olution (STR) after percutaneous coronary intervention (PCI) in STEMI patients enrolled in the ATLANTIC trial.
Methods: ST-segment analysis was performed on electrocardiograms (ECGs) recorded at the time of inclusion [pre-hospital (pre-H) ECG], and 1 hour after PCI (post-PCI ECG) by an independent core laboratory. Complete STR was defined as ≥70% STR.
Results: Complete STR occurred post-PCI in 54.9% (n=800/1456) of patients and predicted lower 30-day composite major adverse cardiovascular and cerebrovascular events (MACCE) (OR 0.35, 95% CI 0.19–0.65; p<0.01), definite stent thrombosis (OR 0.18, 95% CI 0.02–0.88; p=0.03), and total mortality (OR 0.43, 95% CI 0.19–0.97; p=0.04). Post-PCI STR occurred in 57.5% of patients in the pre-H ticagrelor group and in 52.5% of patients in the control group (p=0.055). The degree of STR was significantly greater in the pre-H group (median, 75.0% vs. 71.4%; p=0.049). In multivariate analysis, independent predictors of complete STR were the time from index event to pre-H ECG (OR 0.91, 95% CI 0.85–0.98; p=0.01) and diabetes mellitus (OR 0.6, 95% CI 0.44–0.83; p<0.01); pre-H ticagrelor treatment showed a favourable trend (OR 1.22, 95% CI 0.99–1.51; p=0.06).
Conclusions: Post-PCI complete STR is confirmed to be a valid surrogate marker for cardiovascular clinical outcomes. Coronary reperfusion rates numerically favoured pre-H treatment. In the current era of STEMI reperfusion, patients' delay and diabetes mellitus are independent factors of poor reperfusion and would need specific attention in the future.
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P5547 | BEDSIDE
Early and late changes of multi-marker panel in patients with STEMI after angioplasty

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Background: Post-infarction remodeling is strongly linked with collagen turnover which is influenced mostly by oxidative stress and inflammation, the last being, according to our previous data, triggered by early infiltrated neutrophils (24–48 h) followed by accumulation of macrophages M1 (72 h) and M2 (7–14 days).
Aim: Evaluation of circulating markers of inflammation and oxidative stress in the different periods of 1 year follow up post-infarct evolution: first 2 weeks (each day), 1st month (synthesis of collagen type III), 3rd month (synthesis of collagen type I), 6 and 12 months.
Material and methods: Study was performed in 47 patients (age range of 37–68 years) with STEMI exposed to angioplasty (<12 hours). Circulating levels of 28 markers were determined at admission, 1, 2, 3 ... 14 days, 1, 3, 6 and 12 months. Obtained results were compared with control value of markers determined in 17 apparently healthy persons and admission level (before PCI).
Results: The earliest (first 24 h) significant change was inherent to MMP-8, whose double elevation (averagely from 3,1 up to 6,4 ng/ml) corresponded to period of neutrophil infiltration (24–48 h). Serum levels of IL-1, IL-6 have raised significantly since 48 h, followed by authentic increase of TNF-alpha, IL-8, CRPhs and phospholipase A2 since 72 h. Up to a period of 7 days these markers remained increased, but toward 14th day fell by 24–46% arguably due to macrophage M2 activation. This is consistent to dynamics of anti-inflammatory markers, IL-4 and IL-10 which decreased till 7th, elevated toward 14th day although remained below control. Inflammation boosting was associated by oxidative stress activation during 1st week manifested by malonic dialdehyde (MAD) rise and total antioxidant activity fall. To be noted that markers improvement till 3rd month was poor and a conspicuous dynamics began since 6th month with nearing to control level toward 12th month. However, at this time following markers significantly differed from control value: TNF-alpha (+29,6%), IL-4 (-31,7%), S-nitrosothiols (-17,8%), CRPhs exceeded 3,0 g/L (4,77±0,38) and MAD (+23,6%).
Conclusions: (1). Dynamics of inflammation markers in patients with STEMI during first 14 days after angioplasty conclusive reflect chronologic accumulation of inflammatory cells in necrotic zone. (2). Maximal serum plateau of MMP-8, IL-1, IL-6, IL-6, TNF-alpha and CRPhs goes till 7th day of post-infarct evolution, associated with lowest IL-4 and IL-10. (3). Marker improvements begin since 3rd month with nearing to control toward 12th month, excepting IL-4, TNF-alpha, CRPhs and MAD indicating thus a late statement of inflammation dissemination.

