(median 26 vs. 35y, p<0.001) and more symptomatic, but had no differences in echocardiographic parameters as compared to those with isolated C disease (all p=n.s); 3) cardiac structural phenotype showed worse outcome and significantly higher MVA occurrence than pure electrical disease (32% vs. 4%, p<0.001); 4) overall, optimal treatment including betablockers, ACE-inhibitors and CRT had no considerable impact on prognosis (all p=n.s).

Conclusion: This large collaborative study showed that an integrated C and N evaluation allows for a significant improvement in the knowledge of both natural history of laminopathies and risk stratification in LMNA-associated heart disease.

COMPLEX DECISIONS IN CORONARY INTERVENTIONS

P3171

Impact of untreated coronary artery disease after primary percutaneous coronary intervention on two years clinical outcome: the residual added index

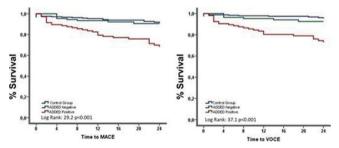
L. Di Serafino, F. Magliulo, F. Ziviello, M. Esposito, P. Cirillo, E. Stabile, B. Trimarco, G. Esposito. *Federico II University Hospital, Division of Cardiology - Dpt of Advanced Biomedical Sciences, Naples, Italy*

Background: Incomplete revascularization after PCI is common, particularly in patients presenting with acute coronary syndromes, and it is associated with a worse prognosis as compared with complete revascularization. In patients presenting with STEMI and multivessel disease, a FFR-based complete revascularization significantly reduces the risk of future events. The Added index, namely the ratio between minimal lumen diameter (MLD) and Duke Jeopardy Score (DJS), showed high accuracy in predicting functionally significant coronary artery stenosis with a cutoff value of 2.23.

Purpose: The aim of the present study was to investigate the prognostic value of the residual Added Index after primary PCI (pPCI) in patients presenting with STEMI and intermediate non-culprit stenosis.

Methods: We included 605 STEMI patients treated with successful pPCI. Patients with previous coronary artery bypass grafts (CABG) or presenting with cardiogenic shock were excluded. Patients were divided into 3 groups: 1) Patients without any residual coronary artery stenosis (n=321, Control group); 2) Patients with at least one residual coronary artery stenosis with a residual ADDED index lower than 2.23 (n=145, ADDED Negative group); 3) Patients with at least one residual coronary artery stenosis with a residual ADDED index equal to or higher than 2.23 (n=139, ADDED Positive group). Primary end points were: 1) major adverse cardiac event (MACE), defined as overall death, myocardial infarction, repeated revascularizations; 2) non-target vessel oriented adverse cardiac event (VOCE), defined as overall death, non-target vessel related myocardial infarction and non-target vessel related urgent or not urgent revascularizations.

Results: Patients included in the ADDED Positive group were older with higher prevalence of diabetes mellitus, hypertension and peripheral artery disease as compared with either Control group and ADDED Negative group. In addition, pPCI of the left anterior coronary artery was more often performed in both Control and ADDED Negative groups as compared with ADDED Positive group in which pPCI was more often performed in the right coronary artery. Follow-up was obtained in 77% of patients at a median of 24 months (14–36 months). Figure 1 shows the Kaplan Mayer analysis for both MACE and VOCE. At multivariate analysis, MACE rate was significantly higher in the ADDED Positive group as compared with ADDED Negative and Control Group (respectively, 29 [27%] vs 9 [8%] vs 18 [7%], adjusted hazard ratio 2.43 [1.38–4.29], p=0.002]) as well as the rate of VOCE (respectively, 25 [23%] vs 7 [6%] vs 9 [4%], adjusted hazard ratio 4.87 [2.17–10.00], p<0.001]).



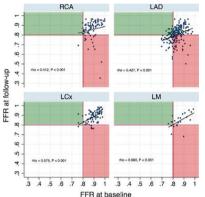
Conclusions: After pPCI, deferring treatment of non-culprit lesions with a positive residual ADDED index value is associated with a significant higher risk of cardiovascular events at 2 years.

P3172 Non-uniform temporal evolution of fractional flow reserve (FFR) in intermediate coronary lesions: what matters?

P.X. Xaplanteris, B. De Bruyne, T. Strisciuglio, M. Pellicano, S. Fournier, G. Ciccarelli, E. Barbato. *Cardiovascular Center Aalst, Aalst, Belgium*

Background/Introduction: To explore if FFR evolves uniformly over time in coronary lesions according to their location and their baseline hemodynamic status.

