culprit lesions (60 vs. 40%, p<0.001). No differences were observed for mortality with respect to either culprit vessel (log-rank-p-value=0.54) or proximal vs. mid/distal location of the culprit lesion within the vessel (log-rank-p-value=0.45). This was also true after multivariable adjustment, independent predictors of outcome were serum lactate, success of revascularization, age, serum creatinine, prior stroke, known peripheral artery disease and left bundle branch block in admission electrocardiogram.

CONCLUSIONS For patients with CS complicating myocardial infarction, the culprit lesion localization seems to be unrelated with mortality.

CATEGORIES CORONARY: Acute Myocardial Infarction KEYWORDS Cardiac shock, Culprit Vessel, Outcomes

TCT-231

Exenatide Does Not Improve Myocardial Salvage In Patients With An Acute Myocardial Infarction Successfully Treated With Primary Percutaneous Coronary Intervention: The First Results Of The EXAMI Trial

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BACKGROUND The Glucagon Like Peptide 1 receptor agonist exenatide is an incretin based compound used for glycemic control in patients with type 2 diabetes mellitus (DM) and has previously been demonstrated to have cardioprotective properties. This double blinded, randomized clinical trial studies the effect of exenatide treatment on myocardial salvage in ST Elevated Myocardial Infarction (STEMI) patients who successfully underwent primary Percutaneous Coronary Intervention (PCI).

METHODS STEMI patients were randomly assigned to either intravenous exenatide or placebo. Study medication was started prior to PCI using 10 µg/h for 30 minutes followed by 0.84 µg/h for 72h. Patients with a previous STEMI, Thrombolysis In Myocardial Infarction flow 2 of 3, multi vessel disease, or DM were excluded. Magnetic resonance imaging was performed within 2-7 days after PCI, to determine left ventricular (LV) volumes and ejection fraction (EF), infarct size and area at risk (using T2-weighted hyperintensity (T2W) and late enhancement endocardial surface area (ESA)). The primary endpoint of myocardial salvage index (MSI) was defined as the difference between the area at risk and total infarct size, as ratio of the area at risk. Secondary endpoints were major cardiovascular events ((MACE) defined as cardiac death, repeat STEMI, coronary artery bypass grafting or repeat PCI), LV end systolic volume (ESV), LV EF and infarct size.

RESULTS In total 91 patients (age 57.4 \pm 10.1 years, 76% male) completed the study. There were no baseline differences between both groups. Symptom to balloon time was 181 \pm 85 vs. 201 \pm 95 min (p=0.24) for exenatide (n=42) and placebo (n=49) respectively. Patients receiving exenatide had significantly more episodes of nausea early after study drug initiation (16 vs 4 patients, p<0.001), but this did not lead to study discontinuation and was treated successfully with metoclopramide in all cases. The MSI was not significantly different between both groups (with ESA 0.30 \pm 0.26 vs 0.26 \pm 0.22, p=0.43 and with T2W 0.35 \pm 0.25 vs 0.33 \pm 0.22, p=0.65 for exenatide vs placebo respectively). There were also no differences in LV ESV (61.3 \pm 26.0 vs 54.9 \pm 25.9 ml/m2, p=0.259), LV EF (52.6 \pm 7.7 vs. 52.4 \pm 6.9%, p=0.53) and infarct size (18.8 \pm 13.2 vs. 18.8 \pm 11.3% of LV mass, p=0.965). No MACE occurred during the in-hospital phase.

CONCLUSIONS Our exenatide treatment protocol does not improve myocardial salvage in STEMI patients successfully treated with primary PCI. This is incongruent with previous clinical trail results. Differences in the exenatide treatment protocols are the most obvious reason for the ambiguous trial results. Further studies are needed to establish the exact role and the optimal treatment protocol of exenatide in this group of patients.

