SA: 2.2±0.1 vs 1.7±0.1; p=0.009; mean±SEM: ACS vs HC: 2.2±0.1 vs 1.6±0.1; p=0.0004; ANOVA for trend: p=0.002) (Figure 1, Panels A and B). Moreover, we proved an increased platelet-monocyte binding after 16h of co-incubation in ACS patients compared to SA patients (ACS vs SA: 24.0±8.1 vs 3.7±2.7; p=0.03 (Figure 1, Panels C).

Conclusions: Plaque instability involves platelet-immune cell interactions, but the details of this cross-talk are not fully elucidated. CD31 has different behaviors strongly depending on cell types and surrounding environment; indeed in ACS it is downregulated on circulating monocytes driving pro-inflammatory responses, while our study highlights an enhanced expression on platelets. This parallels an increased platelet-monocyte binding. Even if further studies are needed to confirm the role of CD31, our data suggest that this multifaceted molecule might be an attractive target to unravel the complex network of platelet-monocyte interaction.

P1693
Elevated counts of naive and memory B cells might be associated with the severity of myocardial damage in the setting of acute myocardial infarction

R. Feldtmann, A. Kuemmel, E. Abdiu, B. Chamling, S. Gross, M. Doerr, S.B. Felix, R. Busch, A. Strohbach. *University Medicine of Greifswald, Department of internal medicine B, Greifswald, Germany*

Objective: Acute myocardial infarction is typically caused by an abrupt blockage of a coronary artery, leading to necrosis of the affected heart tissue. Immediately, distinct leukocyte cell populations infiltrate the injured heart mediating self-repair and regeneration of the injured tissue as well as pathological autoimmune responses. The latter of which may subsequently lead to ventricular remodeling and heart failure. Hence, regulating this immune response may prove beneficial as an adjunct therapy in the management of AMI.

Purpose: B lymphocytes act as important sensors of pathogens and self-antigens and promote both protective and autoimmune effects. We explored the role of subtypes of maturating B lymphocytes in the setting of ischemic injury.

Methods: We quantified the number of mature B cells in arterial blood of 14 patients with ST-segment elevation MI (STEMI) and 17 patients with non-ST-segment elevation MI (NSTEMI). Additionally, 10 healthy individuals were included. Absolute cell counts were assessed in whole blood, drawn from the arterial port prior to coronary intervention, by multi-color FACS analysis. The total number of cells was normalized to the white blood cell count of the whole blood analysis. Linear regression analysis was performed to associate total B cell counts with troponin I and creatine kinase (CK) plasma levels in MI patients. The multivariable regression model comprised sex, age, current smoking, symptom duration, leukocyte count, and CRP-levels. Risk assessment for AMI patients was done using the GRACE score.

Results: The number of CD19+ B cells was significantly increased in STEMI patients (31.9±6.2 x104 cells/mL) compared to healthy controls (13.6±3.3 x104 cells/mL; p=0.026) and the NSTEMI group (15.9±2.1 cells/mL; p=0.016). Results were associated with plasma troponin I and CK and revealed a strong relation of the CD19+ B cell count with the levels of the markers of myocardial infarction (figure 1). Further, investigation of B cell subpopulations revealed a higher number of cells in STEMI patients compared to healthy controls and to NSTEMI group (figure 1). Noteworthy, only naïve B cells and memory cells show a correlation to troponin I (Pearson r=0.770; p=0.001 and r=0.717; p=0.006, respectively) and CK (Pearson r=0.827; p=0.001 and r=0.717; p=0.006, respectively). Finally, the number of memory cells correlates to the 6 months probability of death from admission, which was calculated from the GRACE Score (Pearson r=0.609; p=0.047).

Association of CD19⁺ cell counts with troponin I/CK in AMI patients			
	β-coefficient †	standard error	*p-value (adjusted)*
troponin I	1894.9	294.8	< 0.0001
CK	3607.5	925.9	0.0025

AMI – acute myocardial infarction; CK – creatine kinase; Linear regression analysis was performed to relate CD19⁺ B cell counts with plasma levels of troponin I and CK. The multivariable regression model comprised sex, age, current smoking, symptom duration, leukocyte count, and CRP-levels. † The ß-coefficient indicates the increase in CD19⁺ cell count for a 1 unit increase in plasma troponin I/CK levels.

(B) counts of B cell subtypes	control (n=10)	NSTEMI (n=17)	STEMI (n=14)	*p-value (1-way Anova)*
B lymphocytes (x10⁴)				
CD19⁺ B cells	13.6 ± 6.2	15.9 ± 2.1#	31.9 ± 6.2*	0.0085
naive B cells	6.6 ± 3.3	8.1 ± 5.0 #	15.3 ± 12.1*	0.0223
transitional B cells	1.2 ± 0.6	1.0 ± 0.6 ###	2.9 ± 1.9 ***	0.0006
plasma cells	0.07 ± 0.06	0.1 ± 0.1	0.2 ± 0.2 ***	0.0101
memory B cells	0.9 ± 0.2	1.2 ± 0.9 #	2.8 ± 2.4 *	0.0086

STEMI – ST-segment-elevation myocardial infarction; NSTEMI – non-ST-segment-elevation myocardial infarction; (B) To evaluate the differences of B cells counts a one-way ANOVA was performed followed by a Tukey post hoc test. Results are reported as mean ± SD. # p<0.05 and ## p<0.01 and ### p<0.001 indicate statistically significant differences between STEMI und NSTEMI group. * p<0.05 and *** p<0.001 indicate statistically significant differences to the control group.

