predictive of recovery in function following acute coronary syndromes. Therefore, coronary wave intensity analysis may be a useful adjunctive tool during cardiac catheterisation in the assessment of viability following ACS.

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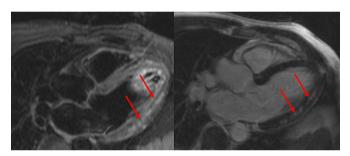
## THE ROLE OF CARDIOVASCULAR MRI IN TROPONIN POSITIVE ACUTE CORONARY SYNDROMES WITH UNOBSTRUCTED CORONARY ARTERIES

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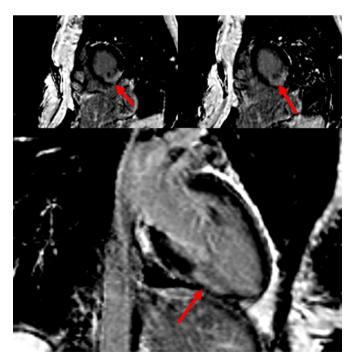
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**Background** Patients with acute coronary syndrome and unobstructed coronary arteries represent a clinical dilemma in whom clinical management is uncertain. Cardiovascular magnetic resonance (CMR) has the potential to non-invasively identify the presence of myocardial infarction or acute myocarditis, thus establishing a final diagnosis with management implications.

 $\pmb{\mathsf{Aim}}$  To assess the diagnostic value of CMR in patients presenting with ACS and unobstructed coronary arteries.



Abstract 134 Figure 1 Acute myocarditis with mid myocardial and epicardial oedema and fibrosis.



Abstract 134 Figure 2 Acute myocardial infarction with late gadolinium enhancement in the mid-cavity and apical inferior walls.

**Methods** From October 2010 to November 2011, 48 patients who presented with troponin positive ACS and unobstructed coronary arteries were consecutively recruited. A comprehensive CMR protocol, including T2 weighted STIR imaging for oedema and late gadolinium enhancement imaging for myocardial scarring, was performed within 4 weeks of the index event.

**Results** In 75% of cases, a cause for the troponin rise was found. Based on the oedema and scarring patterns observed, the most common diagnoses were acute myocarditis (Abstract 134 figure 1) in 40% of cases and acute myocardial infarction with spontaneous coronary recanalisation/embolus (Abstract 134 figure 2) in 19% of the cases. In six patients (12%) a diagnosis of cardiomyopathy was established: dilated cardiomyopathy (n=3), hypertrophic cardiomyopathy (n=1) and Tako-Tsubo cardiomyopathy (n=2). Acute pericarditis was present in two patients (4%). The remaining 25% of patients had a normal CMR scan.

**Conclusion** In the setting of acute coronary syndromes with unobstructed coronary arteries CMR was able to establish a final diagnosis in 75% of patients, identifying acute myocarditis, myocardial infarction with spontaneous recanalisation/embolus, and cardiomyopathies. Establishing a final diagnosis has an important impact in patient management and secondary prevention.

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CHRONIC DPP-4 INHIBITION BY SITAGLIPTIN ENHANCES BOTH GLOBAL AND REGIONAL MYOCARDIAL FUNCTION DURING DOBUTAMINE STRESS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND CORONARY ARTERY DISEASE

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**Background** Glucagon-like peptide-1 (GLP-1) is an incretin hormone secreted by the gut in response to the oral ingestion of nutrients. Through augmentation of myocardial glucose uptake and subsequent reduction in fatty acid oxidation, GLP-1 modulation therapy has emerged as a potential target for improving myocardial oxygen efficiency at times of ischaemic stress, such as occurs in obstructive coronary artery disease (CAD). Sitagliptin is a DDP-4 inhibitor licensed for the treatment of Type 2 Diabetes Mellitus (T2DM) that reduces degradation of plasma GLP-1 (7–36). We investigated whether sitagliptin improved myocardial performance during dobutamine stress echocardiography (DSE) in patients with T2DM and CAD.

Methods 12 patients (aged 69±9 years, 9 men) with T2DM on oral hypoglycaemic therapy (OHT), obstructive CAD and preserved left ventricular (LV) systolic function were studied. Each subject underwent DSE on two separate occasions after an overnight fast; the first (control) while receiving standard OHT and the second after the addition of sitagliptin (100 mg od) for 4 weeks. Tissue Doppler imaging was acquired in three apical views at rest, peak stress and 30 min recovery. Global function was assessed by ejection fraction (EF) using the Simpson's biplane method and mitral annular peak systolic velocity (MASV) averaged over six sites. Regional LV wall motion was assessed using a 12-segment model comprising the base and mid-level of six regional walls. Peak systolic tissue velocity (Vs), strain (S) and strain rate (SR) were calculated for each region from tissue Doppler velocity data averaged over three consecutive beats using an off-line workstation (EchoPac, GE Medical Systems).

**Results** At rest, all parameters of both global and regional LV performance were unchanged after sitagliptin. At peak stress, the rate-pressure product attained was the same for both DSE studies but those after sitagliptin demonstrated significantly enhanced myocardial performance in both global (LVEF  $69.9\pm6.5$  vs  $63.9\pm6.2\%$ , p=0.001; MASV  $12.69\pm3.0$  vs  $11.65\pm3.5$  cm/s,

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