P5548 | BEDSIDE
Comparison of long-term clinical outcomes between patients receiving low-molecular-weight heparin and unfractionated heparin in acute myocardial infarction

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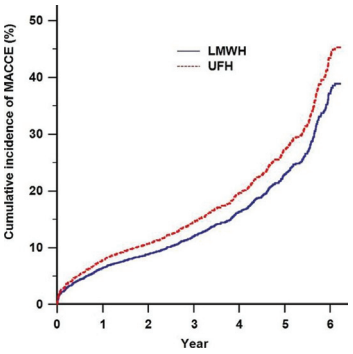
Background: Recent studies show that low molecular weight heparin (LMWH) offers pharmacological and practical advantages over unfractionated heparin (UFH) in acute myocardial infarction (AMI). However, few data exist on the comparison of long-term clinical outcomes between LMWH and UFH in patients with AMI.
Methods: We compared the impact of LMWH to UFH therapy on the long-term clinical outcomes in patient with AMI enrolled in the Convergent Registry of Catholic and Chonnam University for AMI (COREA-AMI). A total of 4,073 patients with AMI were consecutively enrolled from January 2004 to December 2009. Patients were divided into the LMWH or UFH groups according to the type of initially administered heparin. The primary clinical endpoint was a composite of the major adverse cardiocerebrovascular events (MACCE) including all-cause death, nonfatal myocardial infarction and stroke during the 6-year followup.
Results: 657 patients received LMWH (16.1%) and 3416 (83.9%) UFH initially. During 6 years of follow-up, patients who received LMWH had lower rate MACCE (23.6% vs. 20.7%, hazard ratio 0.832, 95% CI 0.694–0.998, p=0.048) than the UFH group. The LMWH groups showed lower rate of all-cause death, cardiac death, nonfatal MI, and nonfatal stroke but did not reach statistical significance (Table 1). After adjusting multiple covariates, treatment with LMWH was associated with a significantly lower rate of MACCE (Hazard ratio 0.807, 95% CI 0.661–0.985, p=0.035).



Cumulative incidence of MACCE

Conclusions: In patients with AMI, initial anticoagulation with LMWH is associated with improved long term clinical outcomes compared to unfractionated heparin.

P5549 | BENCH
Glycocalyx shedding in acute myocardial infarction: interactions with reperfusion and n-acetylcysteine therapy

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Introduction: Conditions of inflammation and oxidative stress can result in damage to the vascular endothelial glycocalyx, increasing vascular permeability and propensity to tissue infiltration by leukocytes. The N-AcetylCysteine In Acute Myocardial infarction (NACIAM) trial (a double-blind, placebo-controlled investigation of the effects of high-dose intravenous N-acetylcysteine [NAC] in PCI-treated acute myocardial infarction [AMI] receiving low-dose glyceryl trinitrate [GTN]) provided a basis to investigate glycocalyx "shedding" during evolving AMI, and its interactions with NAC therapy.
Hypotheses: (a) Glycocalyx shedding emerges post-reperfusion in patients with AMI, and reflects myocardial area at risk.
(b) NAC-induced myocardial salvage is associated with diminution of glycocalyx shedding.
Methods: Patients (n=102) with AMI and ST-segment elevation (STEMI) were evaluated at admission and 3 hours thereafter, having undergone PCI. Randomized therapy was NAC (15g/day) or identical placebo, with GTN infused at 2.5μg/min, for 48 hours. Infarct size, myocardial area at risk (AAR), and myocardial salvage were determined by cardiac magnetic resonance imaging. Plasma concentrations of the glycocalyx marker, syndecan-1 (CD138), were determined by ELISA. Data were subjected to univariate followed by multivariate analyses.
Results: Overall, there was a significant increase in plasma CD138 concentrations (median 60 [42, 92]ng/ml pre-PCI vs. 396 [224, 646] post-PCI, p<0.001).

Abstract P5548 – Table 1. Risk of clinical outcomes in patients using UFH group compared to LMWH using group

	UFH (n=3416)	LMWH (n=657)	Unadjusted HR (95% CI)	P value	Adjusted HR* (95% CI)	P value
MACCE	805 (23.6%)	136 (20.7%)	0.832 (0.694–0.998)	0.048	0.807 (0.661–0.985)	0.035
All-cause death	693 (20.3%)	122 (18.6%)	0.854 (0.704–1.036)	0.109	0.839 (0.679–1.038)	0.106
Cardiac death	394 (11.5%)	85 (12.9%)	1.708 (0.852–1.363)	0.532	1.279 (0.982–1.666)	0.068
Non-fatal MI	80 (2.3%)	12 (1.8%)	1.133 (0.588–2.181)	0.709	0.729 (0.384–1.385)	0.402
Non-fatal stroke	92 (2.7%)	13 (2.0%)	0.728 (0.407–1.302)	0.284	0.577 (0.304–1.094)	0.092