Case Report

A case report: 201Tl/99mTc-Pyrophosphate dual-isotope myocardial SPECT for detecting annular subendocardial infarction induced by a transient shock

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**Summary** The patient was a 74-year-old woman with angina pectoris, who had undergone percutaneous coronary intervention with stent placement in the right coronary artery on October 2, 2007. On November 12 of the same year, she suffered from paroxysmal atrial fibrillation. She was treated with pilsicainide hydrochloride administered by intravenous injection, which was followed by a sudden sinus standstill, with marked bradycardia and a shock state. The patient was then treated with a catecholamine, however, the shock state persisted for about an hour. An electrocardiogram revealed persistent ST depression in leads V4–6 along with elevation of the serum creatinine kinase. A coronary angiography performed on the admission day revealed no abnormality. On the third hospital day, a dual-isotope myocardial SPECT using 201Tl and 99mTc-pyrophosphate demonstrated an annular accumulation of 99mTc-pyrophosphate concordant with the endocardium from apex to the mid-portion of the left ventricle, suggestive of subendocardial infarction. The case is reported here, as there are few reports of subendocardial infarction developing due to ischemia arising from a shock state. © 2010 Japanese College of Cardiology. Published by Elsevier Ireland Ltd. All rights reserved.

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Introduction

Subendocardial infarction developing as a result of ischemia associated with a shock state is not a common occurrence. In virtually all cases, subendocardial infarction develops due to progression of coronary arteriosclerosis or as a complication of percutaneous coronary intervention. In such instances the infarction arises locally in the region supplied by the affected coronary arterial branch. Annular subendocardial infarction has been reported in disorders associated with extensive myocardial ischemia, such as multi-vessel spasm, severe triple-vessel lesions causing severe stenosis, or aortic stenosis, and is rare [1–4]. We recently encountered a rare case of annular subendocardial infarction which developed during a transient shock state in the patient and was successfully diagnosed by myocardial SPECT with 99mTc-pyrophosphate. Herein is a report of the case.

Case report

The patient, a 74-year-old woman, had diabetes mellitus, hypertension, and hyperlipidemia. On June 12, 2007, she had an onset of paroxysmal atrial fibrillation and was treated in our hospital. Subsequent medical workup revealed myocardial ischemia, and stenting was undertaken of a severe stenosis lesion in the distal portion of the right coronary artery on October 2, 2007, with consequent amelioration. On the night of November 12, she complained of persistent chest discomfort. Examination revealed an episode of paroxysmal atrial fibrillation (Fig. 1B), and the patient was treated by intravenous injection of the anti-arrhythmic agent, pilsicainide hydrochloride for defibrillation. This treatment was followed by a sudden sinus standstill, associated with marked bradycardia (Fig. 1C) and a shock state. An electrocardiogram (ECG) showed sinus standstill with supraventricular escape beats (heart rate, 40 bpm). The patient was treated with atropine sulfate and a catecholamine, however, her systolic blood pressure remained in the hypotensive range, at 60 mm Hg. The patient was free of symptoms during this hypotensive state in rest position. After about an hour of onset of this event, her systolic blood pressure returned to normal level and sinus rhythm was restored, but as can be seen from Fig. 1, ECG demonstrated persistent ST depression in leads V4–6 compared to previous ECG (Fig. 1A and D) and the patient was admitted to the hospital to be under observation. There were no specific symptoms and the general condition of the patient remained stable. However, the ST segment depression remained persistent, with elevation of the peak serum creatinine kinase (1797 IU/L; normal: ≤180 IU/L), creatinine kinase-MB (146 ng/mL; normal: ≤5.0 ng/mL), and troponin T (7.17 ng/mL; normal: ≤0.10 ng/mL) levels. As these findings were indicative of possible thrombosis of the stent that had been placed about 1 month previously, a coronary angiography was performed on the day of admission. The coronary angiography, however, did not reveal any abnormality (Fig. 2). On the third hospital day, a dual-isotope myocardial SPECT using 201Tl and 99mTc-pyrophosphate was undertaken. Photopeaks were 70 keV with 20% window for 201Tl and 140 keV with 15% window for 99mTc-pyrophosphate and cross-talk correction was used in reconstruction. These short-axis SPECT images are presented in Fig. 3. A mild decrease in the uptake was noted in the apical portion on the 201Tl myocardial SPECT images (Fig. 3A). Myocardial SPECT images obtained with 99mTc-pyrophosphate showed uptake in areas extending from the apex to the basal-portion, with an annular accumulation of 99mTc-pyrophosphate corresponding to the endocardium from the apex to the mid-portion; a diagnosis of subendocardial infarction was thus made (Fig. 3B). The patient had a practically symptom-free, uneventful course at the hospital until discharged. With echocardiography before discharge, the left ventricular contractile function was normal (ejection fraction: 60%) without enlarged size. The mild left ventricular diastolic dysfunction was preexisting, but did not become worse (E/A ratio: 0.69 and deceleration time: 252 ms). We suspected that this dysfunction was caused by hypertensive myocardial hypertrophy and age. These findings were similar in chronic stage.

