

or 30 days clinical outcomes. Pre-H ticagrelor treatment predicted both lower 30 days ST and new MI without significant interaction with TA.

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#### P4630 | BEDSIDE

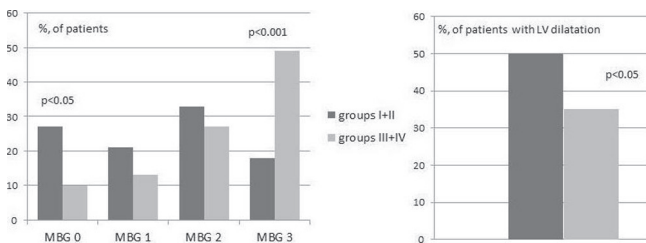
##### Early intensive lipid-lowering therapy prevents development of no-reflow phenomenon and reduces the incidence of post MI LV remodeling

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Statin therapy is recommended for all ACS patients regardless of the initial cholesterol level. Previous studies with statins pre-treatment was associated with the reduction of the no-reflow phenomenon in the patients with reperfused acute myocardial infarction. However, acute effects of different lipid-lowering regimes on microvascular perfusion recovery and left ventricular remodeling in STEMI patients have been insufficiently studied.

The study involved 135 STEMI patients admitted an average of 4.5 hours after symptoms onset and treated with primary PCI. Lipid-lowering treatment was prescribed immediately after hospital admittance. Patients were randomly assigned to one of four groups treated by average (group I and group II) or high (group III and group IV) intensity lipid-lowering therapy. Group I (26 patients) was assigned to atorvastatin 10 mg /ezetimibe 10 mg combination, group II (24 patients) - to atorvastatin 40 mg, group III (42 patients) - to atorvastatin 40 mg/ ezetimibe 10 mg combination, and group IV (43 patients) - to atorvastatin 80 mg. Myocardial Blush Grade (MBG) was used for angiographic assessment of myocardial reperfusion. Echocardiography was performed to all the patients during the first 24 hours after symptoms onset and 90 days after MI development. LV dilatation was defined as at least 25% increase of end-diastolic volume.

Intensive lipid-lowering therapy helped to preserve the microvascular integrity after AMI leading to better functional recovery. MBG 3 was significantly often detected in pts from groups III and IV ( $p < 0.001$ ). Whereas patients in groups I and II significantly often had no-reflow phenomenon ( $p < 0.05$ ) and showed of post-MI LV dilatation after 3 month of treatment in 50% of cases. High intensity lipid-lowering therapy (figure 2) reduced the risk of LV remodeling by 30% ( $p < 0.05$ ).



**Conclusion:** The use of high-intensity lipid-lowering therapy early after STEMI can prevent no-reflow phenomenon during primary PCI and reduce the incidence of post-MI LV dilatation.

#### P4631 | BEDSIDE

##### Rapid rule-out of suspected acute coronary syndrome in the Emergency Department by high-sensitivity cardiac troponin T levels at presentation

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**Background:** The combination of an electrocardiogram (ECG) without signs of ischemia and undetectable (i.e.  $< 5$  ng/L) high-sensitivity cardiac troponin T concentrations at presentation could represent a safe strategy to rapidly rule out myocardial infarction (MI) in subjects with chest pain.

**Purpose:** To explore the negative predictive value of this strategy in ruling out the short and long-term occurrence of acute coronary events through the retrospective analysis of a single centre database of patients visited in the Emergency Department (ED) for suspected for acute coronary syndrome.

**Methods:** We retrospectively evaluated (from January to December 2015) 1,640 subjects with symptoms suspected for acute coronary syndrome who had the initial ECG without signs of ischemia and hs-cTnT measured at presentation and after 3 hours. According to admission hscTnT levels, patients were divided into two groups: those with levels below the lower limit of detection (LoD;  $< 5$  ng/L), and those with levels between 5 ng/L and 14 ng/L (99th percentile). Patients with hscTnT levels  $> 14$  ng/L were excluded from the present analysis. The primary outcome was fatal or nonfatal type 1 MI within 30 days of the ED visit. The secondary outcome measures were the combination of fatal or nonfatal type 1 MI or hospitalization for unstable angina (UA) during the same period, and the occurrence of fatal or nonfatal type 1 MI after 1 year.

