(2.2–3.0) in the control group compared with 2.3 (2.0–2.8) in the symptomatic patients (p<0.001).

When adjusting for hypertension, age, body mass index, and diabetes in multivariate regression analysis the difference between CFVR in controls and symptomatic women was no longer significant (p=0.19). Traditional cardiovascular risk factors only explained little of the variation in CFVR between groups (r2=0.08). Results presented in Table 1.

Population characteristics and results

	Healthy women (n=102)	Symptomatic women (n=919)	p-value (unadjusted)	p-value (adjusted*)
CFVR, median (IQR)	2.6 (2.2–3.0)	2.3 (2.0–2.8)	<0.001	0.189
Age, mean (SD)	61.0 (10.4)	62.3 (9.7)	0.24	<0.001
Hypertension, n (%)	17 (19.1)	492 (53.8)	<0.001	0.011
DM, n (%)	3 (2.9)	117 (12.8)	0.0038	0.015
BMI, mean (SD)	24.9 (3.6)	27.2 (5.4)	<0.001	0.754

*Regression analysis adjusted for hypertension, age, diabetes mellitus (DM) and body mass index (BMI).

Conclusion: We found that the difference in CFVR, a marker of CMD, between women with angina and asymptomatic women was explained by age and cardiovascular risk factors.

P1569
Spontaneous coronary artery dissection in young female acute coronary syndrome patients; a single-centre retrospective data analysis

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Background: Spontaneous coronary artery dissection (SCAD) is an unusual cause of acute coronary syndrome (ACS), rapidly gaining recognition over the last decade. It is defined as a separation of coronary wall layers following intramural haemorrhage. SCAD is predominantly diagnosed in young, otherwise healthy women. Clinical presentation varies from an incidental finding on coronary angiogram to ACS or even sudden cardiac death. Due to the often challenging diagnosis, a striking underestimation of SCAD incidence was highlighted in recent registries.
Purpose: The aim of this large retrospective cohort study in a tertiary cardiac care centre was to re-evaluate SCAD data in young female ACS patients and to compare them to insights from recent literature.
Methods: All coronary angiograms performed for ACS in women ≤50 years old between 01/01/2013 and 30/11/2017 were retrospectively reviewed. Three experienced interventional cardiologists rigorously and independently analysed all images. Lesion assessment was solely qualitative (expert opinion). In order to withhold only true SCADs, patients with atherosclerosis or multiple cardiovascular risk factors were excluded and all investigators needed to fully agree on every SCAD-labelled lesion.
Results: A total of 102 coronary angiograms in ACS were reviewed. The diagnosis of SCAD was established in 27 patients, accounting for 26% of all ACS. SCAD was diagnosed in all ACS subgroups: 2/25 unstable angina (8%), 11/33 non-STEMI (33%) and 16/44 STEMI (32%). The total amount of documented SCADs was 29, as two patients presented with two affected arteries (7%). The main culprit vessel was the left anterior descending coronary artery (63%). Angiographic presentation was predominantly type 2B (55%), manifested as an intramural hematoma extending towards the apical coronary tip. Types I (7%), 2A (17%) and 3 (21%) were less common. Underdiagnosis was notable, as only 33% was correctly diagnosed during index procedure. Due to growing awareness however, a positive trend over time was clearly observed in terms of accurate diagnosis (2013–2016: 2/15 patients = 13% vs 2016–2017: 7/12 = 58%). Eighteen patients (67%) were managed conservatively with excellent outcome. Primary PCI was performed in 9 patients (33%) because of TIMI 0 flow, ongoing ischemia or erroneously presumed atherosclerotic plaque rupture. Five performed PCIs were uncomplicated (56% success rate), while 4 PCIs led to progressive dissection and extensive additional stenting. No fatalities were documented.
Conclusion: This retrospective cohort study highlights the underestimation of SCAD prevalence in young female ACS patients (26%). The correct diagnosis is challenged by often subtle angiographic features in an unusual low-risk population. Since optimal therapy differs greatly from atherosclerotic ACS, a high level of suspicion remains crucial. Furthermore, the importance of increasing awareness among interventional cardiologists needs to be emphasised.