Figure 1

Conclusion: Our data indicate a potential relation of elevated B cell counts with adverse outcomes in patients with acute MI. Furthermore, the strong association of naïve and memory B cells with markers of myocardial damage suggests a pathophysiologically relevant role for these subsets. How B cells contribute to the injured heart itself is an area of further investigations.

Funding Acknowledgements: Supported by the DZHK (German Centre for Cardiovascular Research) and by the BMBF (German Ministry of Education and Research)

P1694
Novel risk factors in spontaneous coronary artery dissection: thyroid disorders

S.J. Camacho Freire[1], M. Garcia Guimaraes[2], L. Gheorghe[1], T. Bastante[2], A.E. Gomez Menchero[1], A.E. Vera[2], J. Roa Garrido[1], J. Cuesta[2], J. Leon Jimenez[1], F. Rivero[2], R. Cardenal Piris[1], M. Pedregal Gonzalez[1], J.F. Diaz Fernandez[1], F. Alfonso[2]. *[1]Hospital Juan Ramon Jimenez, Interventional Cardiology, Huelva, Spain; [2]University Hospital De La Princesa, Cardiology, Madrid, Spain*

Spontaneous coronary dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS). Thyroid hormone affects the metabolism of all tissues in the body. The aim of this study was to analyze the prevalence and implications of thyroid disorders in a cohort of consecutive patients with SCAD.

Methods: This was a multicenter case (SCAD) and control (ACS without SCAD) study performed in 2 Spanish university tertiary hospitals. We evaluated 73 patients with SCAD and 73 controls who suffered an ACS matched by age, gender and clinical presentation. The prevalence of thyroid alterations in both groups was compared. In addition, among SCAD patients, findings of those with hypothyroidism were compared with patients with normal thyroid function.

Results: Mean age was 55±12 years and 77% of patients were women. A statistically significant difference was observed in the prevalence of hypothyroidism between the two groups (26% vs 8%, p=0.004), being most of these patients on thyroid hormone replacement therapy with good metabolic control.

Among SCAD patients, those with hypothyroidism were more often female and had more distal dissections (74% vs 41%, p=0.03), located in curly vessels, and more often were managed conservatively (79% vs. 41%, p=0.007). During a mean follow-up of 4.1±3.8 years, the rate of adverse clinical events was 23% (n=17), with no differences according to the thyroid function status. Some patients with SCAD and abnormal thyroid function suffered iatrogenic dissections and this cohort tended to be associated with a higher recurrence rate (16% vs 7%, p=0.36) and lower rate of spontaneous resolution during follow-up.

Conclusions: There is a high prevalence of hypothyroidism in unselected consecutive patients with SCAD compared with a control group of ACS patients without SCAD. The group with hypothyroidism, with more distal dissections on curly vessels, is more frequently managed conservatively.

P1695
Impaired endothelial function is associated with neointimal abnormalities after drug-eluting stents deployment assessed by optical coherence tomography in patients with ischemic heart disease

Y. Uchicado[1], S. Yoshino[1], T. Takumi[1], D. Kanda[1], K. Ohmure[2], H. Tabata[2], K. Anzaki[2], M. Ohishi[1]. *[1]Kagoshima University, Department of Caridovscular Medicine and Hypertension, Kagoshima, Japan; [2]Izumi Regional Medical Center, Department of Cardiology, Akune, Japan*

Introduction: Neointimal abnormalities such as uncovered strut, late-acquired incomplete stent apposition (ISA), and evagination assessed by optical coherence tomography (OCT) might clarify the pathophysiology of stent thrombosis. Relationship between endothelial dysfunction and neointimal abnormalities in patients with ischemic heart disease (IHD) is not verified.

Purpose: The aim of this study was to assess the relationship between initial endothelial function and neointimal characteristics after 2nd generation drug-eluting stent (DES) deployment in IHD patients.

Methods: A total of 56 patients implanted 2nd generation DES were enrolled (70±11 yrs; 44 men). Sixty two lesions (12636 struts) were analyzed by OCT at 7-months follow-up and endothelial function was assessed by reactive hyperemia index (RHI) using Endo-PAT 2000. Patients were divided into 2 groups; endothelial dysfunction group (RHI<1.67) and normal endothelial function group (RHI≥1.67).

Results: The frequency of uncovered strut, evagination, and/or late-acquired ISA was higher and neo-intimal thickness was thinner in endothelial dysfunction group than in normal endothelial function group (24.1% vs 15.4%, p<0.001; 61.2±58.2 μm vs 103.9±98.5 μm, p<0.001, respectively). Furthermore, neointimal thickness was associated with neointimal abnormalities and RHI, with 80.9% of neointimal abnormalities and 1.47±0.45 of RHI in lowest neointimal thickness quartile, respectively. Multivariate logistic analysis revealed the presence of RHI<1.67, acute coronary syndrome, and hyperuricemia were independent predictors for the presence of neointimal abnormalities.

Conclusion: Impaired endothelial function is associated with neointimal abnormalities after 2nd generation DES deployment assessed by OCT in IHD patients.