Influence of Perfusion Defects on Survival after Coronary Revascularization

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within 60 days; 4 patients who underwent revascularization or had any cardiovascular events between PET and ICA were excluded. Myocardial blood flow at rest (rMBF), at stress with adenosine (sMBF) and myocardial flow reserve (MFR=sMBF/rMBF) were estimated using the 1–compartment Lortie model (Flows were normalized to surface area). Reversibility was assessed using computer-based automated edge detection (QCA). MFR was divided in 3 groups: G1:MFR<1.5, G2:1.5≤MFR<2 and G3:MFR≥2. MFR was estimated using the following formula: MFR = [peak MIBI activity - basal MIBI activity] / [peak stress arterial blood flow]. The extent of ischemia was defined as the sum of the segments with stress-induced perfusion abnormalities and left ventricular dysfunction on gated myocardial perfusion imaging and clinical outcomes in diabetics.

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Objectives: Aim of this study was to find out impact of glycosylated hemoglobin (HbA1C) on extent of perfusion abnormalities and left ventricular dysfunction on gated myocardial perfusion imaging and clinical outcomes in diabetics. Material and methods: This is a prospective study conducted at Karachi Institute of Heart Diseases (KIHD) from January ‘09 to December ‘11. Total 1013 (457 diabetic: 556 non-diabetics control) were included, among diabetic cohort 254 (56%) males and 203 (44%) were females, with a mean age of 58 ± 9 years. Mean duration of diabetes, HbA1C and fasting blood sugar (FBS) was 13.6 ± 89 months, 7.6% ± 1.7 and 137 ± 48 mg/dl respectively. Hypertension, dyslipidemia, positive family history for CAD and smoking was found in 73%, 26%, 35% and 15% respectively. GMPI was performed with exercise (54%) or dipyridamole stress (46%). GMPI was evaluated for size and severity of perfusion defects, transient ischemic dilatation ratio (>1.22), and left ventricle ejection fraction (LVEF) using commercial software (Autoquant®). Coronary angiography (CA) was used as gold standard in patients with positive GMPI ≥ 2 months of GMPI. All these patients were followed up for period of 22 months (12-24 months).

Results: In diabetics: non-diabetes control, GMPI was normal (49%;68%; p<0.0001), fixed (21.16%; p<0.049), reversible (30.16%; p<0.0001) with TID (19.9%; p<0.0001) respectively. ROC analysis reveals diagnostic strength of HbA1C for CAD at each coronary arteries was assessed by CAUC value with a sensitivity of 68.8% and specificity of 81.9% at a cut-off value of >7.3% (p value <0.0001). FBS and duration of diabetes was found to have poor diagnostic strength (p value >0.05). On the basis of criterion HbA1C, cohort was divided into those having value ≥7.3% (Group A) and those with ≤7.3% (Group B). In group A: incidence of fixed (33.09%; p<0.0001), reversible (41.22%; p<0.0001), sum stress score (62.5±2.2); p<0.0001) and sum thickness score (38.8±32.6; p<0.0001), LVEF<53 (16±58.11; p<0.0001) with TID (32.8%; p<0.0001) respectively. Kaplan Meier survival curves revealed event free survival for fatal MI 97.2%/98.3% (p value 0.742) and for non-fatal MI 87.9/97.9% (p<0.05) for group A and B respectively. Conclusions: We conclude that incidence of CAD is significantly higher in diabetics than non-diabetics. HbA1C, but not FBS and duration of diabetes is a reliable predictor for CAD. Extent and severity of perfusion defects, LV dysfunction and incidence of non-fatal MIs are more prevalent at a HbA1C ≥ 7.3%.

