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Primary PCI for STEMI: Gender, Bleeding and Transradial. An Intriguing Combination

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Background: Female gender female has been associated with increased bleeding after primary PCI (PPCI) for STEMI. The influence of the transradial (TR) approach on the bleeding rate in female STEMI patients has not been investigated. Aim: To evaluate gender specific bleeding complications according to access site during PPCI.
Methods: Retrospective and comparative study of 259 women and 1009 male who underwent PPCI for STEMI (2005-2009). End Point: Overall bleeding, access and non access bleeding, large and small hematomas.
Results: Women were older (69±13 vs. 57±13 ,p<0.01) with more frequent arterial hypertension (77% vs.55%,p<0.01) and diabetes (38% vs.24%,p<0.01). They had higher BMI (29±6 vs. 27±4, p<0.01) and creatinine level (1.03±1 vs.0.98±0.6 mg/dl, p<0.02). Transradial PCI was used less frequently in women (41% vs. 59%, p<0.01). The women had more Overall bleeding (23% vs. 13%, p<0.01), access site bleeding (21% vs. 11%, p <0.01), large hematoma rate (3.5% vs. 1.4% (p=0.02) and small hematoma rate (18.5% vs. 9.6%). TR PPCI was associated with a reduction of overall bleeding (5% vs. 26%, p<0.01), access site bleeding (4.5 vs. 23%, p<0.01), small hematoma (4% vs 19%, p<0.01) and large hematoma (0.4 vs. 3%,p<0.01) in the overall population. Rates of bleeding according to access site and gender are presented in the table.
Conclusion: Bleeding complications after PPCI are more frequent in women. Transradial PPCI is associated with a markedly reduced risk of bleeding, however still they are more frequently observed in women due to a high occurrence of small hematoma at the access site.

		Women	Men	p value
Overall bleeding (%)	TR	15	4	<0.01
	TF	28	26	ns
Access Bleeding(%)	TR	14	3	<0.01
	TF	23	21	ns
Large Hematoma(%)	TR	0.1	0.5	ns
	TF	5	2.5	ns
Small Hematoma(%)	TR	13	2	ns
	TF	18	19	<0.01

PCI in STEMI: Prognostic Impact of Nonculprit Lesions PCI During Primary PCI

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Background: Percutaneous coronary intervention (PCI) of the culprit vessel is the conventional treatment in STEMI. However, pts often have significant coronary lesions in other vessels. In patients with STEMI and multivessel disease, it is unknown whether it is safe or even desirable to also treat the nonculprit vessel during primary PCI procedure.
Aim: This study aimed to test the safety of of the culprit and nonculprit lesions PCI with PCI of only the culprit lesion in STEMI during primary PCI.
Method and Results: We used our Database of all pts treated using primary PCI for STEMI. Excluded pts were those with cardiogenic shock and one vessel disease. Patients were allocated into 2 groups: culprit vessel PCI and nonculprit PCI. Of note, 'one time' multivessel PCI was used in only 6.4% of our STEMI patients.

	Culprit PCI [N=928]	Nonculprit PCI [N=64]	P
Age	63±12	63±14	0.8
Anterior AMI	40%	59%	0.01
Contrast (ml)	174±68	279±59	0.003
One year			
Death	6.5%	16%	0.003
Re-MI	5.5%	1.6%	0.2
Stent thrombosis	2.5%	0%	0.2
TVR	13.6%	6.3%	0.09
MACE	22%	22%	0.99

Conclusion: Nonculprit PCI strategy is not advisable in non-shock patients with multivessel coronary diseas in patients with STEMI undergoing primary PCI.

Non-Inferiority of Thrombolysis Comparing Primary PCI in STEMI Within 3 Hours from Symptom Onset

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Background: Randomized trials and meta-analyses have shown superiority of primary PCI to in-hospital thrombolysis as reperfusion therapy in STEMI patients arriving within 6-12 hours after symptom onset.
Objective: To compare in-hospital clinical outcome of STEMI patients admitted to the CCU within 3 hours from symptom onset and treated by primary PCI to that of patients treated by thrombolysis.
Methods: We retrospectively evaluated 148 hemodynamically stable STEMI patients that were admitted during 2007-2011 within 3 hours of symptom onset. All thrombolysis patients underwent coronary angiography at 3-36 hours after hospital admission, including rescue PCI.
Results: Primary PCI was performed in 72 patients and streptokinase was administered to 76 patients. The 2 groups were comparable with regard to age, sex, atherosclerosis risk factors, anterior wall MI [44.7% thrombolysis vs 40.3% primary PCI (p=NS)] and time from symptom onset to door [89 vs. 92 minutes, respectively (p=NS)]. Door to needle time was shorter than door to balloon time [16 vs. 52 minutes ($p<0.01$)]. Seventeen (22%) patients treated by thrombolysis did not reperfuse clinically and underwent rescue PCI [5 (6%) patients had TIMI flow=0, and 2(3%) had TIMI flow=1].We found no difference in outcome regarding bleeding, vascular complications, CPK levels, LVEF, hospitalization length, and in-hospital death.
Conclusion: Thrombolysis therapy, given early in stable STEMI patients admitted within 3 hours of symptoms onset, seems to be an acceptable and non- inferior to primary PCI as reperfusion strategy . However, larger randomized clinical trials are needed to establish this therapeutic approach.

