cardiopathy (28%), followed by plaque disruption (25.3%). Coronary spasm was the mechanism in 8.5%, and coronary emboli in 6.4% of the patients. We only found a 3.2% of coronary dissection. Six percent were type 2 myocardial infarction and 3.2% were a miscellaneous group of mechanisms. In 7.9% of the patients, we did not find the mechanism. Only 11.1% of the patients initially included in the working diagnosis of MINOCA were finally diagnosed of myocardial infarction. There was no mortality during the admission and follow up (Median 9 months) in the MINOCA group, but 8.7% had major adverse cardiovascular events, 12.1% manifested a functional class worse than II and they had a 29.6% rate of rehospitalization. Traditional cardiovascular risk factors were not related to the prognosis of MINOCA patients.

Conclusion: MINOCA frequency is high in the real world (9.3% of patients initially diagnosed of myocardial infarction). Clinical evolution in this type of patients is different one requiring revascularization, although it is not as benign as it was thought. The most frequent mechanism (28%) was stress myocardiopathy. Further studies are necessary to offer the best management possible to MINOCA patients.

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Impact of the definition on incidence and prognosis of type 2 myocardial infarction
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Background: Uncertainties regarding the most appropriate definition and treatment of the 2007 universal definition (T2MI2007), and once not requiring CAD, the mechanism in 9.5%, and coronary emboli in 6.4% of the patients. We only identified 4 and 7 biomarkers differentiating MINCA/MI-CAD and MINCA/control, or MINCA from controls. The results indicate differences in the activation of several physiological pathways between MINCA patients and healthy controls, and between MINCA and MI-CAD patients, three months after the index event. These pathways include inflammation, coagulation/fibrinolysis, renin-angiotensin system activation, arteriosclerosis and cardiac dysfunction. However, the results must be interpreted cautiously because of the small sample size.

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High sensitivity troponin T in combination with risk scores for suspected acute coronary syndromes: An comprehensive evaluation of HEART, GRACE and TIMI scores
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Background: The optimum risk score for suspected acute coronary syndromes (ACS) would, in a large proportion of patients, exclude MACE in those deemed low risk allowing early, safe discharge and early identification of ACS thus allowing prompt treatment. In the high sensitive troponin (hsTnT) era this risk score remains undefined.

Purpose: To comprehensively evaluate the ability of HEART, GRACE and TIMI scores to correctly classify an unselected consecutive suspected ACS population to low and high risk allowing prompt management.

Methods: Consecutive patients presenting to a large secondary care facility with chest pain suspicious for myocardial ischaemia, with hsTnT measurement and presentation electrocardiogram (ECG) were included for analysis. All biomarker positive events at index or readmission underwent two physician, blinded adjudication. MACE rates according to risk classification and ROC curves were computed for each score.

Results: 1642 (median age 59, 52% male) consecutive suspected ACS were included for analysis, there was 100% follow up of readmissions. 169 (10.3%) were adjudicated to have suffered a type 1 MI at index presentation. MACE at 30 days and 1 year occurred in 39.2% (16.7%) and 71.6%, 65.5%, 47.7% and 62.5% of patients were classified to low or high risk using HEART, GRACE and TIMI respectively. ROC analysis demonstrated AUCs (95% Cis) 0.92 (0.91–0.94), 0.79 (0.76–0.82) and 0.86 (0.83–0.88) for HEART, GRACE and TIMI.
Heart rate after resuscitation predicts clinical outcomes in patients with out-of-hospital cardiac arrest due to acute coronary syndrome

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Background: Bradycardia (<60 bpm) is a typical unfavorable side effect during therapeutic hypothermia (TH) in patients resuscitated from cardiac arrest due to acute coronary syndrome (ACS). However, the associations between heart rate (HR) during TH and patients’ clinical outcomes are not well known.

Purpose: We investigated the impacts of HR during TH on clinical outcomes after resuscitation, and whether bradycardia during TH was an unfavorable side effect in patients with cardiac arrest due to ACS.

Methods: Clinical data from 3,687 out-of-hospital cardiac arrest (OHCA) patients between October 2002 and October 2014 were retrospectively analyzed. Of 154 ACS patients underwent successful percutaneous coronary intervention, 85 patients performed TH (target temperature was 33–34°C, procedure time was 24–48 hours) were divided into two groups according to their mean HR during TH (lower HR group <60 bpm, higher HR group >75 bpm; the cut off value was median HR). In the present study, we evaluated about 1 year survival rate.

Results: Overall, 1 year survival rate was 40.0%. The mean HR during TH was 78.7 bpm. Lower HR group had 44 (51.8%) patients, including 18 (21.2%) bradycardia patients; 16 patients did not receive therapeutic interventions for bradycardia. When divided into two groups, patient’s characteristics were not significantly different. However, 1 year survival rate in lower HR group was better than that of higher HR group (85.0% vs. 34.9%, p<0.0001). In univariate analysis, lower HR during TH, shorter time to return of spontaneous circulation, preserved left ventricular function (<40%), and better renal function were significantly associated with higher survival rate at 1 year. Furthermore, a multivariate analysis model which included these factors showed that lower HR was an independent predictor of 1 year survival rate (HR 4.37; CI, 1.01–19.1; P=0.049), while the relationship between tachycardia and mortality was not shown in non-ACS patients (n=116).

Conclusion: Bradycardia was an independent predictor of 3 months all-cause mortality in patients resuscitated from cardiacogenic OHCA, especially due to ACS.