OP143
Evaluation of calcium score among other risk factors in patients with normal myocardial perfusion and normal left ventricular ejection fraction as determined by 18F-FDG-sestamibi myocardial perfusion imaging
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Aim: The aim of this study was to evaluate coronary artery calcium score (CACS) among other potential risk factors for cardiovascular events and/or death in patients with normal myocardial perfusion imaging (MPI) and normal left ventricular ejection fraction. Materials and methods: The study comprised 540 consecutive patients who underwent a CT attenuation corrected 18F-FDG-sestamibi myocardial perfusion imaging protocol with either ergometer bicycle- or pharmacological stress. Patients were referred to MPI by a cardiologist if they had a prevalent risk of ischemic heart disease (IHD), had a history of IHD with renewed suspicion of ischemia, or prior to renal transplantation because of chronic renal insufficiency. A total of 318 patients were included on the basis of a normal myocardial perfusion distribution, a normal left ventricular ejection fraction (LVEF), and a successful assessment of CACS. Data on patient risk factors, history, and events were retrieved from hospital files. Evaluated risk factors were: CACS ≥ 400 male gender, family history of coronary artery disease, hypertension, chronic renal insufficiency, diabetes, smoking, age > 63 years, known IHD, inability to perform ergometer bicycle stress, and hypercholesterolemia. Median follow-up time was 980 days. End points were myocardial infarction or need for revascularization of significant coronary stenosis, stroke or death. Results: CACS ≥ 256 was present in 81% and ≥ 400 in 62 patients. The annualized event rate of ischemic events or death was higher in the group of patients with CACS ≥ 400 (6.6%) compared to patients with CACS 0–399 (1.5%). When comparing ischemic events only the event rate was 4.6% and 0.9% in patients with CACS ≥ 400 and CACS 0–399 respectively. Cox regression analysis showed that CACS ≥ 400 (RR 3.4, p = 0.011, CI 1.3 to 9.0) and inability to perform ergometer bicycle stress (RR 4.8, p = 0.036, CI 1.1 to 21.4) were the most powerful predictors of ischemic events or death, whereas CACS ≥ 400 (RR 4.0, p = 0.034, CI 1.1 to 14.2), diabetes (RR 5.1, p = 0.016, CI 1.4 to 19.0), and male gender (RR 3.8, p = 0.059, CI 0.95 to 15.1) were the most powerful predictors of ischemic events only. Conclusion: CACS ≥ 400 is an important risk factor for ischemic events and death or ischemic events only in patients despite normal MPI and LVEF. CACS should subsequently be considered performed alongside MPI procedure.

OP144
High Dosage Nicorandil Administration on PCI in initial STELV Emitted AMI Patients Can Bring Beneficial Effects.

