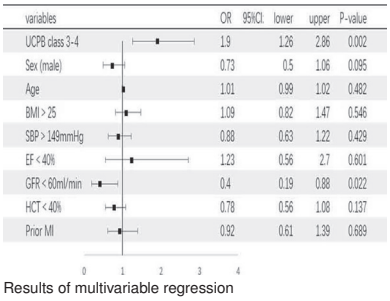
analyzed. Major bleeding were defined as the universal definition of perioperative bleeding (UDPB) class 3 to 4 including following criterias: 1) strenal closure delayed; 2) postoperative chest tube blood loss within 12 hours ≥1001 mL; 3) requiring transfusion of ≥5 U packed red cells (PRBCs) or fresh frozen plasma; 4) surgical reexploration. Baseline characteristics were compared between patients with and without major bleeding. Univariate and multivariate logistic regression analyses were performed to investigate the impact of perioperative major bleeding on postoperative myocardial infarction.
Results: A total of 3830 patients who underwent OPCAB were included in this study. Major bleeding rate was 9.45% (n=362). And postoperative myocardial infarction occurred in 202 (5.27%) patients. Of 362 and 3468 patients with and without major bleeding, 30 (8.3%) and 172 (5.0%) patients suffered from postoperative myocardial infarction (p=0.007). Univariate analyses demontrated that patients with major bleeding were at a higher risk of postoperative myocardial infarction (OR=1.73, CI: 1.16–2.59, p=0.007). Multivariable regression analysis showed that perioperative major bleeding increased the risk of postoperative myocardial infarction during OPCABG (OR=1.90, CI: 1.26–2.86, p=0.002).

Comparison of perioperative bleeding characteristics between MI and without-MI groups

Variables	Without MI group (n=3628)	MI group (n=202)	P value
Chest tube output ≥1001ml	7.6%	11.9%	0.028
Transfusion of PRBCs≥5U	2.5%	5.9%	0.003
HB decrease≥50g/l	33.1%	41.1%	0.019
Reoperation for bleeding	2.1%	5.9%	0.001



Results of multivariable regression

Conclusion: Perioperative major bleeding is an independent risk factor of postoperative myocardial infarction in patients undergoing OPCAB.

P2662
Prognostic value of postoperative high-sensitivity troponin among patients undergoing fenestrated and/or branched endovascular aortic aneurysm repair

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Introduction: Patients undergoing fenestrated/branched endovascular aortic repairs (F/B-EVAR) are particularly at risk of myocardial injury after non-cardiac surgery (MINS). The recently introduced high-sensitivity troponin (HsTnT) may allow better diagnosis of MINS as compared to former troponin assays. However HsTnT prognostic value has not been evaluated in F/B-EVAR patients.
Purpose: Our objectives were to assess the prognostic value of postoperative HsTnT in patients undergoing F/B-EVAR and to identify the optimal threshold of HsTnT that defines MINS in this population.
Methods: Data from 222 adult patients who underwent F/B-EVAR were extracted from a data warehouse that collects data from our intraoperative, biology and administrative management systems. HsTnT values of the first three postoperative days were gathered and the highest value is identified as Peak-HsTnT. After univariate analysis, a multivariate logistic regression model was built to explain the main endpoint of the study, in-hospital mortality.
Results: The primary endpoint occurred in 5.2% of patients. Peak-HsTnT and Day-1 HsTnT were independently associated with in-hospital mortality, OR 1.02 (95% CI 1.00–1.023), p=0.024], OR 1.05 (95% CI 1.00–1.11), p=0.039] respectively. Among HsTnT variables, Peak-HsTnT showed the best predictive performances for the primary endpoint after ROC curve analysis. Peak-HsTnT thresholds of 35 ng/L had the highest Youden index and positive likelihood ratio, the lowest negative likelihood ratio, for the prediction of in-hospital mortality.
Conclusion: After F/B-EVAR, peak-HsTnT was independently associated with in-hospital mortality.
Funding Acknowledgements: Institutional funding

P2663
Results from a multicenter study of transradial iliac artery stenting in Japan

