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Meeting abstract

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1009 Myocardial injury following percutaneous coronary intervention in complex lesion: a cardiovascular magnetic resonance imaging and cardiac marker study

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Introduction

In patients undergoing percutaneous coronary intervention (PCI), 15 to 26% develop elevated creatine kinase isoenzyme MB (CKMB) levels after the procedure. Troponin rise is even more frequent and is observed in 29–48% of patients in a standard daily practice procedure. Little is known about the mechanism of this damage specifically in patient with complex PCI. Cardiovascular Magnetic Resonance (CMR) with gadolinium-based contrast media administration can non-invasively detect myocardial fibrosis. Inversion recovery preparation with segmented gradient echo readout (GRE) using recovery sequences is considered the gold standard sequence for the detection of late gadolinium myocardial enhancement (LGE).

Purpose

Aim of this study was the correlation of pre- and post-procedural changes in cardiac enzymes and extent of initial/post procedural areas of LGE measured by CMR in complex lesion.

Methods

Patients admitted to the hospital for PCI, stable/unstable angina or silent ischemia were enrolled. Only patients with complex coronary lesions are included. LGE CMR scan was performed 24 hours pre- and 24 hours post-PCI. The amount of enhancement was quantified by planimetry based on signal intensity (> 2 SD) of surrounding nulled myocardium.

Results

From a consecutive series of 36 patients with complex coronary lesions using the ACC/AHA score (19 type B, 17 type C) which 2 had a PCI of 2 vessels, 23 patients (63.9%) had stable angina and 13 unstable angina. 36 of them had successful PCI (28 men), 21 patients (58.3%) had troponin I elevation (> 0.04 ug/l) post PCI of which 6 had unstable angina. Thirty four patients had a follow-up scan. Two patients had an unsuccessful second CMR scan due to claustrophobia. Three patients with atypical LGE pattern and troponin I elevation were excluded. All patients (n = 19, 51%) with troponin (+) had more LGE in the post PCI scan than at baseline value (8 type M lesion, and 11 type H lesion). In the troponin (-) group there was no increase in LGE in the post PCI scan. There was a correlation between troponin elevation, severity of the lesion and the amount of new infarction as determined by LGE (r = 0.84).

Conclusion

Little is known about distal embolisation in complex coronary lesions. CMR is a useful tool, allowing non-invasive, rapid and accurate quantification of myocardial

fibrosis without ionizing radiation. This study demonstrates the value of CMR in identifying potential consequences of periprocedural myocardial necrosis after PCI in complex coronary lesion in stable and unstable angina. Lesion complexity could be useful to predict myocardial damage post PCI in term of troponin rise and LGE.