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Coronary vasospasm during a regadenoson stress test

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A 43 year-old male was referred for pharmacological stress testing in 2010 in the setting of increasing dyspnea on exertion and atypical chest pain. He had been diagnosed with hypertrophic cardiomyopathy following a ventricular fibrillation arrest in 2004. At that time, he underwent a negative evaluation for coronary atherosclerosis and intracardiac defibrillator implantation, and had done well since. Upon regadenoson injection (0.4 mg over 10 s) he became tachycardic, diaphoretic, and complained of severe chest tightness. His ECG showed exaggeration of baseline abnormalities, non-specific for ischemia (Figs. 1, 2). He was given aminophylline (150 mg intravenous) with resolution of his symptoms. Myocardial imaging revealed a moderate to large-sized reversible defect involving the anteroseptal, anterior, and inferolateral walls, as well as stress-associated left ventricular dilation (Fig. 3). Subsequent coronary angiography was negative for significant coronary artery disease or anatomical coronary anomalies (Fig. 4).

Adenosine and its analogs provoke coronary vasodilation via activation of A_{2A} type adenosine receptors, and are useful as pharmacological stress agents by exploiting flow-reserve differences between normal and atherosclerotic coronary artery segments [1]. However, coronary vasospasm is a well-known occurrence of adenosine administration, and milder side effects are frequently encountered, likely related to the activation of non-A_{2A} type receptors [2].

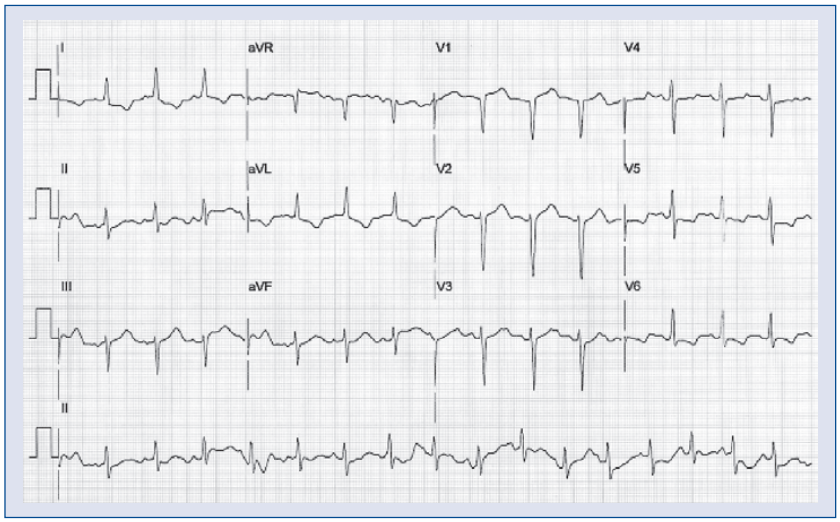

Figure 1. Electrocardiogram demonstrating sinus rhythm with baseline ST-T wave abnormalities.

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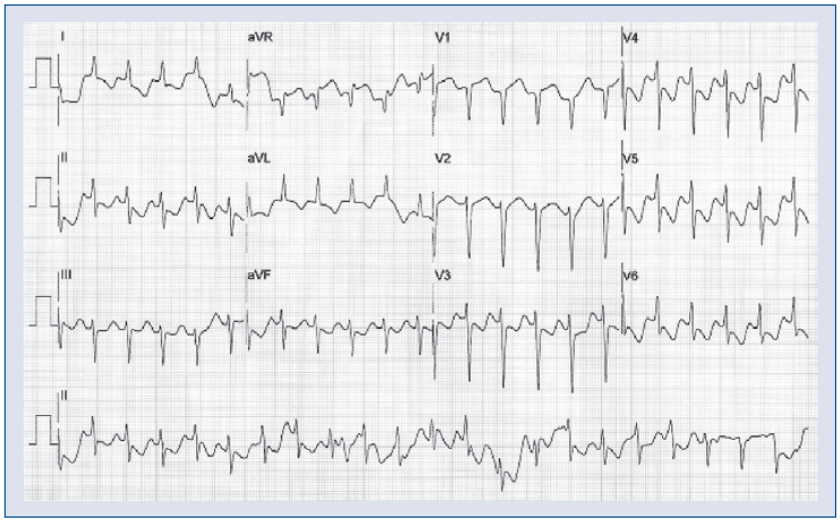

Figure 2. Electrocardiogram at 17 s of recovery after regadenoson injection. Non-specific for ischemia given baseline abnormalities.