Purpose/Methods: Nicorandil administration in AMI patients was reported to reduce infarcted myocardium and prevent left ventricular (LV) remodeling. In addition, high dosage (HD) NCR administration was reported to be effective to CHF. To clarify whether HD nicorandil administration in AMI patients was more effective to salvage myocardium than low dosage (LD) nicorandil administration, 30 patients (age≥62+10;M/F=26/4) with initial AMI with ST segment elevation undergoing successful percutaneous coronary intervention (PCI) were enrolled. Patients were divided to HD group (15patients) and LD group (15patients) at random. HD nicorandil administration was intravenous (0.2 mg/kg/five minutes), intracoronary (2mg), and continuous (0.2mg/kg/h for 24hours) administration. LD nicorandil administration was intravenous (4mg), intracoronary (2mg), and continuous (4mg/h for 24 hours) administration. TC-99m MIBI QGS was done on subacute and chronic period (6 to 9 months). LV ejection fraction (LVEF), end diastolic volume (EDV) and end systolic volume (ESV) using TC99m MIBI QGS were calculated. Total ischemic time (TD) was calculated by the summation of 5-points grading defect score (0=normal perfusion to 4=defect perfusion) of 17 SPECT segments. Extent score (ES) was calculated by the number of hyperperfusion and perfusion defect segments. Exercise tolerance was estimated by anaerobic threshold oxygen consumption (AT-VO2) and peak oxygen consumption (Peak-VO2) using cardiopulmonary exercise test (CPX). Result: There were no significant differences between HD group and LD group in CPK, BNP, AT-VO2 and suacute and chronic EF, EDV and ESV. Some results of HD group were significantly better than LD group in subacute TDS (HD: 8.6±6.7; LD: 16.1±10.4; p=0.01), chronic TDS (HD: 7.2±6.6 vs. LD: 13.7±11.7; p=0.04) and chronic Peak VO2 (HD: 23.5±4.8 ml/min/kg vs. LD: 17.8±4.5 ml/min/kg; p=0.004). Conclusion: High dosage nicorandil administration in AMI patients was effective to reduce myocardial infarction and improve exercise tolerance in comparison with low dosage nicorandil administration.
segment model: 0=normal and 1, 2, 3, and 4 = slightly, moderately, severely and completely compromised perfusion. Data from up to ten years' follow-up were derived from national registers. Results: One fourth (N=527) had either reversible (N=324) or mixed perfusion defects (N=203). Of these, 184 underwent coronary revascularization within 180 days following MPS (group 1), whereas 65% received medical therapy (group 2). Mean follow-up was 5.0 years (range 0.2 – 9.4). Groups 1 and 2 were comparable regarding sex, age, history and ejection fraction (N=255), but differed by SSS [12.8±0.9 vs. 9.5±0.4, p<0.001, N=341], defect size (more large defects in group 1, p<0.0001), and reversibility (higher degree of reversibility in group 1, p<0.0001). All-cause death incidence rates were 3.5% and 4.3%, respectively. A total of 105 (20%) died: 34 (19%) in group 1 and 71 (21%) in group 2 (NS). Perfusion defects were small (SSS 4–8) in 235 (45%), moderate (SSS 9–13) in 131 (25%), and large (SSS >13) in 161 (31%). Of these, 20%, 37%, and 55%, respectively, were in group 1. The Hazard Ratios (HRs) for patients in group 2 having three defects sizes were 1.7 (p=0.31), 2.1 (p=0.11), and 1.9 (p=0.04), respectively. Mild, moderate or marked reversibility (reductions in SSS of ≤5±6, 6–9%, or ≥9% of maximal true value), was found in 267 (51%), 135 (26%), and 125 (24%) of the 527 patients with 19%, 40% and 63%, respectively, in group 1. HRs in the corresponding subgroups in group 2 were 2.3 (p=0.07), 0.8 (p=0.59), and 2.7 (p=0.008), respectively. In 101 patients (19%) with limited reversibility, HR for medical therapy was 1.5 (p=0.31) compared to 1.7 (p=0.31), 2.2 (p=0.22), and 2.7 (p=0.01) in the 426 patients (81%) who had either i) a small defect, ii) a moderate defect with moderate or marked reversibility, or iii) a large defect with marked reversibility. Conclusion: For patients with large perfusion defects and defects with marked reversibility, the benefit of revascularization versus medical therapy was evident, as opposed to patients with smaller defects or defects with less extensive reversibility.

OP146
Ischemia but not necrosis is a predictor of post-stress LVEF drop 6 months after myocardial infarction: a gated myocardial perfusion SPECT study.
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Background: Gated myocardial perfusion SPECT (gSPECT) is able to detect restenosis or progression of coronary artery disease in the early systematic follow-up of myocardial infarction (MI). Although post-stress left ventricular ejection fraction (LVEF) decrease is often associated with ischemia, its explanatory factors after MI remain unclear. Aim: To identify the clinical and gSPECT characteristics associated with a 5% or more post-stress LVEF decrease in patients with earlier MI.
Methods: Two-hundred and thirty six consecutive patients admitted in intensive care unit for acute MI were prospectively included. Six months after discharge, a post-stress/rest gSPECT procedure was performed according to a one day protocol. Post exercise-induced stress gSPECT images were acquired 10 to 15 minutes after intravenous injection of 3.7 MBq/Kg of 99mTc-sestamibi. Rest gSPECT images were acquired 30 minutes after injection of 11.1 MBq/Kg of 99mTc-sestamibi and at least 4 hours after post-stress injection. End-diastolic volume, end-systolic volume and LVEF were determined using GGS5 software. LVEF drop was considered significant if post-stress LVEF was ≤5% compared with LVEF at rest. Summed Stress Score (SSS), Summed Rest Score (SRS), and Summed Difference Score (SDS) were visually evaluated using a 17 segments model. Results: Post-stress LVEF drop was observed in 56 (24%) patients (group A). Demographic and infarct characteristics were similar when compared with patients with unaltered post-stress LVEF (group B). Patients with LVEF drop had significantly higher SSS when compared with patients with unaltered LVEF (median (IQR): 2 (0-5) vs 0 (0-3) p=0.016); they also had more often significant ischemia (i.e. SSS>7) (14% vs 4%, p=0.024). Moreover, rest LVEF was higher in group A than in group B (56% vs 56% (49-63) vs p<0.001). Multivariate logistic regression analysis identified significant ischemia (OR: 1.70, 95% CI: 1.85-7.38) and rest LVEF (OR: 1.07, 95% CI: 1.04-1.11) as independent associated factors of LVEF drop. Conclusion: In patients with previous myocardial infarction, a post-stress LVEF decrease ≥5% is associated with higher incidence of reversible perfusion defects. These results are in accordance with the myocardial stunning model and exclude the potential influence of an extended myocardial necrosis or left ventricular remodeling on post stress LVEF fall following MI.

