

## P3622

**Cardiac X syndrome and myocardial infarction with nonobstructive coronary arteries: cardiovascular risk profiles and prognosis**

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**Background/Introduction:** Similarly to clinical syndromes related to obstructive coronary artery disease (CAD), myocardial ischemia with normal coronaries may have different clinical manifestations: stable effort angina, historically labeled as cardiac syndrome X (SX), and, in the context of acute coronary syndromes, myocardial infarction with nonobstructive coronary arteries (MINOCA).

**Purpose:** We compared two different cohorts of patients having SX or MINOCA, to analyze possible differences in terms of cardiovascular risk factors and long-term prognosis, also exploring gender differences.

**Methods:** We analysed 402 patients with SX (from the RISX "Italian registry of patients with Cardiac Syndrome X") and 150 patients with MINOCA (a cohort derived from 7935 consecutive patients admitted for acute myocardial infarction at our Institution). SX definition included: a) stable angina history, b) positive exercise test, c) epicardial coronary stenosis  $\leq 20\%$ . MINOCA was defined according to the ESC Position Paper 2016 as follows: a) acute myocardial infarction as defined by "Third Universal Definition of Myocardial Infarction", b) epicardial coronary stenosis  $\leq 50\%$ , c) coronary angiogram performed by 48 hours of symptom onset. We examined differences between the two cohorts in terms of: 1) cardiovascular risk factors; 2) long-term prognosis. The endpoint was the composite of all-cause death, acute coronary syndrome, stroke, re-hospitalization, and coronary revascularization.

**Results:** Results when compared to MINOCA group, patients with SX were more likely to have dyslipidemia (SX: 62.2%; MINOCA: 26.7%;  $p < 0.0001$ ) and family history of CAD (SX: 30.2%; MINOCA: 10.7%;  $p < 0.0001$ ); there were no significant differences with regard to the other examined cardiovascular risk factors (smoking, arterial hypertension, and diabetes) (Figure, panel A). In the MINOCA group, male patients were more likely to be current smokers (37.3% vs 20.5%;  $p = 0.022$ ) and female patients were more likely to have arterial hypertension (74.7% vs 55.2%;  $p = 0.012$ ). In the SX cohort, female patients had a higher prevalence of family history of CAD (34.6 vs 17.5%;  $p = 0.001$ ). At six-year follow-up, a total of 22 combined events occurred in SX patients compared to 23 events among patients with MINOCA. Annual rate of combined events was higher among patients with MINOCA (3.17%) compared to patients with SX (1%) (Figure, panel B). Event-free survival was statistically significant higher (Log Rank=5.235,  $p = 0.022$ ) in patients with SX (92.9%  $\pm 1.8\%$ ) as compared with MINOCA patients (85%  $\pm 2.9\%$ ) (Figure, panel C).

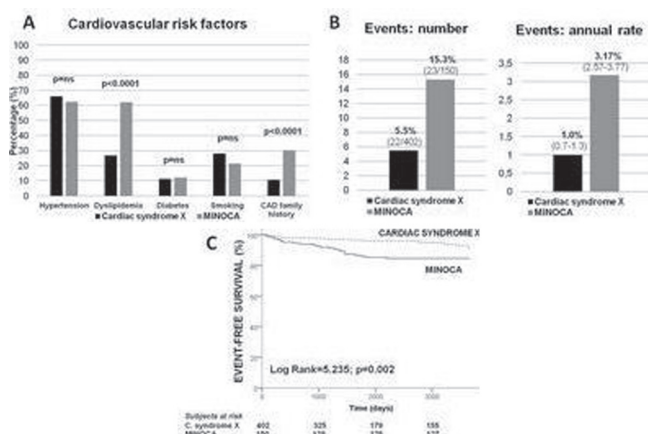


Figure 1

**Conclusions:** Patients with SX appeared to have a worse cardiovascular risk profile when compared to MINOCA patients, who in turn seems to display a higher incidence of adverse events at long-term follow-up.

## P3623

**Impact of blood urea nitrogen/creatinine ratio in coronary artery disease patients underwent successful percutaneous coronary intervention with drug eluting stents: 5-year follow-up results**

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**Background:** It is well established that cardiovascular disease patients with chronic kidney disease (CKD) are associated with higher adverse cardiovascular outcomes. The Blood Urea Nitrogen/Creatinine (BUN/Cr) ratio is used as an

indicator for determining the cause of kidney function. However, there are currently limited data regarding the impact of BUN/Cr ratio in coronary artery disease (CAD) patients undergoing successful percutaneous coronary intervention (PCI).

**Methods:** The outcomes of 1660 consecutive CAD patients underwent PCI with DES were enrolled for the analysis. The study population was classified into two groups; the low BUN/Cr ratio group (BUN/Cr ratio  $< 20$ ,  $n = 928$ ) and the high BUN/Cr ratio group (BUN/Cr ratio  $\geq 20$ ,  $n = 732$ ). The individual and composite major clinical outcomes were compared between the two groups up to 5 years.