P1570
Chemerin as a marker of cardiometabolic risk and subclinical cardiac remodeling in postmenopausal women

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Adipose tisssue is no longer considered only as the energy storage, but is emerging as an active endocrine organ secreting a number of hormone-like compounds termed adipokines, regulating many physiological processes. Chemerin is recently identified adipose hormone, which plays a role in adipogenesis, energy metabolism and inflammation. During the transition from premenopause to postmenopause, many women experience weight gain and central fat deposition, hence we hypothesized that circulating level of chemerin can play a role in the pathogenesis of hypertension and cardiometabolic risk in postmenopausal females.
Methods: The study included 80 women (mean age 57.6±5.4 years), 54 subjects with arterial hypertension and 26 normotensive age matched controls. In all subjects anthropometrical measurements and 24-hr ABPM were performed. Assessment of cardiac and vascular subclinical organ damages was performed, including echocardiographic examination with assessment of left ventricular mass (LVM), (GE Vivid 7.0); carotid ultrasound with measurement of intima-media thickness (IMT), and carotid-femoral pulse wave velocity measurement (PWV) (Sphygmocor).
Fasting blood samples were taken for glucose, insulin and serum lipids concentration. Serum levels of chemerin, nesfatin and obestatin were measured using an immunochemical assay.
Results: Chemerin was significantly associated with metabolic characteristics including BMI (r=0,32, p=0,003), waist circumference (r=0,48, p=0,0001), fasting glucose (r=0,27, p=0,02), and triglycerides (r=0,20, p=0.07). Compared with controls, subjects with hypertension had higher serum chemerin levels (147,8±29,7 vs 132,2±23,6; p=0,05). In logistic regression analysis, one SD increase in chemerin concentration was related with increased risk of hypertension (OR 1,83; 95% CI 1,13–3,46; p=0,04), but the association lost its significance after adjustment for BMI (OR 1,59; 95% CI 0,83–3,02; p=0,15). Nesfatin and obestatin concentrations did not differ between hypertensive and normotensive groups. In multiple linear regression, after adjustment for age and BMI, chemerin was significantly related with left ventricular mass (b=0,23, p=0,02) and left atrial volume (b=0,35, p<0,01), and E/E' (b=0,29, p=0,02) but not with PWV and IMT.
Conclusions: In postmenopausal women serum chemerin is independently associated with left ventricular mass, left atrial volume, parameters of diastolic function and components of metabolic syndrome. Association of chemerin with hypertension is mediated by body adiposity. Correlation of chemerin with left ventricular mass, its diastolic function and left atrial volume suggests its possible role in pathogenesis of obesity-related heart failure.

P1571
Identification of radial vascular wall abnormalities by very-high frequency ultrasound in patients with spontaneous coronary artery dissection and fybromuscular dysplasia: the FUCHSIA study

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Background: Spontaneous coronary dissection (SCAD) is increasingly considered as a disease related to fibromuscular dysplasia (FMD). This case-control study is aimed at identifying common subclinical abnormalities in the vascular wall of the radial arteries of patients with FMD and SCAD by means of a novel ultrasound technique.
Methods: Two 5'-clips from the left radial artery were obtained by Vevo MD (70 MHz probe, FUJIFILM, VisualSonics). Intima-media (IMT) and adventitia (AT) thickness and wall cross-sectional area (WCSA) were measured. Vascular wall disarray was assessed calculating the normalized root mean square error (RMSE/mean) between 20 gray-level profiles crossing the two visible echogenic interfaces.
Results: 12 SCAD patients, 21 FMD patients and 12 healthy controls (C), matched for age (51±12, 47±7, 43±12, p=0.12) and sex (2 M, 1 M, 2 M), were enrolled. IMT (0.182±0.056, 0.170±0.033, 0.133±0.020 mm, p=0.01) and AT (0.108±0.027, 0.116±0.029, 0.076±0.014mm, p=0.001) were higher in SCAD and FMD compared to C. WCSA, but not M/L, was increased too in SCAD and FMD. RMSE/mean was significantly increased in SCAD and FMD compared to controls either in the 1st (1.48±0.84, 1.28±0.52, 0.74±0.26, p=0.01) or in the 2nd interface (2.44±1.38, 1.56±0.58, 1.19±0.93 p=0.01).

Healthy control   FMD patient   SCAD patient
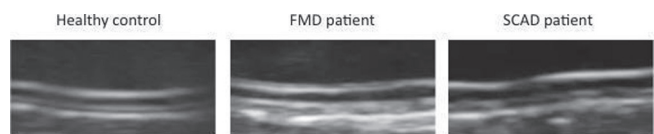
Conclusions: The radial artery walls of SCAD and FMD patients present similar subclinical abnormalities, namely increased thickness and and inhomogeneity, in comparison to controls.