Methods: A retrospective, single-center analysis of all coronary stenoses that were functionally evaluated by consecutive FFR measurements at two time points at least six months apart was performed. Stenoses in vessels that had been revascularized by percutaneous coronary intervention (PCI) and/or coronary artery bypass grafting (CABG) between the two time points were excluded. Results: 414 stenoses from 331 patients were analyzed with a time interval between the FFR measurements of 24 (17, 37) months. FFR follow-up was significantly lower than FFR baseline [0.83 (0.79, 0.90) versus 0.86 (0.82, 0.90), P<0.0001] and the overall dFFR was -0.007 (-0.028, 0.010) per year. In mixed effect repeated measures analysis, only lesion location had an independent correlation with FFR values after adjusting for multiple confounders. The rate of FFR change was not uniform for all stenosis, with faster progression for LAD lesions. The stenoses with higher FFR baseline values deteriorated more rapidly as compared to those with lower FFR baseline values. The rates of change for stenoses with FFR baseline 0.70-0.79, 0.80-0.89 and 0.90-1.00 were 0.010 (-0.011, 0.022), -0.005 (-0.026, 0.010) and -0.018 (-0.035, 0.013) respectively, with P=0.0001 for the Kruskall-Wallis test.



FFR at baseline and follow-up per vessel

Conclusions: Faster functional atherosclerosis progression (lower FFR values) is observed with LAD stenoses. From a practical standpoint, our data suggest to systemically re-assess with FFR intermediate LAD lesions at the occasion of a repeat angiogram. Similarly, progression (or regression) rate of FFR also differs according to baseline values; lesions that have the higher FFR baseline (in the range of 0.9–1.00) deteriorate faster.

P3173

Anatomic complexity and lipid plaque burden in NSTE-ACS smoker patients with or without COPD: insights from the SCAP trial

S. Biscaglia, R. Ruggiero, A. Erriquez, F. Gallo, M. Tebaldi, C. Tumscitz, C. Penzo, G. Campo. *University Hospital of Ferrara, Medical Sciences, Ferrara, Italy*

Background: The effects of COPD on coronary artery lesions are unknown. The Screening for COPD in ACS patients (SCAP) trial enrolled acute coronary syndrome (ACS) patients with smoking habit without known COPD to detect undiagnosed COPD (UCOPD).

Purpose: To evaluate the anatomic complexity (Syntax score) and plaque lipid core burden index (LCBI through near infrared spectroscopy (NIRS)) in patients hospitalized for no ST-elevated (NSTE)-ACS enrolled in the SCAP trial.

Methods: The SCAP trial study protocol mandated to perform Syntax score and suggested to perform NIRS in the culprit lesion and at least in one non-culprit lesion, in all patients presenting with NSTE-ACS. LCBI and Max LCBI 4 mm were calculated in all analyzed vessels by two independent investigators.

Results: SCAP trial enrolled 78 patients with NSTE-ACS. NIRS was performed in 65 patients on a total of 151 vessels. The number of diseased vessels and the Syntax score were comparable between the two groups (UCOPD and no COPD). The LCBI and max LCBI 4mm were significantly higher in the UCOPD group. The same result was found in culprit and in non-culprit coronary arteries (LCBI culprit: UCOPD 119 [102–146]; no COPD 71 [50–119], p=0.01; LCBI non culprit: UCOPD 111 [69–144]; no COPD 77 [46–104], p=0.002; LCBI max 4 mm culprit: UCOPD 424 [399–464]; no COPD 286 [178–385], p<0.001; LCBI max 4 mm non-culprit: UCOPD 337 [286–408]; no COPD 233 [151–333], p<0.001). The number of vessel with max LCBI 4 mm>400 was significantly higher in the UCOPD group

Study results

	All (Patients n=65, Vessels n=151)	UCOPD (Patients n=18, Vessels n=42)	No COPD (Patients n=47, Vessels n=109)	
NSTEMI, no.	43 (66)	13 (72)	30 (64)	0.4
Number of diseased vessels	2 [1-2]	2 [1-2]	2 [1-2]	8.0
Syntax score	13 [5-23.5]	13.5 [5.5-24]	12.5 [5-24.5]	0.7
LCBI	84 [56-120]	117 [70-144]	74 [50-107]	< 0.001
Max LCBI 4 mm	298 [198-401]	406 [302-430]	261 [176-350]	< 0.001

UCOPD: undiagnosed chronic obstructive pulmonary disease. NSTEMI: no ST-elevated myocardial infarction. LCBI: lipid core burden index.