**Results:** The baseline clinical characteristics were similar between the two groups except that the low BUN/Cr ratio group had more male gender, younger age and higher prevalence of previous myocardial infarction (MI), history of CVD, peripheral vessel disease, CKD, current smoker, and current alcoholic. After baseline adjustment by cox proportional hazards regression, individual and composite clinical outcomes up to 5 years showed a higher incidence of MI, ST-segment elevation MI, revascularizations, target vessel revascularizations (TVR), non-TVR, total major adverse cardiovascular events (MACE; composite of total death, any MI, and total revascularizations), TVR MACE (composite of Total Death, Any MI, and total TVR) in the low BUN/Cr ratio group (Table).

Table. Cumulative Incidence of Clinical Outcomes Up to 5 Years

Variables	low BUN/Cr ratio <20 (n=928)	high BUN/Cr ratio ≥20 (n=732)	Log Rank	HR [95% CI]	P value
Total death	78 (8.4)	62 (8.5)	0.599	1.17 [0.83-1.66]	0.369
Cardiac death	45 (4.9)	37 (5.1)	0.806	1.21 [0.77-1.89]	0.412
Non Cardiac death	33 (3.6)	25 (3.4)	0.924	1.05 [0.60-1.82]	0.867
Myocardial Infarction, MI	39 (4.2)	18 (2.5)	0.063	2.06 [1.16-3.67]	0.014
ST-segment elevation MI	28 (3)	12 (1.6)	0.074	2.17 [1.08-4.35]	0.030
Non-ST-segment elevation MI	11 (1.2)	6 (0.8)	0.512	1.9 [0.68-5.33]	0.220
Revascularizations	104 (11.2)	48 (6.6)	0.002	1.72 [1.2-2.46]	0.003
Target Lesion Revascularizations	60 (6.5)	32 (4.4)	0.069	1.44 [0.92-2.25]	0.113
Target Vessel Revascularizations	74 (8)	36 (4.9)	0.015	1.64 [1.09-2.50]	0.019
Non-TVR	39 (4.2)	14 (1.9)	0.010	2.17 [1.15-4.11]	0.017
Stent Thrombosis	10 (1.1)	5 (0.7)	0.400	1.92 [0.64-5.8]	0.248
Total MACE	174 (18.8)	104 (14.2)	0.024	1.44 [1.12-1.85]	0.005
composite of Total Death, Any MI, and total Revascularizations					
TLR MACE	99 (10.7)	64 (8.7)	0.210	1.34 [0.97-1.86]	0.081
TVR MACE	145 (15.7)	94 (12.8)	0.132	1.35 [1.03-1.76]	0.031
composite of Total Death, Any MI, and total TVR					

Adjusted by gender, age, myocardial infarction, hypertension, diabetes, dyslipidemia, heart failure, chronic kidney disease, peripheral artery disease, cerebrovascular accident, current smoker and multi-vessel disease

**Conclusion:** In our study, the low BUN/Cr ratio group increased individual and composite clinical outcomes compared with the high BUN/Cr ratio group. We suggest that more care should be exercised for CAD patients with low BUN/Cr ratio undergoing PCI

## P3624

**Glycoprotein IIb/IIIa inhibitor therapy in ST-segment elevation myocardial infarction: a systematic review and meta-analysis of randomized controlled trials**

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**Background and purpose:** Platelet inhibition is critical in the pharmacologic management of acute ST-segment elevation myocardial infarction (STEMI). Besides modern oral antiplatelet therapy, i.v./i.c. glycoprotein IIb/IIIa inhibitors (GPI) convey strong antiplatelet effects, however evidence for routine use in STEMI is lacking. We aimed to analyze GPI safety and efficacy in a systematic review and meta-analysis of randomized controlled trials (RCT) of GPI therapy in STEMI patients.

**Methods:** Online databases were searched for RCTs comparing periprocedural i.v./i.c. GPI administration to placebo/no therapy up until September 2017. Data from retrieved studies were abstracted and analyzed in a comprehensive meta-analysis. Primary outcome was all-cause mortality, secondary outcomes were recurrent myocardial infarction (MI), repeat revascularization, TIMI flow grade, major/minor bleeding and stroke.

**Results:** Fifteen eligible RCTs including 7,837 patients were included in the meta-analysis: tirofiban (T) was used in seven RCTs, abciximab (A) in six, eptifibatide (E) as well as A+T in one trial each; median follow-up was 30d. Administration of GPI showed a trend towards reduction of all-cause mortality (2.3% (GPI) vs. 2.9%; odds ratio (OR) 0.79, 95% confidence interval (CI) 0.6–1.05;  $p = 0.1$ ), without difference between A/E/T. Recurrent MI (OR 0.61, CI 0.42–0.87;  $p = 0.007$ ), repeat revascularization (OR 0.62, CI 0.48–0.8;  $p = 0.0003$ ) and TIMI flow  $< 3$  at end of procedure (OR 0.59, CI 0.43–0.8;  $p = 0.03$ ) were all significantly in favor of GPI vs. placebo/no therapy, stroke was borderline significant (OR 0.43, CI 0.18–1.01;  $p = 0.05$ ). Major bleeding (OR 1.4, CI 1.1–1.76;  $p = 0.004$ ) and minor bleeding (OR 1.51, CI 1.1–2.07;  $p = 0.01$ ) complications were significantly more frequent in patients with GPI.

**Conclusions:** Routine use of GPI therapy in STEMI patients results in numerical but not statistically significant reduction in mortality. GPI benefits for ischemic complications of STEMI come at the expense of an increase in bleeding events.