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Background: We previously reported the safety and effectiveness of transradial iliac artery stenting from a small-sized single center cohort. To date, large-scaled multicenter studies that address this issue are lacking.
Purpose: We evaluated the safety and efficacy of transradial iliac artery stenting from a multicenter database in Japan.
Methods: Transradial iliac artery stenting was performed in 115 lesions from 105 patients. Approach site was determined at the discretion of the operator. Cases with scheduled multiple sheath insertion for bidirectional approach were excluded. Clinical data were analyzed retrospectively.
Results: From this cohort, the average age was 71.1±8.3 years. Eighty-six (81.9%) patients were male. Diabetes mellitus, hypertension, dyslipidemia, and smoking habit were present in 39 (37.1%), 84 (80.0%), 69 (65.7%), and 78 (74.3%) patients, respectively. Rutherford classification 1, 2, 3, 4, and 5 constituted 40 (34.8%), 42 (36.5%), 28 (24.3%), 3 (2.6%), and 2 (1.7%); while, TransAtlantic Inter-Society Consensus II classification A, B, C, and D were 74 (64.3%), 21 (18.3%), 15 (13.0%), and 5 (4.3%) of the lesions, respectively. Twenty seven lesions (23.5%) were chronic total occlusions. All lesions were successfully treated with a total of 141 stents. Four cases (3.8%) needed additional puncture of the common femoral arteries for successful stent implantation. Fifty two (45.2%), 34 (29.6%), and 29 (25.2%) lesions were treated using 4.5, 5, and 6 French long guiding sheaths, respectively. Ankle brachial index significantly improved from 0.65±0.17 to 0.95±0.15 ($p<0.0001$). None of the patients had any procedural or access site-related complications such as hematoma, major bleeding, blood transfusion, stroke, cholesterol embolism, aortic dissection, or arterial perforation. Radial artery occlusion without symptom was observed in 3 cases (2.9%) after the procedure. There were no target lesion revascularization or complications at 1-month.
Conclusions: Transradial iliac artery stenting is safe and feasible without any specific complications for carefully selected patients compared to traditional transfemoral approach.

P2664
Long-term follow up of first-in-human study in bypass of stenosis av shunt by an autologous in-body-tissue-engineered (biotube) vascular graft

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Background and purpose: An arteriovenous (AV) fistula is the current gold standard for chronic hemodialysis access. However, a substantial number of shunt will fail because of stenosis or obstruction at anastomotic site or venous outflow. Tissue-engineered blood vessels have been proposed for dialysis access as an alternative to prosthetic grafts. We developed autologous collagenous tubular tissues “Biotubes” formed by in-body tissue architecture (IBTA), proposed by us is a regenerative medicine technology that can prepare autologous implantable tissues by using a patient’s body as a bioreactor. IBTA represents a novel and practical approach to regenerative medicine. This report presents the first-in-human results after 2 years of follow-up for the first two patients bypassed with autologous Biotube.
Methods and results: Two female patients had end-stage renal disease and had been receiving heamodialysis with a high probability of failure, because of repeatable stenosis about every 2 or 3 months at venous outflow regions. Biotube vascular grafts with 5 or 6 mm in diameter and 7 cm in length were prepared as autologous collagenous tubular tissues with wall thickness of ca. 1mm by embedding of molds, assembled with a silicone center rod and a stainless steel tube, into patients abdominal subcutaneous pouches for 2 months. The Biotubes after stored for 1 day in a 70% alcohol solution were bypassed by end-to-side anastomoses over venous stenosis region of an AV shunt. Palpable thrill and typical turbulent flow pattern by pulsed-wave Doppler were observed. Monthly angiography showed little change in the implanted grafts with no signs of dilation or stenosis with time points up to 3 months. Although shortening of the Biotubes occurred, dialysis was possible without requiring balloon expansion for 2 years.
Conclusion: This long-term follow up study successfully supported the concept of creating dialysis access from autologous collagenous Biotube grown in patients subcutaneous pouches.

P2665
Misdiagnosing acute aortic syndrome as acute coronary syndrome: a single center experience

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Background: Acute aortic syndrome (AAS) is a diagnostic challenge, and it is frequently confused with acute coronary syndrome (ACS) since its symptoms and electrocardiographic changes may mimic those of myocardial ischemia.
Purpose: The aim of this study is to define clinical and electrocardiographic findings resulting in an inappropriate diagnosis of ACS in patients with AAS.