OP147
Estimation of myocardium at risk and saved myocardium using myocardial perfusion gated-SPECT practiced one month after infarction
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Objective: To estimate myocardium at risk (MR) and saved myocardium (SM) following percutaneous coronary revascularization (PCR) in patients with a acute myocardial infarction (AMI), using myocardial perfusion gated-SPECT (gSPECT). Method. Forty patients (mean age 61.5 years, 8 women) with AMI and primary PCR were prospectively included. They underwent two gSPECT: the first one consisted in the injection of 800 MBq of 99mTc-tetrofosmin prior to the PCR. Four days after the PCR the first study (gSPECT-1) was performed when patient was considered stable immediately after PCR procedure. The second study (gSPECT-2) was performed between the fourth and fifth week following AMI. The area of real MR was quantified by assessing the perfusion defect in gSPECT-1, while the SM was measured by the difference between the MR area and the area of necrosis in gSPECT-2. The MR area in gSPECT-2 was calculated by analysing the discordance between the extension of the perfusion defect and the left ventricular motility defect. Quantification of left ventricular perfusion defects, wall motion, wall thickening, ejection fraction and ventricular volumes were assessed using the GGS software. Results. A significant improvement in perfusion, wall motion, thickening and left ventricular ejection fraction (post vs. pre) was observed when compared gSPECT-1 and gSPECT-2. In gSPECT-2, significant discordance was observed (p=0.007) in cm2 between areas of wall motion and perfusion (extension of perfusion defect < extension of motility defect), making it possible to construct a model to estimate the extent of MR area that correlated well with real areas of MR and SM (Pearson correlation coefficient: 0.78). The degree of concordance for correct classification of patients with SM >50% was 83% (kappa: 0.6). Conclusions. Gated myocardial perfusion SPECT performed one month after early PCR in a first AMI can be used to establish an estimate of MR and SM areas.

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Neurosciences: Dopamine Imaging

OP151
Quantification and Whole-Body Distribution of a Novel Dopamine D2/D3 Receptor Agonist, [11F]MCL-524, in Monkeys: A Prediction for Application in Human Subjects
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Aim: A fluorine-18 radiolabeled dopamine D2/D3 receptors agonist PET radioligand may be the optimal tool for investigation of the interaction between radioligand binding, endogenous dopamine and receptor trafficking in man. We recently reported the initial radiochemistry development of the promising agonist [11F]MCL-524 [1]. In the current study we performed a quantitative analysis of [11F]MCL-524 binding to central D2/D3 receptors and a whole body distribution study in nonhuman primates. Materials and Methods: A total of eight PET measurements were performed on six experimental days in four cynomolgus monkeys. Two monkeys were studied on two experimental days each using a HRRT PET system. On the first day two baseline PET measurements were performed after i.v. injection of respectively [11C]MNPA [2] and [11F]MCL-524. Arterial blood was obtained for

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