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High dose adenosine stress perfusion cardiovascular magnetic resonance (CMR) imaging for detection of coronary artery disease

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Introduction

CMR perfusion (CMRP) imaging using adenosine is increasingly used for the assessment of patients with known or suspected coronary artery disease. At a standard dose adenosine is well tolerated with a good safety profile but on occasion fails to produce a significant haemodynamic response. This may potentially lead to the misdiagnosis of significant ischaemia. We therefore examined whether using a higher dose in these patients to induce a haemodynamic response would lead to the identification of additional perfusion defects.

Methods

We prospectively recruited from 1230 consecutive patients undergoing CMRP. First pass CMRP was performed on a Philips Achieva CV 1.5 T MR scanner (Philips, The Netherlands), with standardised acquisition protocol with standard dose adenosine (SDA) at 140 μg/kg/min for 3 minutes. 3 short axis slices of 10 mm thickness were acquired per cardiac cycle using a single shot prospectively gated balanced TFE sequence (TR 2.5 ms, TE 1.3 ms, Flip angle 50° and voxel size 2.8 × 2.8 mm^2) after the administration of a 0.1 mmol/Kg bolus of intravenous Gadolinium. Heart rate (HR) and blood pressure (BP) were recorded at baseline and during stress. Non responders were defined as those patients with a normal CMRP scan and blunted haemodynamic response (maximum HR increase <10 beats per minute and fall in systolic BP (SBP) <10 mmHg). CMRP in this group was then repeated with high dose adenosine (HDA) at 175 μg/kg/min for 3 minutes. CMRP images were interpreted by 2 independent experienced observers. Coronary angiographic data was analysed by an experienced interventionist who was blinded to the perfusion data. Results obtained at SDA and HDA (expressed as mean value value ± SD) were compared using the student *t* test.

Results

Table 1, 51 patients (4.1%) recieved HDA. HDA was well tolerated with no serious adverse events. Coronary angiographic data was available for 20 patients (39%). HDA stress resulted in a significant increase in HR and RPP in non responders (Table 2). CMRP with HDA identified perfusion defects in 12 patients (24%) which were not present following stress with SDA. In 11 patients (92%)

Table 1: Patient characteristics (n = 51)

Characteristic	Number (%)
Age	62 ± 10.3
Male	29 (83%)
Hypertension	17 (49%)
Diabetes	13 (37%)
Hypercholesterolaemia	21 (60%)
Smoking	6 (17%)
Known coronary artery disease	19 (54%)

Table 2: Haemodynamic response to adenosine stress (n = 51)

	Rest	Standard dose	High dose
Systolic BP (mmHg)	136 ± 19	137 ± 23	133 ± 23
Heart rate (bpm)	68 ± 14	72 ± 16	82 ± 17 (P < 0.01)
Rate pressure product (bpm × mmHg/1000)	9.24 ± 2.4	9.89 ± 2.8	10.9 ± 2.5 (P < 0.01)

the identified perfusion defects correlated with significant stenosis (>70%) on the angiogram.

Conclusion

HDA achieved significant haemodynamic end-points with no significant clinical adverse events. Additional perfusion defects in the non responders at SDA were found using HDA. The perfusion defects correlated with significant coronary stenoses. The use of HDA may be important in certain patients where there is little or no haemodynamic response to SDA.