Citation for published version (APA):
Objective: Visceral artery fibromuscular dysplasia (VA FMD) is being rarely reported, with clinical picture ranging from asymptomatic to life-threatening. The aim of the study is to evaluate the prevalence and clinical characteristics of VA FMD.

Design and method: Out of 330 patients enrolled into ongoing ARCADIA-POL study since January 2015 (instituted as Polish-French collaboration) we present 225 patients (185 females, mean age: 46.1 ± 14.7yrs) with confirmed FMD in at least one vascular bed. All patients underwent detailed clinical evaluation including ABPM, biochemical evaluation, bio-banking, duplex Doppler of carotid and abdominal arteries and whole body angio-CT. In the current analysis, we focused on the prevalence and clinical characteristics of VA FMD (defined as the presence of typical FMD lesions in superior or inferior mesenteric arteries, as well as celiac trunk, splenic and hepatic arteries).

Results: Visceral FMD was present in 32 patients (19.1%). Among them, 75% (n = 24) were females, the mean age was 48.6 ± 17.2 years and 96.9% of patients have multifvessel FMD. 28 patients reported FMD lesions in mesenteric arteries or celiac trunk, 6 patients in splenic and 8 patients in other visceral arteries. In 28 (12.4%) patients, aneurysms were found in visceral arteries, most commonly in splenic arteries (23 pts[10.2%]). In 2 patients, aneurysms were present in more than one visceral artery. In one patient, a dissection of the celiac trunk was identified. Patients with VA FMD most commonly reported abdominal pain (10 patients, 31.2%). 5(15.6%) patients suffered from weight loss and 6(18.6%) from postprandial abdominal pain. 3 patients with VA FMD experienced emergencies. Two patients underwent intestine resection due to acute mesenteric ischemia caused by occlusion of the upper mesenteric artery and of the celiac trunk respectively. In a third patient, the primary manifestation of VA FMD was rupture of hepatic aneurysm complicated by hypovolemic shock. The aneurysm was eventually embolized, with favorable clinical outcome.

Conclusions: In ARCADIA-POL study, VAFMD was present in every fifth patient. The clinical presentations of VA FMD varied from asymptomatic lesions to emergencies, including critical ischemia and life-threatening aneurysm bleeding. Our results support screening for VA FMD in patients with FMD in other vascular beds.

IN-VITRO CHARACTERIZATION OF MOUSE CAROTID ARTERY MECHANICS BY DYNAMIC BAXIAL PRESSURE-MYOGRAPHY

M. van der Bruggen1, K. Reesink1, P. Sprock1, N. Bitsch2, M. Schalkwijk2, T. Delhaas1, B. Spronck1, N. Bitsch1, J. Hameleers1, R. Megens2, C. Schalkwijk2, T. Delhaas1, B. Spronck1, 1CARIM/MUMC+, Maastricht, The Netherlands, 2Ludwig-Maximilians-Universität, Munich, Germany, 3Yale University, New Haven, CT, USA

Objective: Although in-vitro biomechanical behaviour of arteries is commonly studied under quasi-static conditions, dynamic (pulsatile, in-vivo) behaviour may differ substantially. We developed a set-up to characterize mouse carotid artery biaxial mechanics under quasi-static and dynamic conditions, using high-frequency ultrasound to track diameter and two-photon laser scanning microscopy (TPLSM) to capture the microstructure. We aimed to (1) quantify reproducibility and (2) compare quasi-static and dynamic elastic behaviour.

