

Acute anterior myocardial infarction complicated by takotsubo syndrome: the value of multimodality imaging

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A 75-year-old man was admitted to an emergency department, complaining of chest pain persisting for 7 hours. The 12-lead electrocardiogram (ECG) showed ST-segment elevations in leads V₃ through V₆ and decreased R waves in leads V₄ through V₆ (FIGURE 1A, Day 0), compared with the past ECG (FIGURE 1A, Past), which suggested anterior acute myocardial infarction (AMI). Transthoracic echocardiography (TTE) demonstrated a severely hypokinetic anterior left ventricular (LV) wall, especially in the apex, and a hyperkinetic basal inferolateral LV wall (FIGURE 1B and 1C). On hospital admission, laboratory tests showed a white blood cell count of $10.1 \times 10^3/\text{mm}^3$, creatine kinase level of 1278 IU/l, creatine kinase-MB level of 165.4 IU/l, troponin T level >2000 ng/l, and brain natriuretic peptide level of 150 pg/ml. Emergency coronary angiography revealed a 99% stenosis of the proximal left anterior descending artery (FIGURE 1D) without significant stenoses in the other epicardial coronary arteries. After stent implantation, grade 3 Thrombolysis in Myocardial Infarction flow was achieved (Supplementary material, Figure S1). Follow-up ECGs showed negative T waves with QT prolongation in leads V₂ through V₆ on Day 2, which improved within several days (FIGURE 1A). Giant negative T waves with QT prolongation re-emerged in leads V₄ through V₆ on Day 13 and gradually disappeared over 90 days. The exact values of the corrected QT interval on days 0, 2, and 90 were 0.39, 0.68, and 0.4 s, respectively. Serial TTE showed a notable improvement of anterior LV wall motion on day 3 and almost no asynergy on day 14 (Supplementary material, Figure

S1). On dual scintigraphy combined with computed tomography on day 6, the ¹²³I-β-methyl-iodophenyl pentadecanoic acid images showed more extensive myocardial metabolic abnormalities (FIGURE 1E) compared with the ²⁰¹thallium images (FIGURE 1F). This patient might have complicated takotsubo syndrome (TTS) after anterior AMI for the following reasons. First, the serial ECG changes could not be explained by anterior AMI alone, while resurgent giant negative T waves at 2 to 3 weeks were typical of TTS.¹ Compared with the previous ECG, the ECG on admission showed ST-segment elevations in inferior leads and absence of ST-segment depressions except in lead aV_R, which were more often observed in TTS than in anterior AMI.^{2,3} Second, the biomarkers of myocardial ischemia were disproportionately low, considering the wide range of myocardial injury detected by scintigraphy. Third, TTE showed that the asynergy observed in the anterior wall rapidly improved within 14 days. According to the InterTAK criteria, the score in this patient was calculated as 42, which indicated that he was diagnosed with TTS with a 89% sensitivity and a 91% specificity.³ The scoring items included the physical trigger, absence of ST-segment depression (except in lead aV_R), chronic psychiatric stress, and corrected QT interval prolongation. To our knowledge, this is the first case of anterior AMI complicated by TTS illustrated by multimodality assessment, despite challenges to demonstrate the coexistence of these clinical conditions. Takotsubo syndrome after AMI might be a result of great stress and catecholaminergic activation caused by AMI. Since

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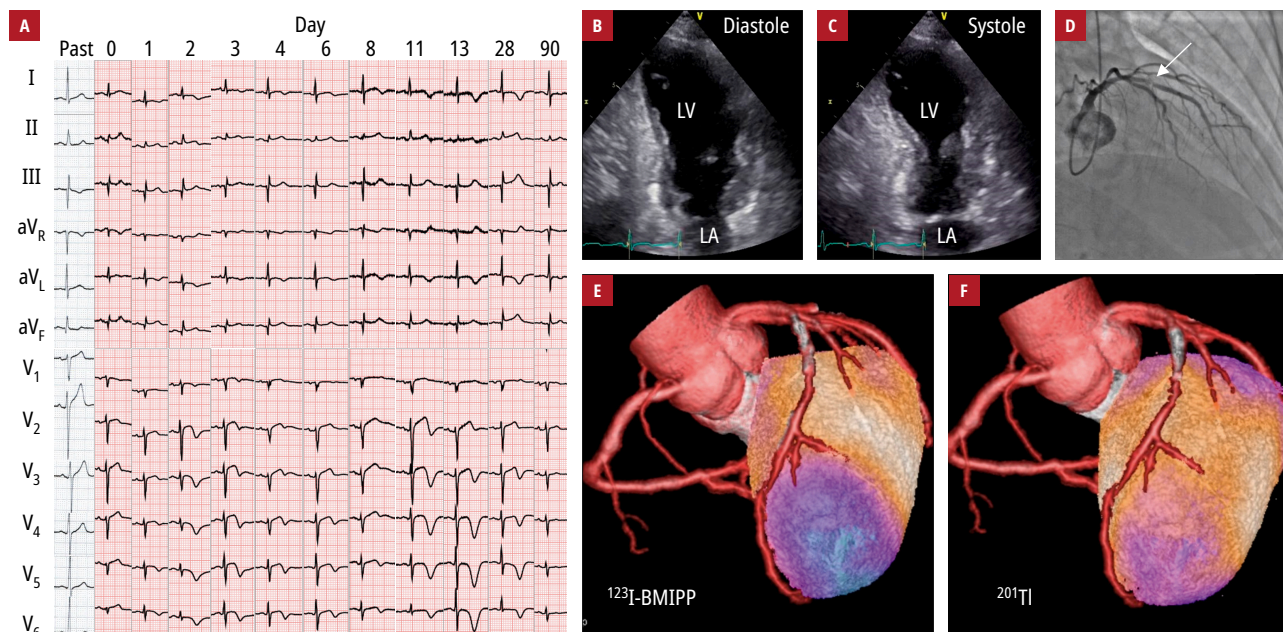


FIGURE 1 **A** – serial electrocardiographic changes; **B, C** – transthoracic echocardiography on hospital admission in diastole (**B**) and systole (**C**), showing a severely hypokinetic apical left ventricular wall and a hyperkinetic basal left ventricular wall; **D** – initial coronary angiography showing a 99% stenosis at the proximal left anterior descending artery (arrow); **E, F** – dual scintigraphy combined with computed tomography using ^{123}I - β -methyl-iodophenyl pentadecanoic acid (showing metabolism; **E**) and ^{201}Tl (showing perfusion; **F**) suggesting a perfusion–metabolism mismatch
Abbreviations: LV, left ventricle; LA, left atrium; ^{123}I -BMIPP, ^{123}I - β -methyl-iodophenyl pentadecanoic acid; ^{201}Tl , ^{201}Tl

the perfusion–metabolism mismatch reflects the metabolically impaired but viable myocardial area after AMI,⁴ scintigraphic imaging, in this case, was consistent with the subsequent functional recovery observed on serial TTE, although this mismatch is not specific for TTS. Further investigations including cardiac magnetic resonance imaging would be useful in establishing the diagnosis.⁵ These observations will help to increase awareness of the possible coexistence of takotsubo syndrome among patients with AMI.

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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