ture cardiovascular events and might help guide the selection of more aggressive therapy such as monoclonal antibody to PCSK9.

P1728

Impact of irregular protrusion immediately after stent implantation on mid-term vascular healing in ST elevation myocardial infarction: Results from MECHANISM AMI optical coherence tomography study

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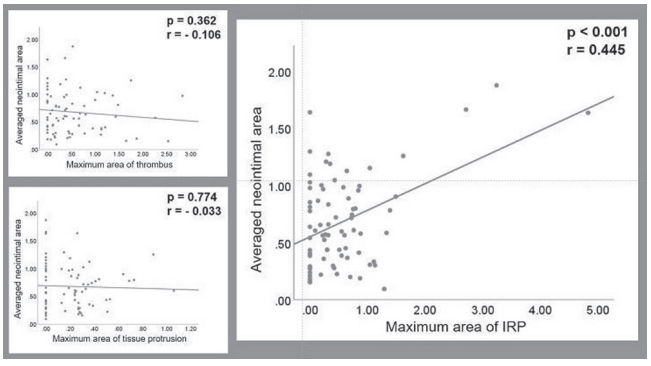
Background: Irregular protrusion (IRP) post stent implantation was observed more frequently in patients with ST elevation myocardial infarction (STEMI) compared to those with stable angina (SAP). It was reported that IRP detected by Optical coherence tomography (OCT) was related to neointimal hyperplasia formation in stented site, which induced independent predictor of target lesion revascularization (TLR) in non-myocardial infarction settings. However, the association between IRP and neointimal hyperplasia in STEMI have not been elucidated.
Purpose: The aim of this study was to clarify the impact of IRP detected by OCT on late vascular healing in patients with STEMI.
Methods: A total of 102 patients with STEMI were registered in MECHANISM-AMI trial. OCT images were acquired immediately and 12 months after index PCI. ISTs were classified to thrombus, irregular protrusion (IRP) and smooth protrusion. We measured the maximum area of each ISTs within the stented segment post index PCI and averaged neointimal area 12 months after index PCI.
Results: Finally, 90 patients (90 stents) enrolled in MECHANISM-AMI trial. There was significant positive correlation between maximum area of IRP post stent implantation and averaged neointimal area 12 months after stent implantation ($r=0.445$, $p<0.001$). However, there were not significant correlation between maximum area of thrombus and smooth protrusion and averaged neointimal area (Figure).



Conclusion: MECHANISM-AMI trial demonstrated that IRP post index PCI for patients with STEMI was associated with higher neointimal hyperplasia 12 months after index PCI. This observation might support the hypothesis that IRP post index PCI was related with 1-year target lesion revascularization in STEMI.

P1729

Low skeletal muscle mass is associated with poor cardiovascular outcome in patients after ST-segment elevation myocardial infarction

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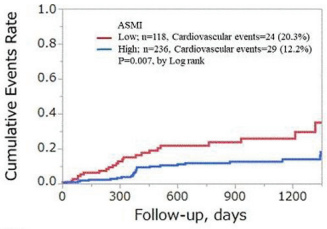
Introduction: Recent studies have demonstrated the importance of sarcopenia, a geriatric syndrome defined as age-related decline in skeletal muscle mass and low muscle strength. Also in patients with ST-segment elevation myocardial infarction (STEMI), indices of muscle strength (ie, gait speed and hand-grip strength) have been known as a prognostic marker, whereas the prognostic value of skeletal muscle mass is unclear.
Hypothesis: We assessed the hypothesis that low appendicular skeletal muscle mass index (ASMI) is associated with an increased risk of secondary cardiovascular events in patients after STEMI.
Methods: We enrolled 354 STEMI patients. ASMI was evaluated with dual-energy X-ray absorptiometry before discharge. The patients were divided into low and high ASMI groups using the lowest tertile of ASMI ($\leq$6.814 kg/m^2 for men and $\leq$5.271 kg/m^2 for women). Patients were followed for cardiovascular events, which consist of death, myocardial infarction, ischemic stroke, congestive heart failure, and revascularization.
Results: During the follow-up period (median 26 months [interquartile ranges 11 and 40 months]), 53 patients experienced cardiovascular events (11 death, 9 myocardial infarction, 6 ischemic stroke, 7 congestive heart failure, and 20 revascularization). The event rate at 26 months after STEMI was significantly higher in patients with low ASMI than those with high ASMI (20.3% vs 12.2%, log-rank p=0.007) (Figure). Even after adjustment with age, gender, renal function, and infarct size, patients with low ASMI had 2.0-fold higher risk of cardiovascular events compared to those with high ASMI (Adjusted hazard ratio 1.97, 95% confidence interval 1.07 to 3.61, p=0.029).



