adrenergic surge [3, 4]. However, MINOCA might, per se, trigger a TTS episode, and vice versa [3, 4]. For instance, severe chest pain due to MINOCA may lead to a subsequent TTS episode [3, 4]. Conversely, TTS might potentially lead to transient coronary thrombo-embolism due to intraventricular stagnation and hypercoagulation. Certain findings including acute heart failure, malignant arrhytmogenesis and delayed or incomplete recovery of myocardial dysfunction likely suggests a MINOCA-TTS combination [1, 3, 4]

Taken together, the present case [1] might be regarded as a co-existence of true TTS and MINOCA. Accordingly, acute coronary ischemia manifesting as an ST segment elevation in the inferior leads [1] (possibly due to MINOCA associated with right or circumflex coronary artery) might have led to a classical TTS episode, and possibly; vice versa. Incomplete recovery of systolic functions on echocardiogram might also substantiate this co-existence in the patient [1].

Finally, the presence of one or more of the below-mentioned conditions possibly suggests MINOCA (in isolation or co-existing with TTS) rather than TTS in isolation, and hence; warrants further diagnostic strategies including cardiac magnetic resonance imaging (MRI) and/or advanced coronary modalities (intravascular ultrasound (IVUS), provocative tests with methylergonovine, etc.) to confirm the MINOCA diagnosis, and to identify the specific MINOCA trigger [1–4]:

- Incomplete, failed or delayed recovery of myocardial dysfunction [1, 3, 4],
- Suspected ‘pseudo-TTS’ pattern [3, 4],

— Acute heart failure and/or malignant arrhythmogenesis [3, 4]
— Overt CAG findings suggestive of MINOCA including dissection flap and severe CSF [2,3] (these only warrant MRI, coronary modalities may be unnecessary).

In conclusion, the differentiation between MINOCA and TTS may be quite challenging [1, 3, 4]. Moreover, these two conditions may also co-exist in certain settings [1,3,4]. However, meticulous evaluation of clinical clues and pitfalls, on a case by case basis, might be of diagnostic value, and might also help determine the need for advanced coronary modalities and cardiac MRI.

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