Myocardial Stunning Demonstrated by Adenosine Myocardial Perfusion Scintigraphy as a Predictor of Significant Coronary Artery Disease

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Abstract: Myocardial stunning may occur after a brief but profound episode of ischaemia that is insufficient to lead to myocyte necrosis but following which there is depression of myocyte function that persists for hours or days. It is thought to be caused by disordered intra-cellular calcium handling and, when seen after stress-induced myocardial ischemia, it indicates profound ischaemia and potentially an adverse prognosis.

INTRODUCTION

Myocardial perfusion scintigraphy (MPS) is effective and cost-effective for the diagnosis of coronary artery disease, for assessing prognosis in ischemic heart disease, for demonstrating global and regional left ventricular function and for detecting viable and hibernating myocardium [1]. Imaging after a stress injection of either of the technetium-99m tracers (MIBI and tetrofosmin) is performed 30 to 60 minutes after stress, meaning that the pattern of tracer uptake reflects stress perfusion and viability whereas the function from ECG-gated imaging reflects function after a short period of rest. If abnormal regional function is seen in the post-stress images that is not present in the later resting images then stunning can be assumed. Post-stress stunning is a predictor of cardiac events in patients with coronary artery disease [2] and post-stress left ventricular ejection fraction provides information on the likelihood of future cardiac death [3].

CASE REPORT

A 64 year old man attended a rapid access chest pain clinic following two episodes of exertional chest pain and breathlessness over the previous six months. He was obese (body mass index 45kg/m²), an ex-smoker and he was on treatment for hypertension and hyperlipidaemia. There were no significant findings on examination and his resting ECG was normal. He was unable to perform dynamic exercise because of his obesity and so pharmacological stress MPS was requested.

A high dose two-day protocol was used with 1000MBq of technetium-99m tetrofosmin injected for the stress study and 1000MBq for the rest study. Stress was performed using adenosine infusion at 140µg/kg/min combined with dynamic exercise to 75W on a bicycle ergometer provoking some chest discomfort but no ST segment changes. Pulse rose from 83 to 105/min and blood pressure from 190/100 to 210/100 mmHg. Emission tomograms were acquired in a standard fashion using a GE Healthcare Optima dual headed gamma camera with ECG gating and 16 frames per cardiac cycle.

The tomograms one hour after stress injection of tracer showed a dilated left ventricle with moderate reduction of tracer uptake in much of the antero-apical region and milder reduction in the septum. Uptake elsewhere was normal. Following a resting injection of tracer the left ventricle was smaller and the pattern of uptake returned almost to normal (Fig. **1**). ECG-gating of the post-stress tomograms showed left ventricular ejection fraction 46% with apical akinesis and paradoxical motion of the apical septum. Following a resting injection of tracer the left ventricle was smaller with ejection fraction 54% and almost normal motion (Fig. **2**). There was therefore an extensive inducible perfusion abnormality in the left anterior descending artery territory with almost normal left ventricular function at rest but stunning in the abnormally perfused territory one hour after stress.

Subsequent coronary angiography showed a 60% distal left main stem stenosis, a proximally occluded left anterior descending artery with retrograde filling from the right coronary artery, a 60% circumflex stenosis and a normal dominant right coronary artery. He was referred for aggressive management of his weight and other risk factors prior to coronary bypass grafting.

DISCUSSION

Myocardial stunning has been documented in animal models and in humans [4] and non-invasive imaging techniques have also shown the phenomenon in clinical settings [5,6]. Several features of ECG-gated MPS can suggest myocardial stunning including significantly lower ejection fraction measured from the post-stress images compared with the post-rest images [5], ventricular dilatation ("transient ischaemic dilatation") [6], and regional wall motion abnormalities after stress, as in this case. The most specific indicator is a transient myocardial motion or thickening abnormality since stress induced dilatation may also be the result of subendocardial ischaemia giving the images the appearance of a dilated ventricular cavity. Using ECG-gated imaging, left ventricular ejection fraction may be

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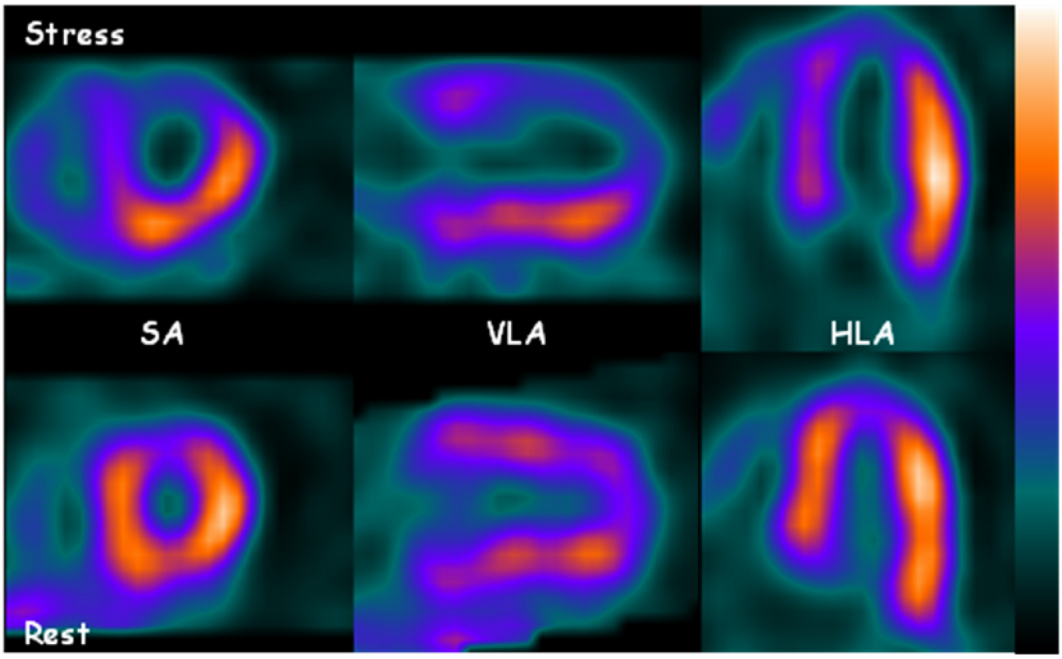