Effects of Treatment on QT-Interval in Patients with Acute ST-Elevation Myocardial Infarction

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Acute myocardial infarction with ST-elevation (STEMI) is associated with abnormalities in depolarization and repolarization. Coronary reperfusion affects the extra-cellular milieu which may result in changes in repolarization. Aim: evaluate the effects of reperfusion therapy on QT interval in patients with acute STEMI.
Methods: One hundred forty six patients acute myocardial infarction, 65 inferior and 81 anterior STEMI were evaluated. All patients with anterior STEMI had coronary angiography, 78 had successful angioplasty and coronary stent implantation, 1 failed and 2 had coronary ectasia and treated with anticoagulants. Patients with inferior STEMI, 29 had primary angioplasty and 36 were treated with thrombolysis. QT intervals in all 12 leads were measured at admission, a day after the procedure and before discharge.
Results: In patients with acute anterior STEMI, in the anteroseptal electrocardiographic leads QT interval increased from 349±35 at admission to 378±49 msec after treatment, $p<0.000002$. In inferior and high lateral leads, QT intervals increased significantly after treatment. No significant changes in QT interval were observed between electrocardiograms after treatment and at discharge. In patients with acute inferior STEMI, QT intervals were normal and did not change significantly after treatment and at discharge.
Conclusions: In patients with acute anterior STEMI, reperfusion therapy lengthened the QT interval, while in patients with acute inferior STEMI, QT intervals did not change significantly.

Hyperuricemia is a Predictor of Infarct Size in Patients with ST-Elevation Myocardial Infarction

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Among 1715 patients, admitted in ICCU Rambam Health Care Campus, Haifa, with acute ST-segment elevation myocardial infarction between January 2008 and March 2010, 356 consecutive patients (mean age 63+15, women 22%) without previous history of myocardial infarction, coronary angiography, angioplasty or bypass grafts, was retrospectively evaluated. Standard treatment included primary percutaneous coronary intervention (PCI) or thrombolytic therapy, aspirin, clopidogrel and statines. Blood samples for serum uric acid were collected during the first 48 hours of admission. Moderate HU was considered as level of uric acid>7 mg/dl and >6 mg/dl for men and women, respectively and severe HU >10 mg/dl for both sexes. Left ventricular ejection fraction (EF) measured by transthoracic echocardiography within first 5 days of admission.

Univariate analysis showed that moderate HU, OR 3.3 (95%CI 1.6-6.7), P<0.001; age>70 years, OR 3.6 (95%CI 1.9-6.9), P<0.0001; primary PCI, OR 0.3 (95%CI 0.2-0.6), P<0.0001; Killip II-III, OR 4.6 (95%CI 1.4-13.1), P<0.0001 and creatinine clearance test (CCT) <50 mg/dl, OR 6.5 (95%CI 3.0-13.9), P<0.0001 were a significant predictors for EF<40%, while severe HU, OR 15.7 (95%CI 4.1-59.9), P<0.0001 and Killip IV, OR 23.6 (95%CI 2.6-216.5), P<0.005 were a strongest predictors. Multivariate analysis after adjustment for age, Killip, CCT, diabetes and primary PCI, revealed that moderate HU reached borderline significance for prediction of EF<40%, OR 2 (95%CI 0.9-4.3), P<0.09, while Killip III, OR 4 (95%CI 2.0-7.9), P<0.0001; Killip IV, OR 16 (95%CI 1.6-159.0), P<0.02 and severe HU, OR 8.0 (95% CI 2.0-33.0), P<0.004 were independent predictors of EF<40%, area under ROC equal 0.73 (95% CI 0.66-0.81).

In patients presenting with first acute ST-segment elevation myocardial infarction, severe HU appear to be an independent predictor of larger infarct size.

Cardiac MRI - A Tool for Routine Early Evaluation of ST Elevation Myocardial Infarction (STEMI)

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Background: Cardiovascular magnetic resonance (CMR) is an emerging modality for the evaluation of acute ST elevation myocardial infarction (STEMI). New software allows myocardial damage accurate quantification.
Purpose: The aim of this prospective study was to report the value of quantitative CMR in routine evaluation of consecutive STEMI patients.
Subjects and methods: The study cohort included 27 consecutive patients who underwent primary angioplasty for first STEMI (26 males, mean age 58± years. CMR studies (1.5T scanner) sequences included steady state free precession, T2, perfusion and myocardial delayed enhancement (MDE Evaluation included LVEF and RVEF calculation; quantification in grams of: MDE and microvascular obstruction (MVO).
Results: Scans were performed within an average of 5.4 days from admission. MDE was present in 26/27 patients with an average of 20.1 gr (15.7 % of myocardial mass). MVO was present in 19/27 patients with an average of 3.26 gr (2.5% of myocardial mass). LVEF was strongly and negatively correlated with MDE (p=0.005) and MVO (p=0.02). MDE was strongly positively correlated with MVO (p=0.0004). RVEF was not significantly correlated with MDE or MVO. Additional findings on CMR studies included LV thrombus in 3/27 and an unknown old MI in a different territory in 1/27.
Conclusions: CMR for STEMI allows accurate MDE and MVO quantification. The importance of this is stressed by the strong negative correlation between MDE and MVO amount and LVEF. MDE and MVO reflect the extent of myocardial injury and predict functional impairment. CMR enables the detection of additional unexpected findings as well.