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Atrial fibrillation is associated with a marker of endothelial function and oxidative stress in patients with acute myocardial infarction

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Background Atrial fibrillation (AF), whether silent or symptomatic, is a frequent and severe complication of acute myocardial infarction (AMI). Asymmetric dimethylarginine (ADMA), an endogenous eNOS inhibitor, is a risk factor for endothelial dysfunction. We addressed the relationship between ADMA plasma levels and AF occurrence in AMI.

Methods 273 patients hospitalized for AMI were included. Continuous electrocardiographic monitoring (CEM) ≥ 48 hours was recorded and ADMA was measured by High Performance Liquid Chromatography on admission blood sample.

Results The incidence of silent and symptomatic AF was 39(14%) and 29 (11%), respectively. AF patients were markedly older than patients without AF (≈ 20 y). There was a trend towards higher ADMA levels in patients with symptomatic AF than in patients with silent AF or no AF (0.53 vs 0.49 and 0.49 $\mu\text{mol/L}$, respectively). After matching on age, we found that patients with symptomatic AF had a higher heart rate on admission and a higher rate of patients with LV dysfunction (28% vs. 3%, $p=0.025$). Patients who developed symptomatic AF had a higher ADMA level (0.53 vs. 0.43 $\mu\text{mol/L}$; $p=0.001$). Multivariate logistic regression analysis to estimate symptomatic AF occurrence showed that ADMA was independently associated with symptomatic AF (OR: 2.46 [1.21-5.00], $p=0.013$) beyond history of AF, LVEF $<40\%$ and elevated HR.

Conclusion We show that high ADMA level is associated with the occurrence of AF. Although no causative role can be concluded from our observational study, our work further supports the hypothesis that endothelial dysfunction is involved in the pathogenesis of AF in AMI.

The author hereby declares no conflict of interest

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Impact of thienopyridines on platelet CD40L biodisponibility after an acute coronary syndrome in relation with bleeding events

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Background CD40 Ligand (CD40L) is expressed on platelets upon ADP stimulation and is involved in haemostasis. CD40L deficient mice exhibit thrombus instability and increased bleeding time.

Methods We investigated the relationships between plasma and platelet-associated CD40L, ADP signaling and bleeding event occurrence in patients receiving thienopyridines one month after a stented Acute Coronary Syndrome (ACS). Basal platelet CD40L surface expression (pCD40L), pCD40L after PAR-1 agonist stimulation (TRAP pCD40L) and platelet released CD40L (rCD40L) were quantified. Results were compared to VASP as a measure of P2Y12 inhibition level.

Results We included 318 patients between November 2012 and June 2014. Thienopyridines treated patients exhibit low pCD40L, TRAP pCD40L and rCD40L in comparison with controls. pCD40L and rCD40L were correlated with PRI-VASP. Thienopyridine treatment strongly reduces rCD40L. Hypersponder to thienopyridine status is associated with high levels of TRAP pCD40L. pCD40L and TRAP pCD40L levels are reduced in the bleeding cohort. In multivariate analysis pCD40L significantly contributes to bleeding risk independently of PRI-VASP.

Conclusion pCD40L and rCD40L levels are reduced by thienopyridines. pCD40L associates with the bleeding risk independently of the VASP levels

and may represent a novel target to assess bleeding risk in thienopyridine-treated ACS patients.

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The Log Book as a new tool for the secondary prevention of coronary artery disease

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Introduction The Log book (LB) project was created by a multidisciplinary team of healthcare professionals from a regional care network and aimed to improve secondary prevention (SP) after acute myocardial infarction (MI) in Cote d'Or. LB includes information and advices for increasing self management of risk factors by the patient.

Methods A prospective interventional study on 469 patients hospitalised for an acute MI in the 2 Intensive Coronary Care unit of Côte d'Or (CHU Dijon and Clinique de Fontaine les Dijon) in 2012 and surviving at 1 year follow-up (FU). LB was randomly given at the time of their hospitalisation by the nursing team, also providing oral advices on risk factors management and CV health self care. Patients who received LB (LB+) were compared with patients without LB (LB-).

Results Patients from LB+ group ($n=307(65\%)$) were younger (57 vs 63y, $p<0.001$) and less frequently women (16 vs 32%, $p<0.001$), diabetic (17 vs 30%, $p<0.001$) or with prior CAD (7 vs 15%, $p=0.008$) than patients without LB (LB- group, $n=162(35\%)$). After matching patients LB+ with LB- based on age, sex, diabetes and GRACE risk score ($n=127$ in each group), baseline characteristics, were similar in the 2 groups. At 1 year FU, there was a trend for more frequent visits to the cardiologist in the LB+ group (2 ± 1 vs 1 ± 1 , $p=0.056$) and cardiac rehabilitation program was more often performed in LB+ patients (69 vs 54%, $p=0.015$). Moreover, weight loss in obese patients and smoke withdrawal rates also showed a trend for improvement in LB+ patients (respectively 71 vs 59%, $p=0.311$ and 69 vs 53%, $p=0.109$). Finally, patients with LB showed a trend toward a lower rate of combined outcomes including recurrent MI, hospitalisation for heart failure and unscheduled PCI than in the LB- group (3 vs 7%, $p=0.155$).

Conclusion These preliminary data of our ongoing regional study suggest the efficacy of LB as a support for CV risk factor self management. In addition, our study provides encouraging data on the potential clinical benefits of this pioneer tool for SP.

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Cardiovascular protection of statins could be mediated by an increase of total bile acids concentration in sera? A pilot study

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Introduction In animal models of atheroma (ApoE-/- and LDL -/- mice), bile acids (BAs) exerts an anti-atherosclerotic effect through the anti-inflammatory action of their receptors, TGR5 and FXR, decreasing dramatically the surface of the atheroma plaque. BAs are cholesterol derivatives synthesized by the liver. In a previous study, we found that a decrease in BAs (lithocholic acid) is an independent risk factor of coronary disease in human.