Methods: Clinical and electrocardiographic data from all patients of the prospective registry of AAS diagnosed in our center from 2012 to 2018 were reviewed. Diagnosis of AAS was confirmed by computed tomography (CT), surgery or autopsy. An inappropriate diagnosis of ACS was considered when double antiplatelet therapy (DAPT) was administered or a coronary angiography was performed before the diagnosis of AAS was established.
Results: 135 patients were admitted for AAS, 89 (66%) with AAS Stanford type A and 97 (72%) with aortic dissections. 40 patients (29,6%) were misdiagnosed with ACS. There were no significant differences in terms of sex or presence of cardiovascular risk factors, nor in the type of AAS. Concerning symptoms, misdiagnosed patients presented more frequently with anterior chest pain (85%, p<0,001) and very few with abdominal pain (7,5%, p=0,008) or with neurological deficits (5%, p=0,049). Regarding ECG, an ischemia-like ECG was found more prevalent in misdiagnosed patients (56,4% p<0,05), being ST-T abnormality the most frequent finding (46,2%).
Conclusion: Almost a third of the patients found to be suffering from AAS were initially misdiagnosed with ACS, especially those presenting with anterior chest pain and ST-T abnormalities in the ECG, so physicians should maintain a high index of suspicion as early treatment is crucial.

INFLAMMATION AND IMMUNITY IN ACUTE CORONARY SYNDROMES

P2666
Endothelial microvesicles-incorporated long non-coding RNA PUNISHER regulates inflammatory responses in THP1 recipient cells

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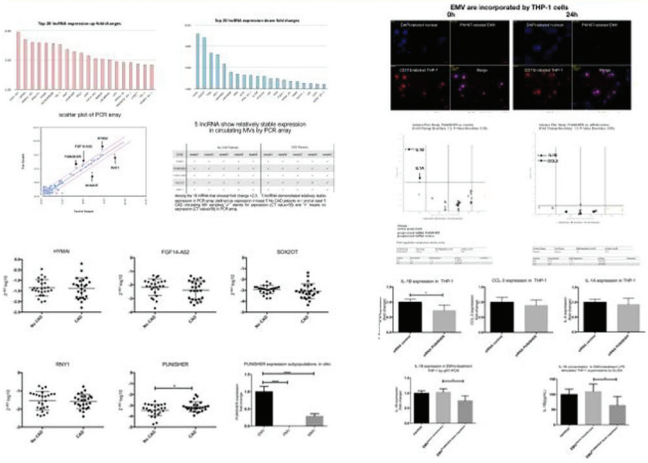
Background: Circulating long non-coding RNAs (lncRNAs) are biomarkers and effectors of cardiovascular diseases. Whether microvesicle (MV)-lncRNAs expression is regulated in coronary artery disease (CAD) is unknown. We aimed to explore the expression of circulating MV- lncRNAs in patients with CAD.
Methods and results: Plasma from 10 patients with angiographically excluded CAD (n=5) and stable CAD (n=5) was collected for MV-lncRNA array analysis. Circulating MVs were isolated from patients' plasma using ultracentrifugation. Flow cytometer and electron microscope were used for MV size characterization. Using a PCR array-based Human Functional lncRNA PCR Array (Arraystar), we identified 18 lncRNAs showing a fold change>2.0 between patients with or without CAD (13 lncRNAs upregulated, 5 lncRNAs downregulated in circulating MVs). Among those, 5 lncRNAs (HYMAI, PUNISHER, FGF14-AS2, S0X2OT, RNY1) revealed relatively stable expression (defined as CT value <38 in at least 9 patients from 10). We then prospectively studied another 50 patients with (n=25) and without (n=25) CAD to confirm lncRNA array results. Single lncRNA rtPCR indicated that only AGAP2-AS1 (PUNISHER) was significantly increased in patients with CAD compared to patients without CAD. In-vitro MV-sorting experiments showed that endothelial cell (EC) was the main source of MV containing PUNISHER.
Next, we demonstrated endothelial microvesicles (EMVs) could be absorbed by human monocytic cells (THP-1 cells) using fluorescence microscopy. In order to explore whether PUNISHER affects inflammation, gene expression array was performed in THP1 cells after PUNISHER knockdown. Results showed 3 inflammatory cytokines were significantly regulated after PUNISHER silencing, including Interleukin-1B (IL-1B), Interleukin-1A (IL-1A), C-C Motif Chemokine Ligand 3 (CCL3). Single rtPCR experiments confirmed that IL-1B expression level significantly decreased after PUNISHER knock-down THP-1 cells. In order to explore whether EMV might transfer functional PUNISHER to THP1 cells, we generated EMVPUNISHER-knockdown and EMVmock-transfected. After treatment of target THP1 cells with different EMVs, IL-1B RNA expression and protein concentration were quantified in THP-1 with qRT-PCR and in supernatants with ELISA. Knockdown of PUNISHER within EMVs was associated with a significantly reduced IL-1B secretion in THP-1 target cells, suggesting that functional lncRNA PUNISHER is transferred from ECs into THP1 cells regulating inflammatory responses in recipient cells.
Conclusion: EMV-incorporated lncRNA PUNISHER is significantly upregulated in patients with CAD. EMV-mediated transfer of functional lncRNA-PUNISHER regulates inflammatory responses in THP-1 recipient cells.

