P3.10: REACTIVITY TO LOW-FLOW IN THE BRACHIAL ARTERY: A POTENTIAL DETERMINANT FOR FLOW-MEDIATED DILATORY RESPONSE

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P3.8
ACUTE EFFECTS OF CONTRAST MEDIA ON CENTRAL HEMODYNAMICS, ARTERIAL STIFFNESS, INFLAMMATORY AND KIDNEY INJURY BIOMARKERS
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Introduction: Contrast-induced nephropathy (CIN) is widely recognized as the third most common type of hospital-acquired acute kidney injury. Despite the impact of CIN on adverse cardiovascular events, little is known about the effects of contrast media on arterial functional properties. The purpose of this study was to evaluate effects on central hemodynamics, arterial stiffness as well as changes in biomarkers of inflammation and kidney injury after contrast media administration.

Methods: The cohort comprised 36 symptomatic peripheral arterial disease (PAD) patients with GFR > 60mL/min/1.73m2 undergoing a lower extremities digital subtraction angiography (DSA) with intra-arterial iso-osmolar iodinated contrast media. Central hemodynamics and arterial stiffness parameters were determined by applanation tonometry (on admission, 8h, 24h after DSA). Serum samples and urine samples were also collected. All samples underwent ELISA testing for biomarkers. Statistically significant differences were calculated by one-way ANOVA and paired t-test.

Results: Augmentation pressure decreased and pulse pressure amplification increased significantly (p = 0.01; p = 0.02, respectively) compared to baseline, but there were no changes in augmentation index and aortic pulse wave velocity. Increase in the levels of serum neutrophil gelatinase lipocalin, urinary kidney injury molecule-1, beta-2 microglobulin, high-sensitivity CRP, myeloperoxidase and interleukin-6 were observed compared to baseline (p < 0.05).

Conclusions: This is the first clinical study to evaluate the complex effects of contrast media on arterial stiffness, central hemodynamics and several biomarkers. The results suggest that in PAD patients without impaired renal function contrast media may induce acute inflammation, moderate tubular injury and changes in central hemodynamics.