Fig. (1). Stress and rest myocardial perfusion tomograms in the short axis (SA), vertical long axis (VLA) and horizontal long axis (HLA). There is an inducible perfusion abnormality in the left anterior descending territory, with transient ischaemic dilation of the left ventricular cavity and apparent reduction in right ventricular counts in the rest images (indicating globally reduced left ventricular counts in the stress images). The images are slightly degraded by the patient's obesity.

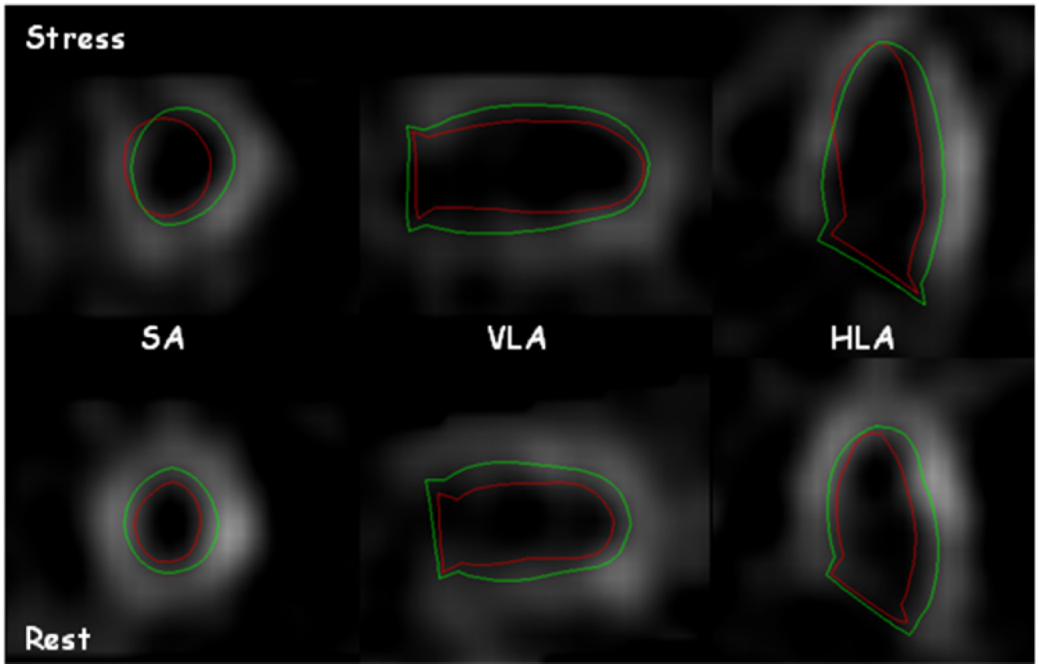

Fig. (2). End diastolic frames from the ECG-gated acquisition in the same orientations as figure 1 with endocardial contours superimposed (end diastole, green; end systole, red). The stress images represent function 45 minutes after completion of stress. The left ventricle is dilated with apical akinesis and paradoxical movement of the apical septum in the post-stress images but there is almost normal motion at rest.

reduced and end diastolic volume may be increased after exercise compared with that after a longer period of rest in patients with inducible myocardial ischaemia [5].

A transient decrease in left ventricular ejection fraction may occur after stress using dynamic exercise but not after pharmacological stress because vasodilator stress induces perfusion heterogeneity and not always frank ischaemia [7]. In this case however there was marked stunning after vasodilator stress and the phenomenon has only rarely been reported [8]. Although the vasodilator stress was combined with dynamic exercise to 75W the pulse and blood pressure response was not marked and would not normally have been sufficient to induce myocardial ischaemia in the absence of another mechanism. Myocardial ischaemia during vasodilator stress occurs in approximately one third of patients with inducible perfusion abnormalities and it is more

common in those with severe coronary stenosis or with coronary occlusion and collateral perfusion [9]. This case illustrates such a phenomenon.

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Received: October 23, 2007 Revised: December 13, 2007 Accepted: February 12, 2008