Objective: In a high-risk population, we aimed to determine how the volume of EAT is linked to coronary artery disease (CAD) and to identify potential EAT-dysregulated pathways in CAD patients specifically related to coronary artery calcification (CAC).
Methods and results: In a prospective cohort of 574 degenerative severe aortic stenosis patients referred to cardiac surgery, we quantified fat depots by computed tomography (CT) and performed a comparative quantitative proteomics of thoracic fat, including EAT, mediastinal (MAT) and subcutaneous (SAT) adipose tissues. We did not find an independent association of EAT volume with the severity, distribution, and complexity of coronary stenosis on invasive coronary angiography or coronary calcification on CT, but patients with CAD presented a specific EAT proteomics profile in comparison with matched-controls without CAD, and compared with MAT and SAT. The EAT proteomic signature of CAD was characterized by up-regulation of pro-calcifying annexins (Annexin A2, ANXA2), fatty acid binding transporters (FABP4) and inflammatory signaling proteins, and by down-regulation of fetuin-A and antioxidant enzymes (Figure 1a). In EAT, ANXA2 regulation was positively correlated with CAC (Figure 1b). EAT gene expression studies confirmed overexpression of ANXA2 and FABP4 in CAD, but no expression of FETUA was detected (Figure 1c, d). Compared with non-CAD, fetuin-A circulating levels were higher in CAD, whereas no fetuin-A pericardial fluid differences were found (Figure 1e, f).
Conclusions: In this high-risk cohort, EAT presented an imbalance of pro-calcifying, pro-inflammatory and lipid transporters mediators. These local EAT-mediated regulatory mechanisms were not reflected by the volume of EAT indicating that a new imaging biomarker of fat biology is needed.

CARDIAC – CTA FOR NON-ATHEROSCLEROTIC CLINICAL SCENARIOS

6182
Does myocardial bridge assessed by coronary CT angiography predict vasospasm of left anterior descending?

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Background: Myocardial bridge (MB) of the left anterior descending (LAD) identified as "milking effect" on invasive coronary angiography (ICA) has been reported to be associated with vasospastic angina.
Purpose: To investigate the relation among MB of LAD assessed by coronary CTA, ICA, and coronary artery vasospasm provoked by acetylcholine (ACh).
Methods: Of 807 patients with neither a history of coronary artery disease nor ≥50% stenoses of major coronary arteries on coronary CTA from 2011 to 2017, 113 patients (58% male, aged 65.9±12.0 years) with suspected vasospastic angina were performed ACh provocation test and enrolled. CTA-MB was classified by CTA as either full (LAD being surrounded completely by the myocardium) or partial (LAD being within the interventricular gorge and in direct contact with left ventricular) myocardium encasement. ICA-MB was defined as systolic phase vessel compression after intracoronary isosorbide dinitrate infusion with >30% reduction in the diameter of LAD. LAD spasm was defined as >50% diameter reduction after intracoronary ACh injection analyzed by computerized quantitative coronary angiography.
Results: Of 113 patients, 64 patients had LAD vasospasm. There were significant differences in current smoking (36% vs. 18%, P=0.04), CTA-MB including full and partial encasement (70% vs. 39%, P<0.01), and ICA-MB (44% vs. 18%, P<0.01) between patients with and without LAD spasm. Diagnostic accuracy, sensitivity, specificity, positive predictive value, and negative predictive value of CTA-MB (full and partial encasement) for predicting LAD spasm were 57%, 70%, 39%, 60%, and 50%, and those of CTA-full encasement only were 60%, 50%, 73%, 71%, and 53%, though those of ICA-MB were 60%, 44%, 82%, 76%, and 53%, respectively. After adjustment for age, sex, and current smoking, CTA-MB was an independent factor associated with LAD spasm (OR 3.45, 95% CI 1.54–7.97, P<0.01). Adding CTA-MB to a model with clinical factors including age, sex, and current smoking for predicting LAD spasm improved C-index (from 0.590 to 0.692, P=0.04), NRI (0.631, P<0.01), and IDI (0.086, P<0.01). Of the patients with LAD spasm (N=64), there were significant differences in the prevalence of multivessel spasm (31% vs. 66%, P<0.01) and JCSA Risk Score (2 [2–4] vs. 4 [2–5], P=0.03) including history of out of hospital cardiac arrest, smoking, angina at rest alone, multivessel spasm, ST elevation, and βblocker use between the patients with and without CTA-MB.
Conclusions: MB assessed by CTA could potentially predict the presence of LAD spasm non-invasively. Meanwhile, multivessel spasm being derived from excessive contraction of vascular smooth muscle or vascular endothelial dysfunction other than LAD systolic compression can be missed if we are focusing on only MB in the LAD.