**Results:** 1,001 patients had initial hscTnT levels  $\leq 14$  ng/L (mean age:  $62.2 \pm 14.7$

years, male: 54.9%). Among 326 subjects (32.5%) with admission hs-cTnT  $< 5$  ng/L (1 fatal or nonfatal type 1 MI was recorded within 30 days, while 11 type 1 MIs (1 fatal and 10 nonfatal, 1.6%) occurred among 675 subjects (67.5%) with admission hs-cTnT levels between 5 and 14 ng/L. Therefore, the negative predictive value (NPV) for the primary outcome measure was 100%. The combination of type 1 MI or hospitalization for UA was recorded in 4/326 subjects with hs-cTnT  $< 5$  ng/L (1.2%; 95% CI: 0.4 – 2.9). Among the 675 subjects with hs-cTnT 5–14 ng/L, 26 events of this secondary outcome were recorded (3.8%, 95% CI: 2.6 to 5.5%), implying a NPV of 98.8% (95% CI, 97.0%–99.5%). The secondary outcome (fatal or nonfatal type 1 MI) at 1 year follow-up mirrored the short-term results.

**Conclusions:** The combination of an ECG without signs of ischemia and admission hs-cTnT level below LoD (i.e. a single sample strategy) has high NPV for acute coronary events both at short- and long-term, suggesting that about 1/3 of patients presenting to the ED for suspected acute coronary syndrome can be rapidly and safely discharged without additional diagnostic tests.

#### P4632 | BEDSIDE

##### Impact of body mass index on hospital mortality in acute myocardial infarction over 15 years: Findings from 27,607 patients of a local myocardial infarction registry

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**Background:** Earlier studies have shown a paradox association of obesity with better outcome in patients with acute myocardial infarction (AMI). Treatment of AMI has changed fundamentally over the past 15 years. In this study we examined the impact of body weight on outcome in patients with AMI and changes over a period of 15 years (from 2001–2015).

**Methods:** Data from the prospective local myocardial infarction registry were analyzed enclosing 27,607 patients treated for AMI in hospitals of Berlin between 2001 and 2015. We studied BMI classes over time and influence of BMI on hospital mortality.

Patients were categorized as underweight (1.3%; BMI  $< 18.5$ ), normal (32.1%; BMI 18.5 to  $< 25$ ), overweight (42.6%; BMI 25 to  $< 30$ ), obesity I (17.1%; BMI 30 to  $< 35$ ), obesity II (4.9%; BMI 35 to  $< 40$ ) and obesity III (2.0%; BMI  $> 40$ ). Three-year periods were analyzed (2001–03: n=3056; 2004–06: n=3646; 2007–09: n=5812; 2010–12: n=6964; 2013–15: n=8129).

**Results:** The total hospital mortality declined during this period from 9.5% (2001–03) to 5.4% (2013–15). Treatment with PCI increased in this period from 55.4% to 83.3% and thrombolysis declined from 24.2% to 0.8%.

Highest in-hospital mortality after AMI was observed in underweight patients with 13.7%. By contrast, lowest mortality was observed in the group of obesity II with 4.2%, followed by obesity I with 5.0% (overweight: 5.6%; obesity III: 5.9%, and normal weight with 7.1%).

In the multivariate logistic regression, including confounding factors (age, sex, STEMI, smoking, Diabetes, hypertension, hypercholesterolemia, previous infarction, previous PCI, previous CABG, CHF, atrial fibrillation, chronic renal failure and primary PCI), the lowest mortality risk was found in patients with a BMI 35 to  $< 40$  (OR=0.71; 95% CI: 0.47–1.09), followed by obesity I (OR=0.79; 95% CI: 0.61–1.01), overweight (OR=0.83; 95% CI: 0.69–0.99) and obesity III (BMI  $> 40$ , OR=1.18; 95% CI: 0.67–2.09) each compared to normal weight.

In the underweight patients survival is worst (OR = 2.02; 95% CI: 1.22–3.36) compared to normalweight patients.

