procedures. The search time of CABG was significantly shorter in the fusion group of images in the control group (16.04 min ± 13.2 min vs 6.4 min ± 4.7 min, p = 0.002), as well as procedure time (31.5 min ± 15.2 min vs 19.4 min ± 6.8 min, p = 0.007), fluoroscopy time (14.76 min ± 8.3 min vs 9.3 min ± 4.2 min, p = 0.04), Air KERMA (822.9 mGy ± 475.5 mGy vs 569.1 mGy ± 242.4 mGy, p = 0.01) and amount of iodinated contrast (121 cc ± 43.5 cc vs 88.2 cc ± 30.8 cc, p = 0.01). 3D reconstruction of CABG from CT with real-time fusion on coronary angiography images may shorten the search time of CABG, procedure time, X-Ray exposure and the use of iodinated contrast compared to standard coronary angiography for diagnostic purposes in patients with CABG.

**Results:** Coronary angiography demonstrated that among pts with CAD and DM2 in 25% cases (n=5) registered atherosclerotic lesion of one CA and in 45% (n=9) lesion of two or three CA compared with the 40% (n=18) and 20% (n=9) respectively in pts with CAD without concomitant DM2 (p<0.05). Total of 312 LV myocardial segments were analyzed by STE. The number of abnormal segments that recovered function in the 1st gr was significantly lower than in the 2nd gr (21.7% vs 28%, p<0.05). Assessing LV functional recovery one year after revascularization pts of the 1st gr demonstrated lower end-systolic longitudinal (LongS,%), radial (RadS,%) and circumferential (CircS,%) strains (LongS = −9.36 ± 6.2 vs −12.73 ± 6.7, p = 0.033; RadS = 16.32 ± 10.8 vs 22.5 ± 11.7, p = 0.026; CircS = −8.2 ± 7.9 vs −12.3 ± 8.5, p = 0.043) than in the 2nd gr (LongS = −11.8 ± 7.2 vs −14.3 ± 7.3, p = 0.015; RadS = 19.8 ± 12.4 vs 23.5 ± 11.3, p = 0.025; CircS = −9.8 ± 8.7 vs −12.5 ± 8.1, p = 0.021).

**Conclusions:** pts with CAD and DM2 had lesser degree of LV functional recovery one year after revascularization compared to those without DM2 that can be explained by peculiarities of CA atherosclerotic lesion. Assessments of LV myocardial viability using STE is recommended for patients selection before revascularization and further follow up.

### 0025

**Validation of genetic risk score predicting cardiovascular death and/or myocardial infarction in a coronary Tunisian population: a 9.6 follow up study**

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**Background:** After myocardial infarction, patients have a very heterogeneous risk of cardiovascular death and/or reinfarction. Furthermore, no genetic approach score, as part of secondary prevention, was found in the literature.

**Aim:** This is a prospective longitudinal study (mean follow up of 9.6 years) including 146 patients, hospitalized between August 1997 and August 2004 for a myocardial infarction (MI) who survived the 30 first day after the MI. Depending on the composite event cardiovascular death and/or myocardial infarction, the genetic score was derived from 43 single nucleotide polymorphisms (SNP) belonging to 26 different genes. In the same study 3 clinical and biological scores were calculated (European SCORE, Hard coronary heart disease and coronary heart death score of Framingham scores). A multivariate analysis using logistic regression was used.

**Results:** Mortality at 10 years was 12.3% with a median time of 5.2±4.8 years. The incidence of the composite event cardiovascular death and/or myocardial infarction was 23.4% with a mean of 8.4±4.1 years. The genetic score predicting the composite event cardiovascular death and/or myocardial infarction is an equation that included age (OR=1.04, 95% CI: 0.98, 1.1) and the following polymorphisms eNOS894 (OR=1.87, 95%: 0.64, 5.43); rs2781665AT arginase (OR = 1.63, 95% CI: 0.69, 3.85); 45TG adiponectin (OR=1.62, 95% CI: 0.65, 4.02); eNOS4a4b (OR=1.29, 95% CI: 0.53, 3.14) and MTP (OR=0.78, 95% CI: 0.35, 1.74). The clinical and biological scores were all correlated with the long-term corresponding predicted. Correction factors were calculated for each score. The genetic score had the best reliability with a Sensitivity = 73.3%, a Specificity = 61.3%, a predictive positive value = 31.11%, a predictive negative value = 90.74%.

**Conclusion:** It seems that using a genetic score provides a good prediction of event cardiovascular death and/or myocardial infarction at long term. A validation of our genetic score and a combined clinical-genetic approach to a large population remains our future hope (table next page).