Quantitative cardiovascular magnetic resonance perfusion mapping as a guide for diagnosis

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Background: There is a lack of gold-standard non-invasive clinical markers derived from quantitative cardiovascular magnetic resonance (CMR) stress perfusion.

Purpose: This study aimed to compare quantification indices testing the hypothesis that they can discriminate possible normal from abnormal groups including microvascular dysfunction (MVD), coronary artery disease (CAD), and non-diagnostic tests due inappropriate response to the stressor agent.

Methods: Four-hundred and thirty-six consecutive patients (n=436, mean age 59.5 yrs) with typical angina and/or risk factors for CAD underwent stress CMR perfusion imaging using a dual-sequence quantitative spiral pulse protocol to estimate quantitative markers on a 1.5 T scanner. Anatomical coronary information, risk factors, and myocardial infarct were evaluated. Myocardial perfusion reserve values (MPR) were adjusted for rate-pressure product. For each perfusion assessment 3 short axis slice locations were imaged per heartbeat over a 60-heartbeat acquisition during an IV bolus of 0.05 mmol/kg of gadolinium contrast. Patients were divided into 4 groups: A) normal perfusion study; B) positive perfusion study due to epicardial coronary artery disease; C) positive perfusion study due to microvascular coronary artery disease; and D) non-diagnostic perfusion study due to inappropriate response to pharmacological stress.

Results: Stress myocardial blood flow (SMBF) and mean adjusted MPR differed between patients with no ischaemia and those clinically diagnosed with MVD (2.41±0.75 vs 1.81±0.52 mL/g/min, p<0.001, 2.78±0.94 vs 2.39±1.02, p=0.009, respectively). Patients deemed to have inadequate hyperaemia as opposed to inducible ischaemia had the lowest mean SMBF of 1.25±0.32 vs 1.80±0.61 mL/g/min (p<0.001); a cut-off value of <1.34 mL/g/min had the best predictive diagnostic accuracy for inadequate stress (area under curve [AUC] 0.875). Of note, comparing MVD vs CAD (single, 2-vessels, multivessel disease) without infarction stress pulmonary transit time (PTT) (centroid 6.9±0.72 vs 5.95±0.58, p=0.026), SMBF (1.80 vs 2.10 mL/g/min, p=0.0075), stress endo (1.60 vs 1.94 mL/g/min, p=0.0013), and stress epi (1.94 vs 2.21 mL/g/min, p=0.021) differed significantly between the two groups. The presence of infarction was shown also to be a significant discriminator between the two groups in logistic regression analysis (OR: 8.3, p=0.030).

Conclusions: This study showed fully quantitative stress markers may be useful in discriminating MVD and CAD patients as well as excluding patients with inadequate hyperaemia.