CASE REPORT

Adenosine-induced coronary vasospasm following drug-eluting stent implantation

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SUMMARY

We present the case of coronary vasospasm during adenosine stress in a patient with a prior drug-eluting stent implantation. The patient had a stent implantation in the left anterior descending coronary artery 3 years ago. Recently, he developed a chest pain and underwent adenosine stress myocardial perfusion single photon emission CT (SPECT). During the adenosine stress, he felt severe chest pain and ST elevation on electrocardiogram. An invasive coronary angiography showed no in-stent restenosis. This phenomenon deemed to be adenosine-induced coronary vasospasm after stent implantation.

BACKGROUND

Adenosine stress myocardial perfusion single photon emission CT (SPECT) is a common procedure in clinical settings. It visualises relative coronary flow reserve in each vessel or induced ischaemia by coronary steal phenomenon. It increases myocardial blood flow immediately during the infusion and its half-life seems to be less than 10 s. Therefore, it would be performed safely in patients with coronary artery disease.

CASE PRESENTATION

A 52-year-old man with longstanding history of smoking was referred to our hospital due to non-anginal chest pain. He had no history of myocardial infarction and any medications. ²⁰¹Tl at rest and adenosine at stress ^{99m}Tc-tetrofosmin myocardial perfusion SPECT were performed. The SPECT image showed reversible perfusion defect in the left anterior descending (LAD) coronary artery territory, therefore he had an invasive coronary angiography which revealed severe stenosis in the proximal LAD. Subsequently, he received a coronary intervention into the proximal LAD using TAXUS (2.5×20 mm, Boston Scientific Corporation, Natick, Massachusetts, USA) and stopped smoking. After 3 years from the intervention, he felt chest discomfort again and had a follow-up stress ^{99m}Tc-tetrofosmin SPECT. His blood pressure was 126/85 mm Hg at rest and 122/79 mm Hg after the stress, and prestress and post-stress heart rate were 59/min and 81/min, respectively. Resting ECG was normal. He developed a worsening chest pain and ST elevation in I, aVL, V2–4, and reciprocal change in III, aVF on ECG during adenosine infusion (figures 1 and 2). Subsequently, he received sublingual nitroglycerine. His chest pain and ST elevation were immediately resolved. These ECG changes started at the early

Figure 1 Electrocardiogram (I, II, III, aVR, aVL, aVF), ST elevation in I and aVL and ST depression in III and aVF (arrows).

phase of adenosine stress and did not resolve immediately after stopping adenosine infusion. The SPECT image demonstrated a small amount of jeopardised myocardium in the mid to distal anterior wall extending to the apex (figure 3A). He performed an invasive coronary angiography 3 days after SPECT, which showed no in-stent restenosis in the LAD (figure 3B C). During catheter procedures, two distal portions of LAD were spastic and showed 100% obstruction without acetylcholine provocation test, therefore intracoronary injections of isosorbide dinitrate and nicorandil were immediately administered.

Figure 2 Electrocardiogram (V1–6) and ST elevation in V2–4 (arrows).

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Figure 3 (A) Stress myocardial perfusion single photon emission CT which shows a small amount of jeopardised myocardium in anterior wall extending to the apex (arrows), (B) left coronary angiogram which shows no in-stent restenosis, (C) right coronary artery which shows no significant stenosis.

OUTCOME AND FOLLOW-UP

Coronary vasospasm in this patient was under control.

DISCUSSION

Unambiguously, coronary vasospasm during adenosine infusion is a rare reaction in patients with coronary artery disease.^{1–3} Coronary vasospasm may occur by regional coronary over-constriction due to dysfunction of (1) vascular endothelium and/or (2) smooth muscle cells.^{4–5} Adenosine causes coronary hyperaemia in activating ATP-dependent K^+ channel (K_{ATP}) via adenosine A_2 receptor in coronary smooth muscle cells.⁶ Marban *et al*⁷ speculated that abnormalities in K_{ATP} channels might cause paradoxical reaction by adenosine stress.⁸ Recently, coronary vasospasm due to endothelial dysfunction after drug-eluting stent (DES) implantation is documented.⁹ Endothelial dysfunction in coronary arteries may be explained by (1) delayed endothelial recovery by DES, (2) direct actions of the eluting drug and (3) hypersensitivity to the polymer of DES. In the present case, SPECT depicted a small amount of jeopardised myocardium in the LAD territory, which was speculated by spastic coronary obstruction of LAD in the following coronary angiography. Another possibility of jeopardised myocardium would be microvascular dysfunction or poor coronary flow reserve in the LAD. Actually, the patient showed adenosine-induced coronary vasospasm after 3 years of DES implantation. Therefore, we speculate that abnormal reactions of coronary smooth muscle cell may be the possible deductions rather than endothelial dysfunction by DES.

DES implantation is, nowadays, a common procedure in patients with a significant coronary artery disease. Non-invasive

stress test including SPECT would be performed for the evaluation of restenosis during the follow-up. In conclusion, we need to pay attention to the paradoxical reaction of adenosine not only during the stress but also at poststress phase (at least a couple of minutes).

Learning points

- ▶ Adenosine usually acts as a vasodilator agent. Adenosine stress rarely provokes coronary vasospasm regardless of whether patients received stent implantation or not.
- ▶ Therefore, at least 2 min of observation after adenosine stress is required.
- ▶ This unexpected phenomenon suggests the abnormalities of K_{ATP} channels.

Competing interests None.

Patient consent Obtained.

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