ischemia is frequent in ESRD patients and is a major predictor of mortality. Interestingly the incidence of significant coronary stenosis (>70%) is low when coronary angiography is performed suggesting other mechanisms such as myocardial microvascular disease (MMD).

We are conducting a prospective observational study (NCT01291771) evaluating the incidence of major adverse cardiac events (MACE) in ESRD patients with proven MMD and no history of coronary disease. 105 patients with positive test for myocardial ischemia will be explored by coronary angiography. Detection of MMD will be performed simultaneously with a pressure sensor/thermistor-tipped guidewire in the left anterior descending (LAD), the left circumflex (LCX) and the right coronary artery (RCA). We will measure Fractional Flow Reserve (FFR) and Coronary Flow Reserve (CFR) allowing calculation of the Index of Microcirculatory Resistance (IMR). These patients will be compared in a 2 years follow-up for the incidence of MACE with 105 ESRD patients without myocardial ischemia on non invasive testing.

We are reporting the preliminary data of patients (n=6) in whom invasive MMD evaluation was performed. Mean age 63±8 years, sex ratio=1, diabetec nephropathy=1, under dialysis=4. 1 patient had significant coronary stenosis, the others had normal angiograms (32%) or non-significant coronary atheroma (50%). In patients without significant coronary stenosis, mean FFR was 0.93±0.02 (LAD), 0.98±0.02 (LCX) and 0.95±0.02 (RCA). Mean CFR was 3.3±2.7 (LAD), 2.3±1.1 (LCX) and 3.1±1.4 (RCA). Mean IMR was 19±6 (LAD), 27±15 (LCX) and 30±26 (RCA).

Preliminary data suggest impaired myocardial microvascular function in ESRD patients with positive test for myocardial ischemia and non significant coronary artery stenosis.

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Copeptin to improve diagnosis of acute STEMI

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Objectives: The aim of this investigation was to test the diagnostic additive value of the C-Terminal Provasopressin (Copeptin) an indirect marker for arginin-vasopressin (AVP) in the detection of STEMI (presenting early (<6 hours after the Symptom Onset (SO).

Methods: We measured plasma copeptin in 464 consecutive STEMI patients (78% men, mean age 63 ± 14 years) immediately after the sheath insertion and before the primary PCI. Of those patients 200 (43.1%) had a blood sampling early after SO (<6 hours) and were included in this analysis. Using the previously established threshold of 4.8pmol/L for copeptin and the local 99th of the upper limit of the normal value for troponin I (0.15μg/L) we evaluated the additive value of the combination of copeptin and troponin I in this AMI patients.

Results: In the 200 Early STEMI presenters, the median time between SO and blood sample was 165 minutes; IQR [125-230], (min 35-max 360). The median value of Troponin I at sheath insertion was 0.59μg/L; IQR [0.13-3.2] and 33.1 pmol/L, IQR [11.6-111] for copeptin. Troponin I was negative at admission in n=56 (28%) STEMI patients and copeptin was negative at admission in n=54 (27%) STEMI patients. Patients with one or two negative biomarkers presented earlier than those with both positive biomarker (median time in minutes 152 [127-215] vs. 180 [125-240]). Among the n=56 patients with a negative troponin value at admission, n=38 (67.8%) had a positive copeptin value (>4.8pmol/L).

Therefore, when copeptin is combined to troponin I, the proportion of STEMI patients non-detected was reduced from 28% to a remaining 9.2% (p<0.001) of very early presenters (median time of 145 min IQR [116-180]).

Conclusions: The combination of copeptin and troponin improves the diagnostic value in AMI patients but cannot rule out the diagnosis of acute STEMI in very early presenters (<3 hours).

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The Gln/Arg of human paraoxonase polymorphisms (PON1 Leu55Met and Gln192Arg; PON2 Ser311Cys) is not related to acute myocardial infarction in the Tunisian population

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Introduction: Paraoxonases (PONs) are closely related antioxidant enzymes encoded by clustered genes on chromosome 7q. Two particular polymorphisms, namely PON1-192 and PON2-311, in the genes encoding the antioxidant enzymes paraoxonase-1 (PON1) and paraoxonase-2 (PON2) have been associated with an increased risk of acute myocardial infarction (AMI). However, previous findings have been contradictory. We evaluated three PON polymorphisms (PON1 Leu55Met and Gln192Arg; PON2 Ser311Cys) in Tunisian patients with AMI.

Methods: 168 AMI patients compared to 169 healthy volunteers.

Results: PONs allele and genotype frequencies did not differ between patients and controls.

The PON polymorphisms (PON1 Leu55Met and Gln192Arg; PON2 Ser311Cys) were not significantly associated with AMI (p=1.11, p=0.09, p=1.46 respectively). No significant differences in age, sex, BMI, waist circumference, total Cholesterol, HDL-C and LDL-C were detected among the three-genotype subgroups of PON1 Leu55Met, PON1 Gln192Arg and PON2 Ser311Cys in the AMI patients.

Conclusions: The PON1 Leu55Met, PON1 Gln192Arg and PON2 Ser311Cys polymorphisms are not related to acute myocardial infarction in Tunisian population.

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Acute coronary syndrome features with Fourier domain optical coherence tomography

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Background and aim: Fourier domain Optical Coherence Tomography (FD-OCT) is a new imaging modality characterized by a high axial resolution...
Common predictors of Blush score and TIMI flow following primary angioplasty

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Achieving TIMI flow 3 during primary angioplasty for acute myocardial infarction (PAMI) was associated with better left ventricular function and better prognosis. Blush score 2 or 3 was an indicator of reperfusion after angioplasty. The aim of our study was to identify predictive factors of TIMI flow 3 and Blush score 2 or 3.

Patients and Methods: Two hundred twenty-eight consecutive primary angioplasty were performed in our department. Culprit artery patency was classified according to TIMI (Thrombolysis in Myocardial Infarction). Culprit artery was considered permeable when TIMI flow was grade 3 and the Blush score was 2 or 3 following revascularization.

Results: A TIMI flow 3 was obtained in 86%. All these following factors were not associated with patency of culprit coronary: gender, cardio-vascular risk factors, location and the culprit artery of AMI, Q wave necrosis, multi-vessel disease and right ventricular extension.

In univariate analysis, left ventricle ejection fraction over than 45% (p=0.014 for TIMI; p=0.01 for Blush) and regression of ST segment elevation more than 50% (p=0.0001 for TIMI; p=0.0010 for Blush) were associated with better TIMI flow and Blush score. Anemia (p=0.01 for TIMI; p=0.05 for Blush), left ventricular failure (p=0.0001 for TIMI; p=0.007 for Blush), shock (p=0.004 for TIMI; p=0.001 for Blush) and residual thrombus (p=0.0001 for TIMI p=0.032 for Blush) were predictive for slow-flow and Blush score 0 or 1.

In multivariate analysis, regression of ST segment elevation more than 50% was the most important predictive factor of blush score 2 or 3 and especially of TIMI 3 (TIMI 3: p=0.0001, CI=95%, OR=25 [8-79]; Blush score 2 or 3: p=0.0001, CI=95%, OR=2.8 [1.6-5.1]).

Conclusion: Regression of ST segment elevation was the most important predictor of TIMI 3 and of Blush score 2 or 3, independently associated with the other factors.