A 70 year old woman was admitted to our hospital on March 20, 2014 due to suspected acute myocardial infarction. Her ECG showed ST elevation in lead V1 and V2 at her family physician’s clinic (Fig. 1). Emergency coronary angiography (CAG) showed normal coronary. However, left ventriculography (LVG) showed severely reduced wall motion in anteroseptum (Fig. 2). LVG did not show apical ballooning. The ECG showed inverted T waves in lead V1 to V5 on next day. Peak creatinine kinase was 153 IU/L at 12 hours after the onset of chest pain. Five days after the admission, the patient had rest nuclear cardiology imaging. Rest 201thallium showed normal perfusion at rest. In contrast, rest 123I–β-methyl-p-iodophenyl-pentadecanoic acid (BMIPP) showed moderate intensity of defect in anterior to septum (Fig. 3). Thus, there was perfusion and fatty acid mismatch in anterior. This 123I BMIPP finding agreed with ECG changes and LVG findings. Although, we did not perform acetylcholine provocation, the acute chest pain syndrome might be associated with vasospastic angina (1,2). Takotubo cardiomyopathy was also another possible causes of this chest pain syndrome (3). However, LVG findings did not agree with the typical apical ballooning pattern. In the current case, 123I BMIPP might accurately detect stunned myocardium related to acute chest pain syndrome (1,2,4,5).

Fig. 1 Serial electrocardiographic changes

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Fig. 2  Coronary angiography  
(A) Left ascending artery, left circumflex, and right coronary artery showed normal coronary arteries.  
(B) Left ventriculography showed severe wall motion abnormality in anteroseptal wall.

Fig. 3  
(A) Rest $^{201}$thallium myocardial perfusion imaging showed mild intensity of perfusion defect in inferior due to diaphragmatic attenuation artifact.  
(B) Rest $^{123}$I-beta-methyl-p-iodophenyl-pentadecanoic acid showed moderate intensity of defect in anterior and anteroseptum. There is perfusion and metabolism mismatch in anterior and septum.
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Conflicts of Interest
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References