Discussion

Subendocardial infarction that occurs due to ischemia associated with a shock state is not a common occurrence; however, we recently encountered a rare case of annular subendocardial infarction which was thought to have developed during a transient shock state and was diagnosed by myocardial SPECT with 99mTc-pyrophosphate. The dual-isotope myocardial SPECT was performed using 201Tl and 99mTc-pyrophosphate and the scans in Fig. 3A and B are of the same magnification. The annular radioisotope uptake seen at the mid-portion of the left ventricle on the 99mTc-pyrophosphate myocardial SPECT is situated at the circumference just medial to the short-axis image of 201Tl myocardial SPECT. From these findings, we diagnosed as representing annular subendocardial infarction. Such clearly delineated annular uptake has never been demonstrated on 99mTc-pyrophosphate myocardial SPECT in any of the previously reported cases of annular subendocardial infarction [1–4].

According to reports of annular subendocardial infarction diagnosed by 99mTc-pyrophosphate myocardial SPECT, the causes include multi-vessel coronary spasm, severe triple-vessel lesions of the coronary arterial tree causing a severe stenosis, and aortic stenosis [1–4]. These are all valid as pathophysiological states in which extensive myocardial ischemia may occur. In the present case, however, none of these was considered as the cause of the annular subendocardial infarction, and it seemed very likely that the myocardial ischemia was caused by the prolonged transient shock state that occurred as an adverse reaction to pilsicainide hydrochloride.

Pilsicainide hydrochloride is an anti-arrhythmic agent, which acts by inducing blockade of the sodium channels of the cell membrane. Bradyarrhythmias are included as adverse reactions of this agent. In the present case, we needed to administer a short catecholamine infusion for bradycardia and hypotension induced by intravenous injection of this agent. The hypotension persisted for about an hour, but was eventually complicated unexpectedly by causing subendocardial infarction. We could probably have prevented this complication by being more aggressive in...
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Figure 1  (A) The electrocardiograms during sinus rhythm before admission about 1 month previously. (B) Paroxysmal atrial fibrillation on emergency room visit. (C) Bradycardia after administration of pilsicainide hydrochloride. (D) Sinus rhythm after recovery of bradycardia. The electrocardiograms after recovery of bradycardia demonstrated ST depression in leads V₄₋₆.

managing the blood pressure while the patient was in a shock state.

In a shock state, the sympathetic nervous system is activated, giving rise to vasoconstriction via the vascular smooth muscle α₁ receptor, which results in decreased peripheral blood flow [5,6]. Oligemia occurs initially in the skin and skeletal muscles, because vascular resistance varies from organ to organ, so as to maintain the blood flow, to the maximum extent possible, to vital organs such as the brain and heart. In the present case, however, subendocardial infarction alone developed without apparent damage to any other viscera. One possible mechanism of this is the presence of coronary microangiopathy, which is often recognized in the diabetic patient [7]. It is likely that the subendocardial tissues were exposed to ischemia due to hypotension in the presence of coronary microangiopathy. However, the pathophysiology of coronary microangiopathy is still unknown, and there is no supporting report of this mechanism. Other possible mechanisms are catecholamine-induced cardiomyopathy as typified by Takotsubo cardiomyopathy or multiple coronary artery spasm. There was a possibility that these mechanisms occurred without decreased left ventricle wall motion.

Myocardial SPECT obtained with ⁹⁹ᵐTc-pyrophosphate is a particularly useful diagnostic imaging method to confirm the presence of subendocardial infarction or other types of MI. However, it is difficult to perform myocardial scintigraphy on a patient in a shock state because the general condition of such a patient is unfavorable in most such instances. Possibly owing to limitation of this diagnostic modality, there have been no reported cases of subendocardial infarction developing in a shock state. Furthermore, dual-isotope imaging was reported to have a technical limitation. The quality of this imaging is degraded by cross-talk correlation [8]. In spite

Figure 2  The coronary angiography during persistent ST depression in leads V₄₋₆ did not reveal any abnormality.
Figure 3 201Tl myocardial SPECT images showed a mild uptake decrease in the apical portion (A). 99mTc-pyrophosphate myocardial SPECT images showed clearly delineated annular uptake corresponding to the endocardium from the apex to the mid-portion of the left ventricle (B). These scans are the same magnification percentage.

of these limitations, we were successful in clearly delineating the annular subendocardial infarction by imaging in the present case.

References