CATEGORIES CORONARY: Acute Myocardial Infarction

TCT-232

Primary Percutaneous Coronary Intervention In Nonagenarian Patients With ST Elevation Myocardial Infarction: In-Hospital Mortality And Outcomes At One Year Follow-Up

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BACKGROUND Limited information is available about the efficacy and outcomes after primary percutaneous coronary intervention (P-PCI) in very elderly patients (pts) with ST Elevation Myocardial Infarction (STEMI).

METHODS 23 nonagenarian pts were treated (1% of the total STEMI population underwent P-PCI). We evaluated in-hospital, 6-months and 1 year-mortality in a retrospective analysis of nonagenarian pts admitted at our Department with STEMI and treated with P-PCI from November 2004 and December 2013. A bivariate analysis was carried on with exact Fisher's test, identifying those variables associated with mortality. Odds ratios (ORs) from univariate logistic regression analyses were performed.

RESULTS All pts received aspirin and 300 mg clopidogrel loading dose. Mean age: 91.2 yrs (range 90-96). 65% was women. Mean left ventricular ejection fraction (LVEF) at the admission: 38.9% (23% of pts with LVEF<35%). Advanced Killip class (3-4) at presentation: 10 pts (43%). Baseline characteristic: 13% of pts with prior revascularization, 17.3% prior stroke, 21.7% diabetes, 43% hypertension, 8.6% atrial fibrillation. No dementia (good mental status). Mean renal function evaluated by creatinine clearance measured by the Cockcroft-Gault equation: 38.7 mL/min (range 16.8 - 72.9). Mean hemoglobin value: 13.7 gr/dL. Mean number of vessels treated per pts: 1.04, showing a strategy of treating the culprit vessel only. 3 left main (LM), 8 left anterior descending coronary artery (LAD), 4 circumflex coronary artery, 8 right coronary artery (RCA). The radial approach was performed in 65% (100% of cases from 2012). The proportion of radial to femoral shift was 6%. An average of 1.26 stents per pt were implanted (100% were bare metal stent). In 4 pts we performed P-PCI without stent. No Glycoprotein IIb/IIIa were used. Intra-aortic balloon pump was implanted in 1 pt. The TIMI flow 2-3 post P-PCI was achieved in 78.2% of pts (angiographic success was achieved in 20/23 pts). In 1 pt occurred acute renal failure post P-PCI and in 1 pt occurred major bleeding; no stroke. The overall in-hospital mortality rate was 34.7% (one pt died during the procedure). Cumulative mortality after discharge at 6 months was 14% and at 1 year was 28%. LVEF<30 showed a higher risk of in-hospital mortality and cumulative mortality at 6 months. Killip≥3 showed a higher risk of in-hospital mortality. LM and LAD showed a higher risk of in-hospital mortality, cumulative mortality at 6 months and at 1 year.

CONCLUSIONS Our data suggest that primary P-PCI in nonagenarian pts can be performed with an acceptable bleeding risk. The in-hospital mortality is significant but the cumulative mortality at 6 months and 1 year is low, showed a good success rate of the P-PCI strategy. The radial approach is feasible and safe. The invasive strategy in selected very elderly population should be offered. Further studies are needed to evaluate the benefit of P-PCI versus non P-PCI strategy in the very elderly population.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS Elderly, Primary percutaneous coronary intervention, Radial

TCT-233

Prognostic value of left ventricular global function index in patients after ST-segment elevation myocardial infarction

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BACKGROUND The left ventricular global function index (LVGFI) is a novel indicator of left ventricular performance. Its prognostic value in patients after ST-segment elevation myocardial infarction (STEMI) is unknown. We sought to evaluate the prognostic value of LVGFI measured by cardiovascular magnetic resonance (CMR) imaging after STEMI

METHODS Two hundred eligible STEMI patients (56 ± 11 years, 16 % female) revascularized by primary percutaneous coronary intervention (PCI) were followed-up for 3.1 [2-4.1] years for major adverse cardiac events (MACE). MACE was defined as a composite of death, nonfatal myocardial re-infarction and new congestive heart failure. All patients underwent CMR imaging within 2 [2-4] days after STEMI. Late enhancement and cine images were acquired to assess myocardial injury as well as myocardial function, including LVGFI.

