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

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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 Łoboz-Rudnicka et al. [1] has described a patient with MINOCA who was initially diagnosed as having a classical TTS episode. Accordingly, we would like to highlight certain pitfalls and clues in the differentiation between MINOCA, TTS, and the 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 the left anterior descending (LAD) and right coronary arteries (RCA). At first glance, this might denote co-existing or isolated "true TTS" in the patient given the normal distribution of her coronary arteries (each coronary artery perfusing its own territory) on coronary angiogram (CAG). Importantly, recovery from a "pseudo-TTS" pattern exhibits a potential correlation with the mitigation of myocardial ischemia [3, 4]. Therefore, was the recovery from wall motion abnormalities predominantly spontaneous, or was it 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, 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, a meticulous search for 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 common triggers, including an 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 thromboembolism due to intraventricular stagnation and hypercoagulation. Some clinical situations including acute heart failure, maligant arrythmias, and delayed or incomplete recovery from myocardial dysfunction suggest a likelihood of 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 ST-segment elevation in the inferior leads [1] (possibly due to MINOCA associated with the 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]. These conditions are

- Incomplete, failed, or delayed recovery of myocardial dysfunction [1, 3, 4],
- A 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.

Article information

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