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Genetic polymorphisms associated with in-stent restenosis in patients with acute coronary syndrome

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Purpose: To investigate whether FGB, F2, F5, F7, ITGB3, SERPINE1 or MTHFR genetic polymorphisms are associated with in-stent restenosis (ISR) following coronary stenting in patients with ST-elevation acute coronary syndrome (ACS).
Methods: Studied were 233 patients with ST-evelevation ACS, who signed the informed consent protocol. All the patients were divided into two groups: 1 group comprised 105 patients (93 males (88.6%), mean age 56.7±10.2) with ISR after percutaneous coronary intervention (PCI) (confirmed by coronary angiography), 2 group - 128 patients (103 males (80.5%), mean age 56.3±10.8) with an uncomplicated postoperative period. In all the patients we analyzed genetic polymorphisms of FGB (rs1800790), F2 (rs1799963), F5 (rs6025), F7 (rs6046), ITGB3 (rs5918), SERPINE1 (PAI-1) (rs2227631) and MTHFR (rs1801133) before PCI, and also coronary angiography data, ECG, laboratory findings.
Results: We found that genetic polymorphisms of FGB (allele A), F2 (allele A), F5 (allele A), F7 (allele A), ITGB3 (allele C), SERPINE1 (allele 4G) and MTHFR (allele T) alone were not significantly associated with ISR in patients after PCI ($p>0.05$ for all polymorphisms). However for genetic polymorphisms associations regression analysis revealed that combinations of FGB (allele A)+ITGB3 (allele C) (OR 2.1 (95% CI 1.05–5.1, p=0.03)), SERPINE1 (allele 4G)+ITGB3 (allele C)+FGB (allele A) (OR 2.8 (95% CI 1.1–7.4, p=0.02)), SERPINE1 (allele 4G)+ FGB (allele A) +ITGB3 (allele C)+F7 (allele A) (OR 3.5 (95% CI 1.4–6.9, p=0.009)) and FGB (allele A)+ITGB3 (allele C)+F7 (allele A) (OR 4.2 (95% CI 1.2–7.8, p=0.01)) polymorphisms showed independent association with in-stent restenosis in patients after PCI.
Conclusion: Our findings suggested that combinations of minor alleles of FGB+ITGB3, SERPINE1+ITGB3+FGB, SERPINE1+ FGB+ITGB3+F7 and FGB+ITGB3+F7 are significantly associated with in-stent restenosis in patients after percutaneous cardiac intervention.

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Real world clinical predictors of myocardial infarction with non-obstructive coronary arteries according to 2016 ESC definition

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Background: According to 2016 ESC Position Paper, Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is a working diagnosis settle at the point when a patient admitted for a myocardial infarction (MI) has no obstructive coronary arteries. The aim of this work is to analyze the differences between MINOCA an obstructive related MI and propose predictors of MINOCA.
Methods: Analytical and observational study developed in a University Hospital, which covers 220.000 individuals. We analyzed demographic and clinical data of all consecutive MINOCA patients admitted during a 3 year period (2015–2017) and compared them with 269 consecutive patients with acute MI and obstructive coronary arteries. We used definitions and management of 2016 ESC Position Paper on MINOCA. Those variables with a statistical signification lower than $p<0.01$ in univariable analysis were included in a logistic regression analysis to determinate independent predictors of MINOCA.
Results: During a 3 year period, 118 patients fulfilled the 2016 ESC criteria of MINOCA. Table 1 shows the results of univariable and multivariable analysis.

Table 1. Results of univariable and multivariable analysis

	Univariable			Multivariable		
	MINOCA Total: 118	Obstructive MI Total: 269	p	Odds Ratio	IC 95%	p
Age (years)	61.77±15.8	67.1±12.9	<0.01	1.02	0.99–1.05	0.135
Sex	50.8%	22.4%	<0.01	2.37	1.01–5.52	0.046
Allergies	20.3%	7.4%	<0.01	3.26	1.09–9.75	0.035
Emotional stress	72.1%	34.5%	<0.01	2.41	1.06–5.42	0.034
Conective tissue disease	7.6%	1.5%	<0.01	4.79	0.53–42.91	0.162
Psychiatric disease	29.1%	12.6%	<0.01	1.98	0.72–5.44	0.184
Tobacco	45.2%	65.1%	<0.01	0.45	0.19–1.05	0.067
Diabetes	18.6%	36.6%	<0.01	0.27	0.09–0.72	0.009
Dislypidemia	41.9%	56.9%	<0.01	0.49	0.21–1.10	0.085
Initial cardiac rate (bpm)	89.34±29.1	78.3±18.4	<0.01	0.97	0.95–0.99	0.005
Troponine I (ng/ml)	6.29 ±12.7	31.09 ±54.2	<0.01	1.06	1.00–1.062	0.008
Creatinquinase (U/L)	478.4±1188	896.9 ±1350	<0.01	1.00	0.99–1.00	0.861
ECG: ST depressed	5.1%	15.7%	<0.01	0.15	0.03–0.6	0.011

Conclusion: MINOCA patients had a different clinical profile than obstructive MI. The independent predictors of MINOCA at the admission of an MI were: female sex, recognition of emotional stress, antecedents of allergies, higher cardiac frequency, absence of diabetes, absence of ST depression and lower peak levels of troponine.