RESULTS Patients suffering a MACE event (n = 20, 10%) had a significantly lower LVGFI (p = 0.001). In Kaplan-Meier analysis, a

decreased LVGFI was associated with a reduced MACE-free survival (p = 0.007). Multivariate Cox regression analysis revealed a decreased LVGFI as an independent predictor for MACE (hazard ratio = 5.24, 95 % CI 1.70-16.17, p = 0.004) after adjusting for microvascular obstruction, multivessel disease and diabetes. In ROC analysis, LVGFI was a strong predictor for MACE (AUC = 0.73, CI 0.61-0.85). The predictive value of LVEF was similar (AUC = 0.74, CI 0.61-0.87).

CONCLUSIONS LVGFI assessed by CMR is a strong predictor of MACE within 3 years after first STEMI.

CATEGORIES CORONARY: Acute Myocardial Infarction

TCT-234

Quality Comparison Of Different Primary Angioplasty Activation Service Models Within A Single Cardiac Centre

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BACKGROUND The pan-London PPCI network uses a 24/7 "Paramedic Activated" service model with the London Ambulance Service (LAS) across 8 Cardiac Centres - paramedics autonomously activate the PPCI service from the community. This model aims to reduce ischemia time by autonomous paramedic activation. Our Centre also serves a catchment area surrounding London in which a "Cardiologist Screened" service model is used in conjunction with the South East Coast Ambulance Service (SECAMB) - paramedics telemetry link an ECG to our Centre and discuss the case with the on-call Cardiologist. If the case does not meet STEMI criteria, the SECAMB crew is redirected to the nearest ED. This model intends to rationalize the number of false activations in a catchment area with a much larger geographical distribution (and therefore transfer times) than London. There are no previous reports comparing a dual PPCI activation model within one Centre. We compared the False Activation rates between our two service models, as a metric of quality.

METHODS Over a 20-month period (January 2013 - August 2014) there were 2077 consecutive activations of our PPCI service. Cases were categorized by source of activation - Paramedic Activated (LAS) or Cardiologist Screened (SECAMB, cardiac center ED or non-cardiac center ED). Cases were also categorized as True Activations (STEMI with PPCI or emergency CABG) or as False Activation (not STEMI, but may have undergone emergency angiography).

RESULTS 942 (45.3%) cases were Paramedic Activated via LAS (LAS Cohort) and 1038 (50.0%) cases were Cardiologist Screened via SECAMB (SECAMB Cohort). 24 (1.2%) patients self presented to our ED and 73 (3.5%) to local EDs - both were excluded from further analysis for the purpose of comparing the two ambulance service models. The proportion of False Activations was significantly higher in the in the SECAMB cohort compared to the LAS cohort: SECAMB 805 patients (77.6%) and LAS 470 patients (49.9%); χ2 164.8 (p<0.001). 708/1038 of the SECAMB activations did not meet STEMI criteria were diverted to local EDs. Of the SECAMB activations actually transferred to our Centre (330 cases), the False Activation rate was significantly lower compared to the LAS Cohort: SECAMB 97 patients (29.4%) and LAS 470 patients (49.9%); χ2 41.6 (p<0.001).

	LAS	SECAMB	Cardiac Centre ED	Non-Cardiac Centre ED
True Activations	472	233	16	45
False Activations	470	805	8	28
Total Activations	942	1038	24	73
False Activation Rate	49.9%	77.6%	33.3%	38.4%

CONCLUSIONS On initial inspection our data does not support the notion that a Cardiologist Screened service should have a lower False Activation rate than a Paramedic Activated service. However once diverted SECAMB referrals are excluded, the False Activation rate is lower in the Cardiology Screened pathway. Our study highlights an interesting portal to examine quality of PPCI activation service models.

