Atrial Arrhythmias and Scintigraphic “D-shape” Sign in Pulmonary Artery Hypertension

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Abstract

Pulmonary hypertension significantly changes biventricular anatomy and physiology, frequently evolving to clinical deterioration and right ventricular failure. The case of a woman developing atrial arrhythmias complicating dipyridamole stress in concomitance with scintigraphic “D-shaped” left ventricle is briefly reported. Although rare, our finding may suggest that nonselective vasodilators should be used with caution in this clinical setting.

Keywords: Arrhythmias, “D shaped,” pulmonary hypertension

Introduction

Pulmonary hypertension (PH) determines dramatic biventricular remodeling and several physiological modifications of right ventricular function.[1] In atrial septal defect (ASD), moderate/severe PH frequently marks an elevated risk of supraventricular tachyarrhythmias as atrial flutter, atrial fibrillation, and atrioventricular nodal reentry tachycardia.[2] The development of these arrhythmias is frequently accompanied by clinical deterioration and right ventricular failure.[3]

We describe the development of unusual fast supraventricular arrhythmias complicating vasodilator stress in concomitance of scintigraphic “D-shaped” left ventricle (LV), highlighting that dipyridamole should be used with special caution in this clinical context.

Case Report

A 77-year-old hypertensive woman with noncorrected ASD (ostium secundum) and moderate PH was referred to vasodilator stress Tc-99m sestamibi gated-single-photon emission computed tomography (SPECT) (2 days stress/rest protocol) due to nonanginal chest pain superimposed to class III dyspnea. She was under aspirin, losartan, and atenolol after previous nonembolic recurrent episodes of atrial flutter. Atenolol was discontinued 48 h before stress. Laboratory tests were normal.

After dipyridamole infusion (0.56 mg/kg), the patient installed tachycardia-dependent left bundle branch block followed by atrial flutter at 175 bpm (A) and atrial fibrillation 2 min after aminophylline administration [Figure 1]. Standard loading dose of amiodarone was then required. Poststress nongated SPECT images revealed marked right ventricle enlargement and “D-shaped” LV [Figure 2]. LV perfusion was normal.

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How to cite this article: Ferrando-Castagnetto F, Ricca-Mallada R, Selios V, Ferrando R. Atrial arrhythmias and scintigraphic “D-shape” sign in pulmonary artery hypertension. World J Nucl Med 2017;16:75-7.
“D-shaped” remodeling of LV has been extensively described in acute or chronic right ventricle overload,[4] although data refer almost exclusively to echocardiographic examination.[5,6] In fact, most information about this anatomical finding in myocardial scintigraphy is limited to isolated reports and small case series.[7,8] In an observational retrospective report, Movahed et al. found that “D-shaped” LV in SPECT imaging correlated well with right ventricle overload; a history of ASD was found in 38% of this sample.[9]

It is difficult to find a plausible mechanism explaining electrical complications induced by dipyridamole in the clinical setting we described. In fact, an increase in intracellular adenosine levels is more likely to reduce the occurrence of atrial arrhythmias instead of precipitating it, there were no ionic or metabolic disorders, and perfusion imaging tended to exclude important potential ischemia as a precipitating event. However, we cannot exclude the possibility that beta-blockers discontinuation could have played an important role in the development of arrhythmias.

As phenotypic predisposition to electrical complications during vasodilator stress in patients exhibiting scintigraphic “D-shaped” LV is still unknown, inverted (rest–stress) protocol could not be systematically indicated in all patients with PH derived to SPECT. Safety profiles of dipyridamole, adenosine, and new selective A2A-receptor agonists (e.g., regadenoson) should be compared in patients with PH derived to myocardial perfusion imaging.

**Financial support and sponsorship**
Nil.

**Conflicts of interest**
There are no conflicts of interest.

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