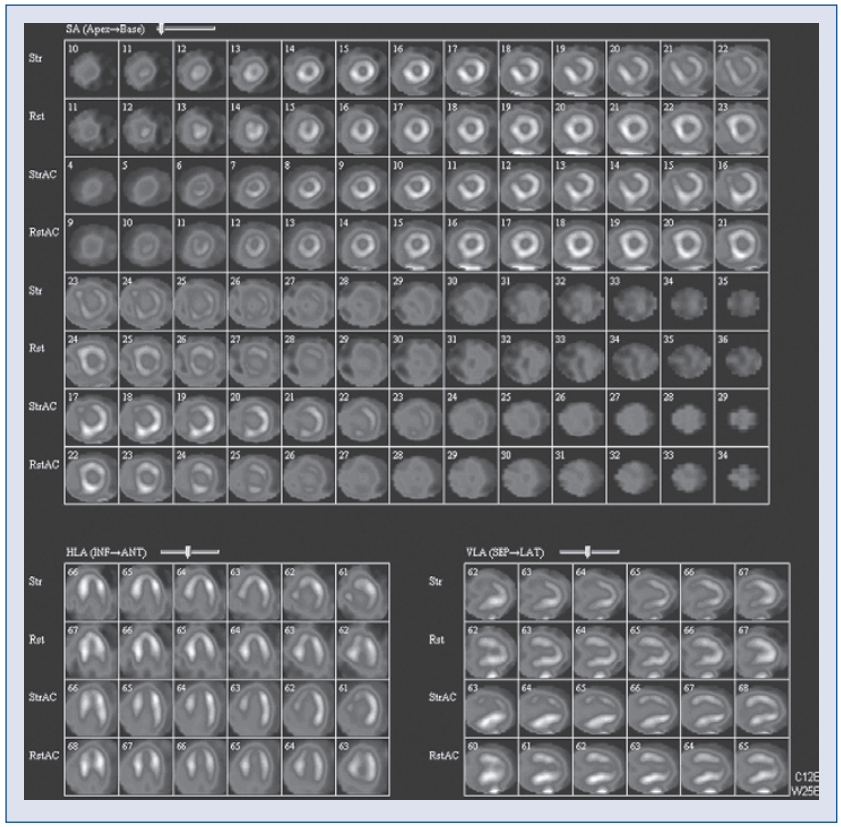

Figure 3. Myocardial perfusion images obtained after regadenoson injection demonstrating perfusion defects in a multi-vessel distribution.

Regadenoson is a newer pharmacologic stress agent with a ten-fold higher selectivity for the A_{2A} adenosine receptor. With comparable efficacy in the detection of ischemia, regadenoson is also associated with fewer side effects than adenosine [3]. To the best of our knowledge, there have been no previous reports of regadenoson-associated coronary vasospasm.

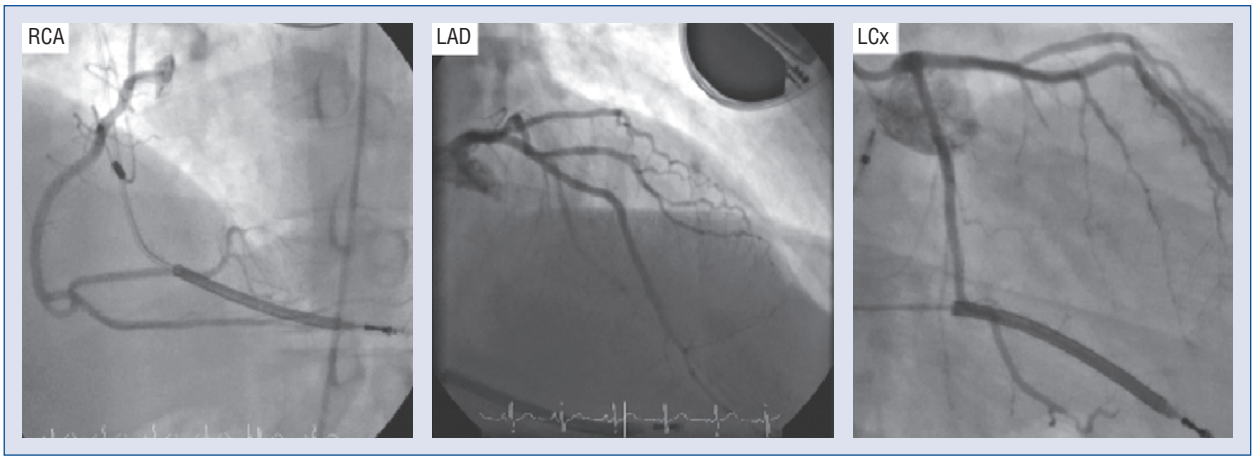

Figure 4. Coronary angiography depicting the absence of atherosclerosis; RCA — right coronary artery; LAD — left anterior descending artery; LCx — left circumflex artery.

In this case, the occurrence of clinical angina with a temporary perfusion defect, in the absence of coronary atherosclerosis, is consistent with acute coronary vasospasm. Regadenoson-mediated adenosine receptor activation as the provocateur of coronary vasospasm in this instance is substantiated by the temporal relationship between the perfusion defect at the onset and subsequent pharmacologic reversal of adenosine receptor activation.

The mechanism of coronary vasospasm upon adenosine receptor activation remains poorly understood. Proposed mechanisms include genetic differences in proteins downstream of the clinical target (A_{2A} receptor), such as the vascular K_{ATP} channel [4], or activation of non-A_{2A} adenosine receptors [5]. The present case supports the notion that this phenomenon is probably a consequence of A_{2A} adenosine receptor, and not off-target receptor, activation.

As we gain more clinical experience with adenosine analogs, the comparative incidence of vasospasm between agents with different selectivities for the A_{2A} receptor may yield additional mechanistic insights.

For the present time, we suggest that the practitioner be aware of this possible complication, and be prepared to provide receptor antagonism should signs of myocardial ischemia develop upon regadenoson administration.

Conflict of interest: none declared

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