CATEGORIES OTHER: Quality, Guidelines and Appropriateness Criteria

KEYWORDS Primary PCI, ST-segment elevation myocardial infarction network, ST-segment elevation myocardial infarction, acute

TCT-235

Poor R-wave Progression on Admission Electrocardiograms Predicts Left Anterior Descending Culprit in Patients with non-ST-segment Elevation Myocardial Infarction

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BACKGROUND Poor R-wave progression (PRWP) on electrocardiograms has been reported to be associated with old myocardial infarction (MI) due to an occlusion of the left anterior descending (LAD) artery. However, PRWP can be observed in patients presenting with acute MI who do not have history of MI. We hypothesized that PRWP on admission electrocardiograms predict LAD culprit in our cohort of patients with non-ST-segment Elevation Myocardial Infarction (NSTEMI).

METHODS We performed a retrospective analysis of 481 consecutive NSTEMI patients who underwent coronary angiography from January 2013 to June 2014. Patients with ventricular paced rhythm and bundle branch block were excluded. In addition, patients with history of MI, coronary artery bypass grafting, or heart failure were excluded. Electrocardiograms on admission were interpreted in a blinded fashion, and PRWP was defined as a R-wave amplitude <3mm in lead V3 on presenting electrocardiograms. Patients were divided into those with and without PRWP. An independent cardiologist blinded to the clinical data visually interpreted all coronary angiograms. LAD culprit was defined as LAD stenosis≥70% with either thrombus or ulcerative base. Baseline and angiographic characteristics were recorded. In addition, in-hospital heart failure, in-hospital and 30-day major adverse cardiac events (MACE) including death, recurrent myocardial infarction, ventricular fibrillation, and target vessel revascularization were recorded and compared between the two groups.

RESULTS Among 310 patients included in the final analysis, 73 patients (23.5%) had PRWP. No statistically significant difference was observed in baseline characteristics between the two groups. Patients with PRWP had a higher rate of Killip class>1 on admission than those without (17.8% vs. 8.9%, p=0.032). Although peak troponin I values were comparable between the two groups (median [interquartile]; 0.61 [0.15-3.38] ng/ml vs. 0.92 [0.13-6.47] ng/ml, p=0.58), patients with PRWP had a lower left ventricular ejection fraction than those without (57 [39-60] % vs. 60 [55-65] %, p= 0.004). With respect to angiographic finding, patients with PRWP had a higher rate of LAD culprit than those without (35.6% vs. 23.2%, p=0.035). The incidence of in-hospital heart failure was higher in patients with PRWP than those without (20.5% vs. 9.3%, p=0.009). There was a trend toward a higher incidence of in-hospital MACE (4.1% vs. 1.7%, p=0.36) and 30-day MACE (8.2% vs. 3.4%, p=0.10) in patients with PRWP.

CONCLUSIONS In our cohort of patients with NSTEMI, PRWP on admission electrocardiograms was associated with the presence of LAD culprit. There was a trend toward a higher incidence of in-hospital and 30-day MACE in patients with PRWP.

CATEGORIES CORONARY: Acute Myocardial Infarction

KEYWORDS Left anterior descending coronary artery, Non-ST-segment elevation acute coronary syndromes

TCT-236

The role of ADAMTS13 in acute myocardial infarction: cause or consequence?

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BACKGROUND ADAMTS13 is a metalloprotease that cleaves von Willebrand factor (VWF), thereby reducing its prothrombotic properties. There is considerable evidence that VWF levels increase and ADAMTS13 levels decrease in STEMI patients. It is unclear if this contributes to no reflow, infarct size and intramyocardial hemorrhage (IMH). The aim of this study was to determine the role of ADAMTS13 in patients with acute myocardial infarction; and to investigate the benefits of recombinant (r)ADAMTS13 in a porcine model of myocardial ischemia-reperfusion with dual antiplatelet therapy and heparin.

METHODS In 49 consecutive PCI-treated STEMI patients, blood samples were collected directly after and up to 7 days following PCI.