6183
Prevalence of myocardial bridging in patients with hypertrophic cardiomyopathy: a coronary computed tomography study

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Background: Reported prevalence of coronary artery bridges (CBs) in patients with hypertrophic cardiomyopathy (HCM) varies between 15% and 55% in autopsy and in invasive coronary angiography, their prevalence has never been reported using computed tompgraphy (CT).
Purposes: The objective of this study was to evaluate the frequency and pattern of CBs as assessed by CT in patients with HCM and to evaluate their possible association with non- sustained ventricular tachycardia (NSVT).
Methods: The study population consisted of 85 patients with HCM (56.4±12.4 years; 77% male), and 79 aged and sex matched normal subjects (52.8±12.5 years; 77% male), who were referred for coronary CT angiography. The following parameters were measured: number, length, and depth of CBs within left ventricular (LV) myocardium; and their course in relation to the left and right ventricular and atrial myocardium. The association between CBs and NSVT was evaluated using Fischer's exact test.
Results: Myocardial bridges were more common in HCM than in normal subjects (59/85; 69% vs. 26/79; 33%; retrospectively, P<0.001). As compared with normal subjects CBs in HCM patients were longer (25.3±13.1 mm vs. 18.5±14.9 mm; P=0.045) and deeper within LV myocardium (2.9±1.7 mm vs. 1.6±1.1 mm; P=0.001). No difference was evident between HCM with or without CBs in regard to LV mass (238±88g vs. 235±88g; retrospectively, P=0.92) or coronary blood volume (6.95±2.1 ml vs. 7.12±1.8 ml; retrospectively, P=0.96). In a subgroup of 38 patients with HCM the prevalence of CBs was similar in patients with and without NSVT (16/38; 42% vs. 22/38; 58% retrospectively; P=0.67).
Conclusion: The frequency of myocardial bridging in patients with HCM observed by coronary CT angiography was 69%, higher than in angiographic and pathologic series, but was not associated with increased risk of NSVT. The number, length, depth, and course of CBs differ between patients with HCM and normal subjects.

6184
Identification of coronary collateral vessel by coronary computed tomography

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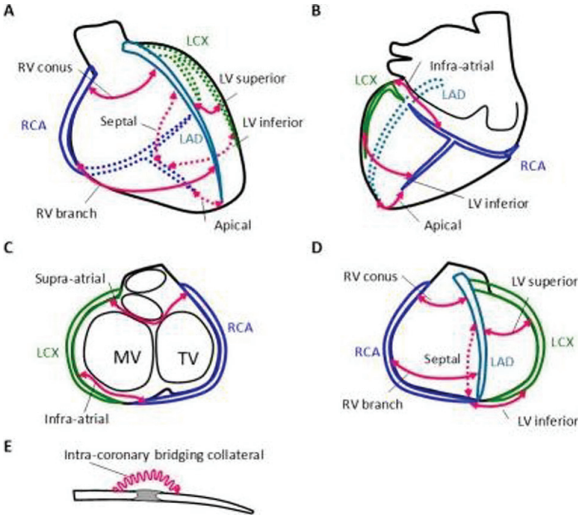
Background: Well-developed coronary collateral is protective from myocardial ischemia, mitigates myocardial infarct, and improves survival in patients with totally occluded coronary arteries. However, systematic non-invasive description of collateral is limited. We investigated non-invasive identification of coronary collateral using coronary computed tomography angiography (CCTA).
Methods and results: In this multicenter registry, we investigated collaterals supplying 1,019 totally occluded arteries from 910 patients who underwent both CCTA and coronary angiography. CCTA identified collateral by continuous vascular connection between donor and recipient vessels. Angiographic well-developed collateral was defined by Rentrop score=3 and collateral connection score=2, which respectively reflects function and size of collateral flow. In per vessel anal-



Nine different collateral pathways