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Prevalence of familial hypercholesterolaemia and familial combined hyperlipidaemia in very young survivors of myocardial infarction and association with the severity of atheromatus burden

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Background and aims: Heterozygous familial hypercholesterolaemia (heFH) and familial combined hyperlipidaemia (FCH) are associated with early onset coronary artery disease (CAD). We assessed the prevalence of HeFH and FCH among young survivors of myocardial infarction (MI) and compared patients' characteristics with these 2 lipid disorders.
Methods: We prospectively recruited 382 young survivors of MI (≤40 years). Fasting lipids, lipoprotein(a) [Lp(a)], apolipoprotein A and apolipoprotein B levels were determined. Using Dutch Lipid Clinic Network [DLCN] algorithm patients having HeFH (possible, probable or definite) were identified. Patients with apolipoprotein B levels >120 mg/dL and triglyceride levels >200 mg/dL were classified as having probable FCH. Common carotid artery intima-media thickness (CCA-IMT) was measured by B-mode ultrasonography.
Results: Eighty-one patients (21.2%) had definite/probable HeFH and 56 (14.7%) had probable FCH. Seventeen patients fulfilled the criteria for both HeFH and FCH (21% of FH were also classified as FCH while 30.3% of FCH patients were also classified as having HeFH) and removed from further comparisons. Patients with HeFH had higher levels of cholesterol, LDL-cholesterol, Lp(a) and apolipoprotein B while patients with FCH had higher levels of triglycerides and lower levels of HDL-cholesterol (Table). The prevalence of metabolic syndrome was higher in patients with FCH compared with those with HeFH (67 vs 16.4%, $p<0.001$). Patients with heFH had more extensive CAD (3 vessel disease: 32.8 vs 13.2%, p=0.028) and greater right CCA-IMT (0.71±0.16 mm vs 0.56±0.08 mm, $p<0.001$) and left CCA-IMT (0.72±0.15 mm vs 0.57±0.08 mm, $p<0.001$) compared with patients with FCH.

Lipids	Familial hypercholesterolaemia (n=64)	Familial combined hyperlipidaemia (n=39)	p value
Cholesterol (mg/dL)	340.8 7±74	255.6±36	<0.001
Triglycerides (mg/dL)	137.8±42	285.9±82	<0.001
HDL-cholesterol (mg/dL)	40.2±11	35.2±12	0.032
LDL-cholesterol (mg/dL)	268.7±72	162.7±25	<0.001
Apolipoprotein A (mg/dL)	119.9.1±29	118.7±26	0.859
Apolipoprotein B (mg/dL)	159.1±43	141.7±18	0.040
Lipoprotein(a) (mg/dL)	39.3±40	18.3±21	0.005

Conclusions: Both HeFH and FCH are common among patients with premature MI but HeFH is associated with more atheromatous burden in coronary and carotid arteries.

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Predictors of new-onset atrial fibrillation after acute coronary syndrome

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Introduction: Atrial fibrillation (AF), the most commonly encountered clinical arrhythmia, can be an acute coronary syndrome (ACS) complication.
Aims: To determinate incidence and predictors of new-onset AF during hospitalization of patients (pts) admitted due to ACS.
Methods: A multicentre, observational, retrospective study was performed during 10/2010–3/1/2018 period. Data on demographic, comorbidities, complementary exams performed, clinical presentation and outcomes were retrieved from the Portuguese Registry on ACS. Pts with new-onset AF during hospital stay were compared with those without new-onset AF (including those with previous AF or with AF in the electrocardiogram at admission). Variables with a p value <0.05 in univariate analysis were included in a logistic regression model and predictors of new-onset AF were identified.
Results: Of the 16.524 pts included, 732 (4.4%) had new-onset AF during hospitalization (73.6% were male).
Independent risk factors of new-onset AF occurrence were: age ≥75 years (OR 2,73, CI 2.25–3.33, $p<0.001$), previous heart failure (HF) (OR 1.43, CI 1.02–2.02,p=0.038), chronic obstructive pulmonary disease (COPD) (OR 2.53, CI 1.1–2.12, p=0.012), beta-blocker (OR 1.29, CI 1.02–1.64, p=0.036) or acetylsalicylic acid (OR 1.37, CI 1.09–1.72,p=0.007) therapy, ST-elevation myocardial infarction (STEMI) (OR 1.96, CI 1.59–2.40,$p<0.001$), diastolic blood pressure ≤110 mmHg (OR 1.77, CI 1.02–3.06,p=0.042) or creatinine value at admission ≥1 mg/dL (OR 1.35, CI 1.11–1.65, p=0.003), left ventricular ejection fraction ≤50% (OR 1.7, CI 1.04–2.79,p=0.035) and non-invasive mechanical ventilation support (OR 2.34, CI 1.6–3.44, $p<0.001$), temporary pacemaker implantation (OR 2.46, CI 1.66–3.62, $p<0.001$), HF development (OR 2.21, CI 1.77–2.77, $p<0.001$) or sustained ventricular tachycardia (VT) (OR 2, CI1.26–3.21, p=003) occurrence during hospitalization.
Family history of coronary disease (OR 0.42, CI 0.23–0.78, p=0.006), previous coronary angioplasty (0.62, CI 0.45–0.86, p=0.004) or permanent pacemaker/implantable cardioverter defibrillator implantation (0.2, CI 0.06–0.65,