At 9M, early atherosclerosis with focally distributed eccentric intimal thickening was predominantly (90%) present, in a minority (10%) healthy vessel wall was observed. The majority progressed to eccentric lipid-rich lesions (78%) at 12M, the remaining segments (22%) preserved a fibrous appearance. At 15M, third of the lesions progressed and showed calcification (33%). Lipid-rich lesions were still predominantly present (56%), in a minority fibrotic plaque was preserved (11%). Fig.1 is a typical example of atherosclerosis development, with eccentric intimal thickening at 9M progressing in plaque thickness and circumferential extent at 12 and 15M.

Our atherosclerotic swine model offers the possibility to mimic the clinical situation with atherosclerosis progression. Combination with serial OCT imaging can provide new possibilities for preclinical studies allowing studying dynamic vessel response over time with a reduced no. of animals.

P2405 | BENCH
Comparison amount of human herpes viruses DNA in vessels from different group of patients

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Aim: It is generally thought, that myocardial infarction (MI) is the one of the main causes of death in developed countries. Vulnerable atherosclerotic plaque more likely can rupture and lead to myocardial infarction. The cause of these events’ maturation and rupture may be associated with inflammation and local and/or systemic immune activation. But it’s still unknown, if there any infectious agent in plaques that may lead to such an activation. We have investigated if the presence of human herpes viruses DNA in vessels of different group of patients is associated with plaque instability.

Methods: We included 30 patients hospitalized with MI and who died from MI or it’s complications. Coronary arteries samples were taken during autopsy. Macroscopic observations of the tissues allowed their classification in 3 groups: 1) clean samples, without macroscopic signs on atherosclerosis, 2) non-ruptured plaques; 3) ruptured plaques, usually MI-causative vulnerable plaques with thrombus or ulceration. Also we analyzed samples obtained from 10 patients who dyed from coronary-artery–disease unrelated causes. In these samples there was no significant evidence of atherosclerosis.

Results: HHV-1, HHV-2, HHV-3, HHV-4, HHV-5, HHV-6, HHV-7, HHV-8 and HHV-9 were found in more than 93% of samples. HHV-1&2, HHV-3, HHV-5, and HHV-8 were found in more than 80% of the samples. HHV-4, HHV-6 and HHV-7 were the less frequent contained at least one HHVs. HHV-1&2, HHV-3, HHV-5, and HHV-8 were found in 49% to 55% of the samples. Analysis of the distribution of HHVs between the three group samples did not reveal any significant difference. However, in samples from patients with non MI-related cause of death, DNA of HHV-1&2, HHV-3, HHV-5, HHV-6 and HHV-9 was found in 80% HHV and HHV7 in 80% and HHV4 in 70% of samples.

Conclusion: We found that all HHVs are present in artery samples of people, who either died from MI or other causes. The higher viral DNA load in arterial tissues did not correlate with the presence of plaque or the stability thereof. Interestingly, HHV-8, the virus associated with Kaposi’s sarcoma, a disease usually developed by immunosuppressed patients, was abundant in this cohort of non-immunosuppressed patients.

P2406 | BENCH
Advancement intracranial atherosclerosis is present in asymptomatic patients, but is almost devoid of microvessels

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Purpose: There is increasing evidence on the importance of intracranial atherosclerosis in stroke and vascular dementia, yet not much is known on the phenotype of intracranial atherosclerosis. Since neovascularization within atherosclerotic plaques is a source of intraplaque hemorrhage, we focused on the presence of microvessels and intraplaque hemorrhage.

Methods: 283 segments of intracranial vessels were obtained from 18 Circles of Willis (CoWs) autopsies of asymptomatic human subjects. Segments included the communicating, middle cerebral and the superior cerebellar artery and proximal anterior cerebral and anterior cerebral artery. Atherosclerosis was classified on H&E and EvG stained slides according Virmani et al. 2000 and subsequently grouped in early and advanced lesions. Microvessels and plaque hemorrhage were detected by immunohistochemistry staining (CD31 and GlycoporphinA). All samples were analyzed by counting the number and size quantitatively.

Results: 58 of the 283 segments (20%) had no lesions, 150 (53%) early and 77 (27%) advanced lesions. Proximal segments had more advanced atherosclerosis than distal segments (38 (71.7%) vs. 15 (28.3%).) Only one proximal segment contained a ruptured lesion, with massive intraplaque hemorrhage and intimal microvessels. Microvessels were found in 24 other segments (9%), but only in the adventitia and predominantly in advanced lesions in 18 of the 53 (34%) versus 6 of the 135 early lesions (4%). The adventitia was complete in 69 segments (24%), incomplete in 169 segments (60%) and 45 out of 283 (16%) lacked any adventitia, which may be attributed to adventitial stripping during sampling.

Conclusions: More than 25% of asymptomatic vessel segments of the Circle of Willis had advanced atherosclerosis, in particular the proximal segments. We only found one ruptured plaque with intraplaque hemorrhage and intimal microvessels. Microvessels were present, but only in 9% of the segments, and mainly in the adventitia of segments with advanced lesions. However the adventitia was remarkable absent or incomplete in the vast majority of segments, which may have led to an understimation of the number of microvessels.

P2408 | BEDSIDE
Carotid plaque inflammation as a marker for the presence of severe coronary artery disease in patients evaluated for chest pain

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Purpose: Intima-media thickness (IMT) measurement in carotid arteries has been widely used as a surrogate marker for coronary atherosclerosis. However, IMT does not provide any functional characteristics, especially regarding the inflammatory status. Microvascular rarefication (MR) allows non-invasive in vivo measurement of internal thickness of tissues, reflecting inflammation. The purpose of this study was to evaluate the predictive value of thermal heterogeneity measured by MR for the diagnosis of significant coronary artery disease (CAD).

Methods: Consecutive patients (n=246) hospitalized for chest pain and scheduled for coronary angiography were evaluated by 1 ultra-sound echo-color Doppler (US-ECO) study of both carotid arteries, and 2 temperature measurements were performed using 2-millimeter thermal heterogeneity (ΔT), measured by MR, was assigned as the maximum temperature along the carotid artery minus the minimum temperature.

Results: Significant CAD was found in 200 patients (81%) documented by coronary angiography. Mean IMT was significantly higher in 180 (90%) of all carotid arteries 0.80±0.49°C. Carotid arteries with carotid artery disease (n=313), fatty plaques (n=52) had higher ΔT compared to mixed (n=181) and calcified (n=80) (1.23±0.55°C versus 0.87±0.44°C versus 0.67±0.49°C respectively, p<0.001). Plaques with regular surface (n=81) had higher ΔT compared to plaques with regular (n=232) (1.20±0.45°C versus 0.76±0.48°C, p<0.01). Heterogeneous plaques (n=71) had higher ΔT compared to homogeneous (n=246) (1.25±0.56°C vs. 0.50±0.36°C, p<0.001). Microvessels were similar in left and right carotid arteries (0.78±0.48°C versus 0.84±0.52°C, p=0.12). In all carotids, there was a correlation between left and right carotid plaque ΔT (r=0.001, R=0.28). In all lesions, there was a correlation between ΔT and IMT (r=-0.001, R=0.25).

Conclusions: Patients with coronary artery disease have similar inflammatory activation in the carotid plaques as detected by microvascular radium. Thus, the systemic response producing this diffuse inflammatory activation in patients with atherosclerosis need to be further investigated.

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