Dobutamine stress echocardiography related sustained ventricular tachycardia in a patient with ischaemic cardiomyopathy

Amrit S Lota, Neda Noroozian, Steven Zaw and Ihab S Ramzy

Cardiology Department, Northwick Park Hospital, Middlesex, UK

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A 66 year-old Asian man; with a complex history of ischaemic heart disease presented with cardiac and troponin negative chest pain. His ECG showed sinus bradycardia with old left bundle branch block. The transthoracic echocardiography showed severely impaired left ventricular systolic function (EF 30-35%). The patient had had coronary artery bypass grafting at age of 42 years and remained asymptomatic until age of 56 years when he presented with incessant ventricular tachycardia requiring amiodarone, lidocaine and electrical cardioversion. Coronary angiography at that time showed occluded vein grafts to the circumflex and diagonal arteries. The right coronary artery (RCA) was small and received collaterals from the left system. A myocardial perfusion scan showed extensive inferolateral infarction but no evidence for reversible ischaemia. An implantable cardiac defibrillator (ICD) was thus inserted.

Over the following years, the patient underwent multiple PCI procedures with rotational atherectomy to the native LAD and associated vein graft, but subsequently developed in-stent restenosis. He also had PCI to the native RCA. He declined a minimally invasive grafting of the left internal mammary artery (LIMA) to the LAD and re-implantation of a new ICD after ex-plantation of the original one due to sepsis. His coronary artery disease remained stable following a recent admission with Troponin positive event which was confirmed by a repeat angiogram, therefore, had up-titrated anti-anginal medications. A low dose beta-blockade was discontinued due to documented sinus pauses up to 3 seconds. Following the recent admission, a joint cardiology-cardiothoracic meeting discussion recommended a stress echocardiogram with high-dose dobutamine to guide towards considering re-do bypass grafting.

A dobutamine stress echocardiogram (DSE) was performed (40 mcg/kg/min + 300 mcg atropine) by an experienced operator and the patient achieved 84% of target heart rate. At peak stress, he experienced hot sensation in the chest for which he was given GTN spray. Study analysis confirmed no evidence of inducible ischaemia (Figure 1), but three minutes into recovery, he developed sustained ventricular tachycardia at a rate of 218 bpm (Figure 2), but without obvious haemodynamic compromise. Intravenous metoprolol and amiodarone were administered and sinus rhythm was restored after approximately twenty minutes. The patient was observed overnight in CCU, and discharged few days later with a plan to refractory angina clinic referral in a tertiary centre, and to be assessed by an electrophysiologist for potential device therapy.

Figure 1: Apical 2-chamber view at rest and peak stress with contrast for IV cavity opacification.
Discussion

Stress echocardiography has become widely accepted as a safe, reliable and cost-effective modality for the evaluation of patients with suspected myocardial ischaemia and for prognostic stratification in patients with known coronary artery disease. While the benefits of this non-invasive assessment tool are clearly apparent, related complications e.g. life-threatening ventricular arrhythmias could happen although very rare.

Arrhythmias occurring during DSE have been extensively investigated. One of the first large series to examine the safety of DSE found that in a group of 1118 patients, 65% had no arrhythmia, 23% had single premature atrial and ventricular complexes, 0.7% developed an atrial arrhythmia and overall there were no episodes of sustained ventricular tachycardia (VT) or ventricular fibrillation. In the 3.5% of patients with episodes of non-sustained VT, 60% had no other features to suggest myocardial ischaemia and therefore the prognostic significance of this finding was uncertain. It has been proposed that in this subtype of patient, VT may be attributed to the direct adrenergic arrhythmogenic effect of dobutamine through myocardial beta receptor stimulation or dobutamine-induced reduction in plasma potassium rather than true myocardial ischaemia. This is supported by the similar finding of exercise-induced VT in healthy hearts, which has been attributed to catecholamine-sensitive enhanced automaticity or catecholamine-related delayed after depolarisations.

More recent studies continued to document the safety of echocardiography with pharmacological or exercise stress. In 2005, the overall incidence of dobutamine induced sustained monomorphic VT (lasting more than 30 seconds) was found to be only 0.3% in a cohort of 2688 patients. It was shown that DSE-induced VT had no predictive value for the identification of coronary artery disease and conferred no adverse prognostic significance with a negative DSE study over the longer-term. On EPS, VT only occurred in two patients both of whom were known to have ischaemic heart disease with reduced left ventricular function and pre-existing ICD devices. However, other investigators have not identified left ventricular dysfunction as a predictor of DSE induced VT.

In conclusion, numerous studies have shown that episodes of DSE-induced VT are not related to the presence or severity of coronary artery disease and the prognostic significance of this finding remains unclear. In our case, the sustained VT was delayed by several minutes into recovery which makes it difficult to ascertain its direct relationship to dobutamine. Furthermore, our patient is known to have ischaemic myocardium, significant scarring as well as documented history of ventricular arrhythmia, in the past, requiring ICD implantation.

Correspondence to:
Dr Ihab Ramzy, MD MSc PhD
Northwick Park Hospital
Watford Road
Harrow
Middlesex HA1 3UJ
E-mail: ihab.ramzy@nhs.net

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