Design and method: After euthanasia, eight carotid arteries from four male surplus mice were mounted between glass micropipettes. Four carotids were tested directly after euthanasia, four on the day thereafter. Pressure (P) was recorded at the distal pipette; axial force (F) was recorded by a load cell. First, arteries were stretched to in-vivo length and exposed to quasi-static pressure inflation from 0–200 mmHg. Second, axial stretch (lz) was varied for constant pressures of 60/100/140 mmHg to determine an axial stiffness coefficient. Third, vessels were exposed to pulsatile pressures (systolic pressures of 80/120/160 mmHg, at 5 Hz) and at frequencies (f) of 2.5/5/10 Hz at 120 mmHg. Single-point pulse wave velocity (PWV; Bramwell-Hill) was determined and compared with corresponding PWVs calculated from the quasi-static pressure-diameter curve. The axial stiffness coefficient was obtained as the local slope of the F-lz curve. Fourth, information on adventitial collagen-structure deformation was obtained using second-harmonic generation TPLSM. The first three protocol steps were performed in duplicate to determine coefficients of variation (CVs).

Results: CVs for PWV were ~9% for low-medium and 27% for high pressure. For f2.5 and f10 Hz these were 16% and 26%. Dynamic PWVs were higher than quasi-static PWVs for all test conditions (p < 0.012; mean ± SD, at f15 Hz: PWVP80 2.4 ± 0.2 vs. 2.1 ± 0.1 m/s, PWVP120 6.3 ± 0.6 vs. 5.0 ± 0.5 m/s, PWVP160 13.3 ± 1.9 vs. 10.3 ± 2.2 m/s, and at P120 mmHg: PWVf2.5 5.9 ± 0.7 vs. 3.5 ± 0.3 m/s, PWVf10 3.1 ± 0.9 vs 2.5 ± 0.2 m/s). Axial stiffness coefficients increased with pressure 60–140 mmHg (1.3 ± 0.4, 2.4 ± 1.0, 4.9 ± 2.8 grams). Measurements from fresh and non-fresh vessels were not significantly different (p > 0.099). From 60–100 mmHg undulation of adventitial collagen strands disappeared, whereas from 100–140 mmHg orientation changed (Figure).

Conclusions: Our innovative set-up shows well-acceptable reproducibility and demonstrates the importance of quasi-static and dynamic conditions when studying arterial mechanics.
brachial blood pressure (BP). Whether it can be changed independently of BP in the short term is uncertain but recent observations suggest that AS may be specifically modulated by the autonomic nervous system (ANS). Here we compared effects of device guided breathing (DGB, known to reduce sympathetic activity) with nifedipine on cfPWV.

Design and method: Patients with essential HT on pharmacological treatment (mean ± SD age 48 ± 14 years, n = 19) had cfPWV (SphygmoCor) and brachial BP (Omron) measurements performed before and after DGB and oral administration of nifedipine 10 mg. The two interventions were performed consecutively in single visit with the patient lying in supine position. DGB is a biofeedback technique which slows the breathing rate to <10 breaths/minute and decreases BP via its action on the ANS. Nifedipine is a short acting peripheral arterial vasodilator that tends to increase sympathetic activity.

Results: Baseline systolic BP (SBP) 150.4 ± 12.6 and diastolic BP (DBP) 89.8 ± 8.7 mmHg, heart rate (HR) 65.9 ± 11.2 bpm; cfPWV 10.3 ± 2.4 m/s. Compared to nifedipine, DGB caused less reduction of both brachial SBP and DBP: decrease of 12.3 (95% CI 8.4, 16.1) vs. 16.1 (12.7, 19.5) mmHg in SBP for DGB and nifedipine and 5.9 (3.1, 8.7) vs. 10.4 (7.1, 13.5) mmHg in DBP (P < 0.05 for difference between DGB and nifedipine). DGB caused a greater reduction in cfPWV: decrease of 1.2 (0.8, 1.8) and 0.7 (0.1, 1.3) m/s for DGB and nifedipine respectively (P = 0.02 for difference between DGB and nifedipine). HR decreased during DGB by 3.6 (1.2, 5.9) bpm and increased after administration of nifedipine: 7.6 (3.9, 11.3) bpm.

Conclusions: DGB had a greater impact on AS despite a smaller effect on BP. These results demonstrate that cfPWV can be changed independently from BP in the short-term and support a specific role of the ANS in regulating cfPWV.