Summary: Endothelial microvesicles-incorporated long non-coding RNA PUNISHER regulates inflammatory responses in THP1 recipient cells



P2667
Osteocalcin expression confers divergent roles of endothelial progenitor cells in cumulative inflammation-induced coronary calcification Development

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Background: Osteogenic circulating endothelial progenitor cells (EPC) that carry the osteoblastic marker osteocalcin (OCN) may implicate in vascular calcification. However, its role in relation to EPC phenotypic characterization in coronary calcification promulgated by inflammatory stress remained unknown.
Purpose: To investigate the effect of cumulative inflammation load on incident coronary calcification and mechanistic relations with EPC OCN phenotypic expression.
Methods: We conducted a retrospective clinical-pathophysiological cohort analysis of patients with rheumatoid arthritis (n=145). Data on serum C-reactive protein (CRP) was retrieved from each quarterly measurements in the past 60 months immediately preceding the date of CT coronary angiography and EPC measurements. Raised CRP was defined as >0.35mg/dL. A dichotomous score of 1 was assigned to each biomarker encounter with any raised CRP >0.35mg/dL, whereas each CRP ≤0.35mg/dL was conferred a score of 0. A time-adjusted CRP aggregate score as estimate of cumulative inflammation load over the preceding 60 months was composed conglomerating all biomarkers encounters. Flow cytometry was performed to measure the OCN-positive (OCN+) CD34+KDR+ and OCN+CD34+ circulating EPC. Conventional early circulating EPC CD34+CD133+KDR+ was determined. Presence of CT-detected coronary calcification was defined as any Agatston score >0.
Results: 50% patients (n=72/145) had CT-detected coronary calcification. CRP burden score was associated with presence of coronary calcification (P=0.004), which remained independent after multivariable adjustment for age, gender, history of hypertension, diabetes mellitus, hyperlipidemia, fasting levels of LDL-/HDL-cholesterol, triglycerides and glucose, creatinine, systolic/diastolic blood pressure, smoking status, use of statins and disease-modifying antirheumatic agents, and duration of rheumatoid arthritis (highest versus lowest quartile OR=4.8, 95% CI 1.2–19.2, P=0.026, Figure 1A). Furthermore, CRP score was significantly associated with increased OCN+CD34+KDR+ (Pearson R=0.27, P=0.002) and OCN+CD34+ EPC (R=0.22, P=0.010), but not CD34+CD133+KDR+ EPC (P=0.2). Strikingly, ROC curve analyses revealed divergent effects of osteogenic EPC (Figure 1B, OCN+CD34+ EPC: AUC=0.60, P=0.034; OCN+CD34+KDR+ EPC: AUC=0.59, P=0.053) versus conventional early CD34+CD133+KDR+ EPC (AUC=0.40, P=0.034) on coronary calcification.

Figure 1A

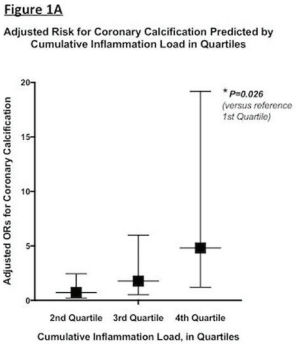
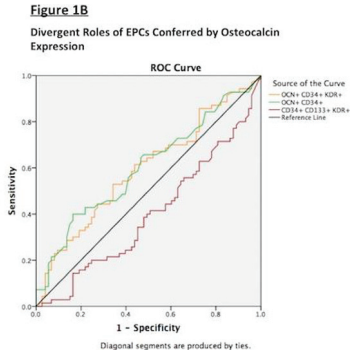


Figure 1B



Conclusions: OCN expression confers a detrimental pathophysiological role to circulating EPC in cumulative inflammation-induced coronary calcification, exemplified in rheumatoid arthritis.
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