##### development of hospital mortality over time in the different BMI groups

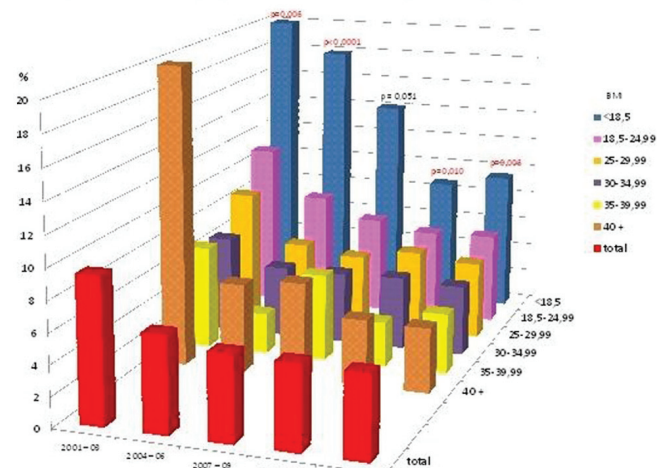


Figure 1

The development of hospital mortality over time in the different BMI groups is represented in figure 1.

The inverse relation of higher BMI and lower mortality maintained throughout treatment periods 2001–2015. However, a U-shaped relation (2001–03) was replaced by a more J-shape relation as also very obese patients showed no increased mortality in recent years.

**Conclusion:** The obesity paradigm in patients with acute myocardial infarction is confirmed in this large registry and is maintained despite changes in acute coronary therapy over time. Patients with overweight and obese patients show a better survival after AMI than normalweight. By contrast, patients with underweight have increased risk of mortality. The obesity paradigm is independent of smoking status and comorbidities or treatment regimen.

**P4633 | BEDSIDE**  
**Relationship between circadian variation and plaque characteristics in patients with ST-segment elevation myocardial infarction**

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**Background:** The relationship between circadian variation and plaque characteristics have never been investigated in acute myocardial infarction.

**Aims:** To examine the effects of circadian variation on plaque characteristics in patients with ST-segment elevation myocardial infarction (STEMI).

**Methods:** This study included 106 patients with STEMI who underwent primary percutaneous coronary intervention within 24 h of onset. Optical coherence tomography was used to assess plaque features of the culprit lesion. Patients were divided into two groups: morning onset group (myocardial infarction onset between 06:00 to 11:59) and the others.

**Results:** Thirty three patients (31.7%) were in the morning onset group. There was no difference in minimal lumen area, length of thrombi, plaque erosion and incidence of plaque rupture between the two groups. However the presence of thin-cap fibroatheroma (TCFA) was significantly higher in patients of the morning onset group than in the others (76% vs. 41%, p=0.0005). Also, the fibrous cap thickness was significantly thinner in the morning onset group than in the others (72.5±20.1 μm vs. 92.9±37.1 μm, p=0.0034). Multivariable logistic regression analysis adjusted for morning onset, age ≥75 years, hypertension, diabetes mellitus, dyslipidemia, and current smoking identified morning onset as an independent predictor of the presence of TCFA (odds ratio = 5.2, 95% confidence interval: 2.05–14.3, p=0.0004).

Predictor of the presence of TCFA

	Odds ratio (95% CI)	p value
Morning onset	5.18 (2.05–14.3)	<0.001
Age ≥75yo	1.84 (0.69–5.07)	0.222
Hypertension	1.17 (0.42–3.33)	0.766
Diabetes mellitus	1.23 (0.52–2.91)	0.635
Current smoking	1.35 (0.50–3.71)	0.549
Dyslipidemia	1.44 (0.53–4.02)	0.467

**Conclusion:** The main findings are that patients with morning onset of STEMI may have more vulnerable plaque characteristics.

**P4634 | BEDSIDE**  
**The clinical impact of chronic total occlusion on acute myocardial infarction patients from mie acs registry**

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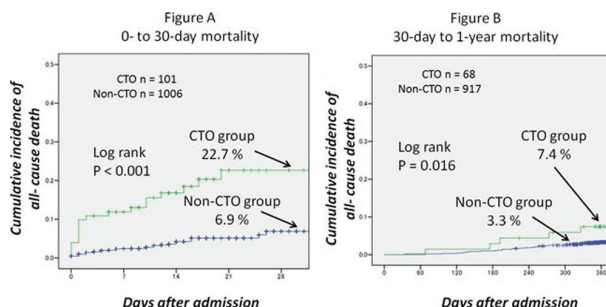
**Objective:** Previous studies reported the concomitant chronic total occlusion (CTO) in a non-infarct-related-artery (IRA) in the setting of acute myocardial infarction (AMI) was associated with poor prognosis. However, these studies were confounded with several limitations such as relatively small sample and retrospective studies of single center.