No. at Risk					
Low ASMI	118	76	55	38	20
High ASMI	236	188	145	105	53

Kaplan-Meier estimates of cumulative incidence of cardiovascular events based on Appendicular Skeletal Muscle Mass Index tertiles. The cut-off values defining the low Skeletal Muscle Mass Index were $\leq$6.814 kg/m^2 for men; and $\leq$5.271 kg/m^2 for women. Red line indicates Low ASMI; Blue line indicates High ASMI. ASMI = Appendicular Skeletal Muscle Mass Index

Conclusion: In patients after STEMI, low ASMI was significantly and independently associated with an increased risk of poor cardiovascular outcome.

P1730

Redefining unstable angina: novel insights regarding incidence, patient characteristics, pathophysiology and outcome

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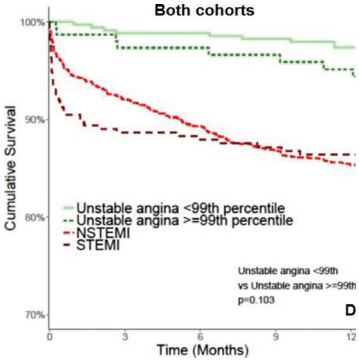
Background: Unstable angina and non-ST-elevation myocardial infarction (NSTEMI) are often thought to have similar incidence, characteristics, pathophysiology, and outcome, and are therefore treated similarly.
Objectives: Assess the incidence and compare characteristics and outcome of unstable angina and NSTEMI
Methods: We enrolled 8992 patients with acute chest discomfort presenting to the emergency department from two large multicenter studies (4122 APACE and 4870 High-STEACS). Final diagnosis was adjudicated by two independent cardiologists using all clinical information including serial measurements of high-sensitivity cardiac troponin (hs-cTn).
Results: Unstable angina was adjudicated in 366/4122 (8.9%) and 137/4870 (2.8%) patients in APACE and High-STEACS, respectively, and NSTEMI in 622 (15.1%) and 651 (13.4%). Coronary artery disease was pre-existing in 73% and 76% of patients with unstable angina. At one-year, all-cause mortality in APACE was 3.3% (95%-confidence interval 1.2–5.3) in unstable angina, which was substantially lower as compared to NSTEMI (10.4%, 7.9–12.9), and similar to non-cardiac chest pain (NCCP) (2.3%, 1.6–3.0). This difference was confirmed in High-STEACS, with a one-year mortality of 5.1% (0.7–9.5) in unstable angina, 22.9% (19.3–26.4) in NSTEMI, and 10.6% (9.5–11.7) in non-coronary chest pain. In contrast, the rate of future MI in APACE was comparable in unstable angina and NSTEMI (11.2%, 7.8–14.6 and 7.9%, 5.7–10.2), and higher than in NCCP (0.6%, 0.2–1.0), especially in patients with stable hs-cTn levels above the 99th percentile.



Conclusions: Major differences in pathophysiology, patient characteristics, and outcomes between patients with unstable angina and patients with NSTEMI sug-

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