Conclusion: In patients with no-reflow, oxidants are increased, while serum paraoxonase-1 activity and antioxidants are decreased. This result shows that increased oxidative stress may have role in the pathogenesis of no-reflow.

P5518 | BEDSIDE
Influence of endothelial progenitor cell capturing stent on coronary microvascular function in drug eluting stent era

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Background: Although positive effects of Drug eluting stent (DES) reduce inflammation and restenosis, negative effects delay re-endothelialization and impair microvascular function. Delayed re-endothelialization and impaired microvascular function are linked to stent thrombosis and adverse clinical outcomes after DES use. Rapid re-endothelialization by an endothelial progenitor cell capture (EPC) stent was demonstrated in various preclinical studies. Here we investigated the effect of a novel progenitor cell-eliciting stent on coronary microvascular function related to EPC stent have been seldom investigated and reported.

Methods: Seventy four consecutive patients undergoing percutaneous coronary intervention including stable angina and acute coronary syndrome were enrolled in the study. Microvascular function was estimated after intervention at 6-month follow-up by measuring index of microvascular resistance (IMR) using intracoronary pressure temperature sensor-tipped guidewire. Thermolitication curves were obtained during maximal hyperemia. The IMR was calculated from the ratio of the mean distal coronary pressure at maximal hyperemia to the inverse of mean hyperemic transit time.

Results: Twenty one patients (age: 67±9 years, M/F=12:7) received EPC stent. 53 patients (age: 61±12 years, M/F=20:26) received 2nd generation drug eluting stent (zotarolimus eluting stent or everolimus eluting stent). There were no significant differences in baseline clinical and angiographic characteristics. At 6-month follow-up, we measured pulse wave velocity in both the Complior (carotid to femoral-PWVc) and the arterial waveform analysis (PWVva) to assess wave reflections in CAD patients.

Conclusion: Seventy four consecutive patients undergoing percutaneous coronary intervention including stable angina and acute coronary syndrome were enrolled in the study. Microvascular function was estimated after intervention at 6-month follow-up by measuring index of microvascular resistance (IMR) using intracoronary pressure temperature sensor-tipped guidewire. Thermolitication curves were obtained during maximal hyperemia. The IMR was calculated from the ratio of the mean distal coronary pressure at maximal hyperemia to the inverse of mean hyperemic transit time.

P5519 | BEDSIDE
Lipoprotein-phospholipase A2 is associated with increased arterial stiffness and abnormal wave reflections linked with impaired coronary flow in patients with CAD

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Lipoprotein phospholipase A2 (Lp-PLA2) is an emerging inflammatory marker with prognostic value. Aortic wall properties and wave reflections determine coronary perfusion, LV function and have an independent prognostic value. We investigated the association of Lp-PLA2 with arterial stiffness and abnormal wave reflections in CAD patients.

Methods: In 70 patients with angiographically documented CAD we measured pulse wave velocity using both the Compilor (carotid to femoral-PWVc) and Arteriograph apparatus (PWA-cologmic method). By means of pulse wave analysis (Arteriograph apparatus) we calculated the augmentation index (AI) of the arterial wave reflection, the diastolic area (DAI) of the aortic pulse wave and distal reflection area (DRA). We assessed wave reflection, patients were also categorised into 2 subgroups according to the mean distal coronary pressure at maximal hyperemia to the inverse of mean hyperemic transit time. The mean distal coronary pressure at maximal hyperemia to the inverse of mean hyperemic transit time.

Results: In patients with no-reflow, oxidants are increased, while serum paraoxonase-1 activity and antioxidants are decreased. This result shows that increased oxidative stress may have role in the pathogenesis of no-reflow.

Conclusion: 25-hydroxyvitamin D level is reduced in proportion to the extent and complexity of CAD. Moreover, 25-hydroxyvitamin D may play a role on pathogenesis and severity of coronary atherosclerosis.

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Obstructive sleep apnea is associated with increased inflammatory activity in non-obese patients with coronary artery disease: a cross sectional study

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Background: Inflammation has a crucial role in the development of coronary artery disease. Obstructive sleep apnea (OSA) is a common condition in CAD patients and associated with increased inflammation. The impact of OSA on inflammatory activity in CAD is unknown.

Methods and results: Inflammatory markers, High-sensitive (Hs)-CRP, Interleukin (IL)-6, IL-8 and Tumor Necrosis Factor (TNF)α were studied in a non-obese cohort of revascularized CAD patients with OSA (Apnea Hypopnea-Index [AHI] ≥15 events/h; n=267) and non-OSA (AHI <15 events/h; n=104), respectively. CAD patients with OSA had higher levels of Hs-CRP (3.02 vs 2.41 mg/ml; P<0.001), IL-6 (11.66 vs 6.27 pg/ml; p<0.001), IL-8 (1.81 vs 1.52 pg/ml; P=0.075) and TNFα (6.06 vs 5.41 pg/ml; P=0.019) compared to the levels seen in non-OSA. In a univariate regression analysis, OSA, based on AHI≥15 events/h was significantly correlated with all four biomarkers but only with IL-6 in the multivariate model (Odds Ratio [OR] 1.33, 95% Confidence Interval [CI] 1.06; 1.65). Applying Oxygen Desaturation Index (ODI) ≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5 events/h and Hs-CRP (OR 1.28; 95% CI 1.02; 1.62) as well as between ODI≥5}