

SIMULATION OF PERCUTANEOUS CORONARY INTERVENTION: MRI AND MDCT ASSESSMENT OF CORONARY MICROEMBOLI

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Hypothesis

Microembolization of the coronary arteries occurs after spontaneous erosion or rupture of atherosclerotic plaque and during percutaneous coronary interventions (PCI). Determination of dislodged microemboli volumes during PCI that cause visible microinfarct is difficult. Thus, the purpose of this transitional animal study was to determine threshold microemboli volume that causes visible microinfarct on contrast enhanced MRI and MDCT. Cardiac injury biomarkers and histochemical staining were used to confirm the presence of myocardial injury.

Methods

Eighteen pigs were randomized to receive either 16mm³ or 32mm³ volumes of 40-120µm diameter microemboli (Biosphere Med, Rockland, MA). The volumes of delivered microemboli in this study were in the range of emboli retrieved from patients at autopsy (1, 2). We calculated using the following equation $V=(4*\pi*r^3)/3$, where r is the radius of microemboli. The mean of 80µm in diameter of 40-120µm microemboli was used for the calculations.

Under X-ray guidance, a 3F catheter was placed selectively beyond the second diagonal branch of the LAD coronary artery to produce microinfarcts. The reasons for using the LAD coronary artery, 40-120µm microemboli diameters and the selected volumes are that pathologists found at autopsy that 89% of microemboli are lodged in coronary vessels less than 120µm in diameter and 73% of these microemboli are dislodged in the territory subtended by the LAD coronary artery (1, 2). Diagnostic coronary angiograms were performed to visualize blood flow in the LAD artery before and after microembolization. Three days after coronary intervention, delayed contrast enhanced MDCT (64-slice) and MRI (1.5T) were performed. CT iohexol (3ml/kg of 350mg/ml) and MR Gd-DTPA (0.1.5mmol/kg) contrast media followed by saline chase of 40ml were injected IV using power injector. A semi-automatic threshold method was used to measure the microinfarct on MRI/MDCT. Cardiac injury biomarkers (creatinine-kinase MB and troponin I) and viability triphenyltetrazolium chloride (TTC) stain were used to confirmed myocardial injury at the time of imaging.

Results

MR and CT imaging. The contrast between microinfarcted and viable myocardium after administration of MR and CT contrast media was significantly greater on MRI (2.4±0.2) than on MDCT (1.6±0.1) after delivering 40-120µm microemboli. A 32mm³ microemboli volume caused sufficient microvascular obstruction that led to reproducible visualization of speckled myocardial microinfarct on DE-MRI and DE-MDCT. The volume of 16mm³ was insufficient to cause reproducible visible microinfarct, where 2/3 of the animals showed visible microinfarct. The semi-automatic threshold method showed microinfarcts in all 18 animals. The mean size of microinfarct on DE-MRI and DE-MDCT and TTC was not significantly different between the modalities (6.1±0.7% of LV mass, 6.2±0.6% and 6.3±0.7%, respectively) after microembolization by 16mm³ volume. A 32mm³ volume produced larger microinfarct (8.2±0.3% of LV mass, 8.8±0.5% and 7.9±0.4%, respectively) compared with 16mm³ volume.

Laboratory analysis. The levels of creatine-kinase MB and troponin I were 1670±370U/L and 0.52±0.28ng/ml in animals that received 16mm³ and 1060±235U/L and 0.68±0.4ng/ml in animals that received 32mm³ microemboli volume of 40-120µm diameter microemboli at 3 days after microembolization. TTC showed heterogeneous pattern of unstained necrotic myocardium compared to brick-red viable myocardium. Thus, TTC staining and the elevation of cardiac injury biomarkers confirmed the of presence of myocardial injury as a result of myocardial necrosis in all studied animals.

Discussion and Conclusions

This cardiac MRI and MDCT study established the threshold coronary microvascular obstruction that produces visible microinfarcts. Unlike hypoenhanced pattern of microvascular obstruction seen after reperfusion of acute myocardial infarct, microvascular obstruction caused by coronary microemboli produces a speckled pattern of enhancement of microinfarct. Current approaches, such as intracoronary administration of urokinase, extraction coronary atherectomy, directional coronary atherectomy, laser angioplasty, ultrasound thrombolysis, and AngioJet rapid thrombectomy have failed to produce satisfactory results in terms of achieving a significant reduction in distal embolization. Thus, MRI and MDCT modalities may be useful in evaluating the effectiveness of new therapies and future distal filtration devices. Furthermore, MRI and MDCT can be used interchangeably for visualizing microinfarct after PCI.

References

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2. Schwartz RS, et al. JACC 2009; 54:2167-2173.