ESC Congress 2016
Abstract: P5237

Coronary atherosclerotic plaque burden is associated with reduced pericoronary adipose tissue volume: data from application of a novel 3D Computerised Tomography imaging method

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Topic(s): X-ray Computed Tomography (CT)

Citation: European Heart Journal (2016) 37 (Abstract Supplement), 1053

Background: Perivascular adipose tissue (AT) is directly implicated in vascular disease development. Imaging studies have associated pericoronary adipose tissue volume (PVAT) with coronary atherosclerosis.

Purpose: To explore the associations between PVAT volume and coronary atherosclerotic plaque burden.

Methods: In Study A, in 258 patients undergoing coronary CT angiography (CTA) on 64-slice CT scanner, a 40mm segment of the proximal right coronary artery (RCA) was analysed on a dedicated workstation for plaque burden and composition. PVAT volume was calculated by tracking of fat (-190 to -30HU) around the same segment a) at 5mm from RCA wall (as previously suggested, PVAT vol-5mm), b) at a distance equal to vessel diameter (PVAT vol-d) and c) by tracking a constant volume of tissue around RCA (PVAT vol-c) irrespectively of vessel size. Epicardial (EpAT), and pericardial (PerAT) AT volumes were also quantified. In Study B, PVAT was quantified around the culprit lesion in 5 NSTEMI patients on admission and 4 weeks later, to search for dynamic changes in PVAT volume during the post-NSTEMI period, as well as in stable CAD patients (control subjects).

Results: PVAT vol-5mm (not shown) and PVAT vol-d (a) were positively correlated with fibrous plaque volume (defined as 65-260HU) but this association was biased by disease-related vascular remodelling; PVAT vol-c (a more appropriate index to overcome technical biases) was strongly inversely correlated with fibrous plaque (b) [but not calcium (c)] volume and coronary plaque burden (defined as fibrous plaque volume to total vessel volume, d). PVAT vol-c was strongly associated with EpAT (e) and PerAT (f) volume in the same patients. In study B, there was an increase in PVAT vol-c at 4weeks in NSTEMI (but not in stable CAD) patients compared to baseline (g), suggesting an expansion of PVAT with withdrawal of vascular inflammation.

Conclusions: We now describe a novel method to quantify PVAT volume in human coronaries to avoid technical bias related with vascular remodelling. We show that plaque development is associated with less PVAT volume around human coronaries, a finding opposite to what was believed so far. Moreover, resolution of vascular inflammation during the post-NSTEMI period is associated

with significant expansion of PVAT, suggesting that PVAT expansion is dynamic, and responds both to systemic & local stimuli derived from the vascular wall.