**Methods:** We analyzed consecutive 1107 patients (861 men, and mean age 68±13 years old) with AMI who were enrolled in the Mie ACS Registry, which is a prospective and multicenter registry consisting of 15 hospitals with cath labo facilities in Japan, between January 2013 and December 2014. Patients were divided into two groups (CTO group: patients with CTO in non-IRA, n=101; non-CTO group: patients without CTO, n=1006). Primary end point was defined as all-cause mortality.

**Results:** During average follow-up time of 406 days (range, 1 to 1052 days), 112 patients (10.1%) reached primary end point. The clinical characteristics of the 2 groups were shown in the table. CTO group had higher prevalence of Killip class 3 to 4 and received less emergent PCI procedure. The 30 days mortality of CTO group was significantly higher than that of non-CTO group (Figure A; 22.7% versus 6.7%; Logrank p<0.001), and 30-day to 1-year mortality of CTO group were also significantly higher (Figure B; 7.4% versus 3.3%; p=0.016). In multivariate analysis, the concomitant CTO was an independent predictive factor for 0- to 30-day mortality (hazard ratio: 2.84; 95% confidence interval (CI): 1.64–

4.91, p<0.001), but not for 30-day to 1-year mortality (hazard ratio: 1.43; 95% CI: 0.64–3.20, p=0.39).

Clinical characteristics	CTO (n=101)	Non-CTO (n=1006)	P value
Age	70.0±12.7	67.5±12.9	0.056
Male	80 (79.2%)	781 (77.6%)	0.80
DM	38 (37.6%)	333 (28.6%)	0.067
Cr	1.11±0.83	0.97±0.79	0.11
Killip classification			<0.001
I	51 (51%)	815 (81%)	
II	18 (18%)	85 (8.4%)	
III	13 (13%)	43 (4.3%)	
IV	19 (19%)	57 (5.7%)	
Emergent PCI	77 (76%)	900 (90%)	<0.001



**Conclusions:** The concomitant CTO in a non-IRA of AMI patients was the independent predictor of short term outcomes, but not of mid-term outcomes.

**P4635 | BEDSIDE**  
**Mortality rate of patients with atrial fibrillation in STEMI, and mortality prediction factors in this high risk group**

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**Background:** Atrial fibrillation (AF) is related to major adverse cardiac events (MACEs) in acute myocardial infarction (AMI).

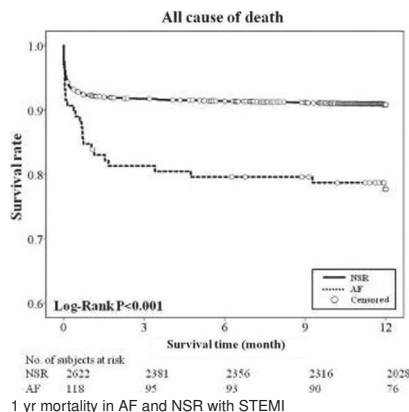
**Purpose:** We tried to show the clinical impact of AF in STEMI patients who underwent primary PCI, and predictive factors to mortality in this group of patients.

**Methods:** To compare the 1 year mortality rate between AF and normal sinus rhythm (NSR) in STEMI patients who presented ER <12 hours after chest pain onset, and underwent primary PCI within 90 min. after ER arrival, we used the Korean Acute Myocardial Infarction Registry (KAMIR) which enrolled AMI patients (N=14,329) from 2008 to 2009.

**Results:** We discarded data of NSTEMI or ambiguous ECG rhythm (N=6,133), delayed presentation (N=2,188), delayed PCI (N=652), incomplete 1 year follow-

Table 1. Clinical predictive factors for mortality in AF with STEMI

	Hazard ratio	95% CI	p-value
Age (year)	1.60	0.97–2.66	0.069
cTnI (1 pg/ml)	15.79	1.62–154.21	0.018
Triglyceride (1 mg/dL)	1.04	1.00–1.08	0.044
LDL-C (1 mg/dL)	0.89	0.80–0.99	0.028
NT-proBNP (1 SD)	3.37	1.34–8.49	0.010
PCI result (unsuccessful)	859.68	2.27–325387.8	0.026



1 yr mortality in AF and NSR with STEMI